Formaldehyde

November 2006

National Industrial Chemicals Notification and Assessment Scheme
GPO Box 58, Sydney NSW 2001, Australia  www.nicnas.gov.au
Preface

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

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

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

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

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

This Priority Existing Chemical report has been prepared by the Director, NICNAS, in accordance with the Act. Under the Act manufacturers and importers of Priority Existing Chemicals (applicants) are required to apply for assessment. Applicants for assessment are given a copy of the draft report and 28 days to advise the Director of any errors. Following the correction of any errors, the Director provides applicants and other interested parties with a copy of the draft assessment report for consideration. This is a period of public comment lasting for 28 days during which requests for variation of the draft report may be made. Where variations are requested the Director’s decision concerning each request is made available to each respondent and to other interested parties (for a further period of 28 days). Notices in relation to public comment and decisions made appear in the *Commonwealth Chemical Gazette*. A person may apply (within 28 days) to the Administrative Appeals Tribunal (AAT) for review of decision(s) where the Director has refused to vary the draft report as requested.

The draft formaldehyde report was published in September 2005. Several parties submitted applications to vary the draft report. Following the Director's decisions concerning the variation requests, the Formaldehyde Council Inc., Australian Wood Panels Association Inc and Plywood Association of Australasia lodged applications with the Administrative Appeals Tribunal (AAT) in November 2005. All parties withdrew their applications before the hearing and the final order to dismiss the applications was made by the AAT in October 2006. This report is the final published report.

In accordance with the Act, publication of this report revokes the declaration of this chemical as a Priority Existing Chemical, therefore manufacturers and importers wishing to introduce this chemical in the future need not apply for assessment. However, manufacturers and importers need to be aware of their duty to provide any new information to NICNAS, as required under Section 64 of the Act.

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For the purposes of Section 78(1) of the Act, copies of assessment reports for New and Existing Chemical assessments are freely available from the web (www.nicnas.gov.au) and may be inspected by the public at the library of the Office of Australian Safety and Compensation Council (OASCC). Summary Reports are published in the Commonwealth Chemical Gazette (http://www.nicnas.gov.au/Publications/Chemical_Gazette.asp), which are also available to the public at the ASCC library.

Copies of this and other Priority Existing Chemical reports are available on the NICNAS website. Hard copies are available free of charge from NICNAS from the following address:

GPO Box 58, Sydney, NSW 2001, AUSTRALIA
Tel: +61 (2) 8577 8800
Fax: +61 (2) 8577 8888
Free call: 1800 638 528

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

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

More information on NICNAS can be found at the NICNAS website:

http://www.nicnas.gov.au

Other information on the management of workplace chemicals can be found at the website of the Office of the Australian Safety and Compensation Council (OASCC):

http://www.ascc.gov.au
Overview and Recommendations

Overview

Formaldehyde (CAS No. 50-00-0) was declared a Priority Existing Chemical on 5 March 2002 in response to occupational and public health concerns.

Formaldehyde occurs naturally in the atmosphere through a variety of biological and chemical processes. As a result of various metabolic processes, formaldehyde is naturally present in the human body at very low concentrations. It is also produced incidentally in the course of natural processes and human activities that involve the combustion of organic materials, such as bush fires and fuel.

Formaldehyde is manufactured in Australia as aqueous solutions known as ‘formalin’, at approximately 55 000 tonnes per annum (calculated as 100% formaldehyde). Formalin and products/mixtures containing formaldehyde are also imported at approximately 90 tonnes (100% formaldehyde) per year. In addition, approximately 700 tonnes per year of paraformaldehyde (a significant source of formaldehyde) is imported.

Uses

The main industrial use of formaldehyde and paraformaldehyde is for the manufacture of formaldehyde-based resins, which are widely used in a variety of industries, predominantly the wood industry. Formaldehyde is also used directly or in formulations in a number of industries including medicine-related industries (such as forensic/hospital mortuaries and pathology laboratories), embalming in funeral homes, film processing, textile treatments, leather tanning, and a wide range of personal care and consumer products. The concentrations of formaldehyde in these products range from 40%, such as in embalming and film processing solutions, to < 0.2%, such as in the majority of cosmetics and consumer products.

Environmental exposure, effects and risks

Formaldehyde is water soluble and biodegradable. Its major environmental release is to the atmosphere, where it breaks down in a short period of time. Direct release to the aquatic compartment and soil is expected to be minor and significant removal occurs through biodegradation. The short atmospheric lifetime of formaldehyde and worst-case predicted environmental concentrations indicate that no significant risks to non-human organisms through atmospheric exposure to formaldehyde are expected. A low environmental risk to terrestrial organisms is also predicted due to likely low concentrations of formaldehyde in aquatic systems and soil.

Health effects

In humans and experimental animals, formaldehyde is readily absorbed by all exposure routes. When inhaled, it reacts rapidly at the site of contact and is quickly metabolised in the respiratory tissue.

Following acute exposure via inhalation, dermal and oral routes, formaldehyde is moderately toxic in animals. Humans experience sensory irritation (eye, nose and
respiratory tract irritation) at levels in air of 0.5 ppm formaldehyde and above. Evidence clearly indicates that formaldehyde solution is a skin irritant and a strong skin sensitiser.

The available human and animal data indicate gaseous formaldehyde is unlikely to induce respiratory sensitisation. Lung function tests suggest that asthmatics are no more sensitive to formaldehyde than healthy subjects. Limited evidence indicates that formaldehyde may elicit a respiratory response in some very sensitive individuals with bronchial hyperactivity, probably through irritation of the airways.

No systemic toxicity was observed following repeated exposure to formaldehyde in animals and humans. Effects at the site of contact show clear dose-related histological changes (cytotoxicity and hyperplasia). A no-observed adverse-effect level (NOAEL) of 1 ppm (1.2 mg/m³) by inhalation and a NOAEL of 15 mg/kg bw/day by oral administration were identified for histopathological changes to the nasal tract and the fore- and glandular stomach in the rat, respectively.

Formaldehyde is clearly genotoxic in vitro, and may be genotoxic at the site of contact in vivo. Overall, formaldehyde is considered to have weak genotoxic potential.

The possible relationship between formaldehyde exposure and cancer has been studied extensively in experimental animals and humans. There is clear evidence of nasal squamous cell carcinomas from inhalation studies in the rat, but not in the mouse and hamster. Although several epidemiological studies of occupational exposure to formaldehyde have indicated an increased risk of nasopharyngeal cancers, the data are not consistent. The postulated mode of action for nasal tumours in rats is biologically plausible and considered likely to be relevant to humans.

There are also concerns of an increased risk for formaldehyde-induced myeloid leukaemia, however, the data are not considered sufficient to establish a causal association. In addition, there is currently no postulated mode of action to support such an effect. NICNAS will maintain a watching brief on the issue of leukaemia and formaldehyde exposure.

Based on the available nasopharyngeal cancer data, formaldehyde should be regarded as if it may be carcinogenic to humans following inhalation exposure. Formaldehyde meets the National Occupational Health and Safety Commission’s (NOHSC) Approved Criteria for Classifying Hazardous Substances (NOHSC, 2004) as a Category 2 carcinogen (Risk phrase R49, may cause cancer by inhalation). This classification should replace the current classification of Carcinogen, Category 3 (R40, limited evidence of a carcinogenic effect) in the Hazardous Substances Information System (DEWR, 2004). Other classifications that remain applicable are: toxic by inhalation, in contact with skin and if swallowed (R23/24/25), causes burns (R34), and may cause sensitisation by skin contact (R43).

Based on animal and limited epidemiology data, formaldehyde is unlikely to cause reproductive and developmental effects at exposures relevant to humans.

The critical health effects of formaldehyde for risk characterisation are sensory irritation, skin sensitisation and carcinogenicity. Although gaseous formaldehyde is a known eye and upper respiratory tract irritant in humans, the limitations of the available data and subjective nature of sensory irritation do not allow identification of a definitive no-observed-effect level (NOEL). The lowest-observed-effect level (LOEL) for sensory irritation in humans is 0.5 ppm. Formaldehyde solution is also a strong skin sensitiser.
A 2-stage clonal growth model was developed by the Chemical Industry Institute of Toxicology (CIIT) in the United States to assess the respiratory carcinogenic risk of formaldehyde to humans. This is a biologically-based, dose-response model that incorporates mechanistic data. The model takes into account respiratory tract physiology and regional air flow in animals and humans. It is considered a more reliable estimate of cancer risk than the use of standard default assumptions, due to the incorporation of all available biological data.

The table below shows key estimates of the human carcinogenic risk for public and occupational exposure (for non-smokers) using the CIIT model.

<table>
<thead>
<tr>
<th>Exposure Concentration</th>
<th>Predicted Additional Respiratory Cancer Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Public</td>
</tr>
<tr>
<td>0.10 ppm (100 ppb)</td>
<td>H 0.3 in 1 million</td>
</tr>
<tr>
<td>0.30 ppm (300 ppb)</td>
<td>H 1 in 1 million</td>
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<tr>
<td>1.00 ppm (1000 ppb)</td>
<td>H 33 in 1 million</td>
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</table>

**Public exposure and health risks**

Formaldehyde is naturally present in the air we breathe and in the food and water we eat and drink. In addition, a wide range of human domestic and industrial activities is responsible for both direct and indirect release of formaldehyde into the atmosphere from diffuse and point sources. The principal route of public exposure is by inhalation, via indoor and outdoor (ambient) air.

The estimated environmental exposures to formaldehyde using modelling techniques indicate that the maximum annual average concentration of formaldehyde in urban air is 5.5 ppb and the maximum 24-h average is 23.5 ppb. Based on the CIIT carcinogenic risk estimates of formaldehyde to humans (see table above), the public health risk of respiratory tract cancer after repeated exposure to formaldehyde levels in ambient air is low (less than 1 in a million). The risk of sensory irritation to the public is also low based on the comparison of the NICNAS proposed ambient air standard (80 ppb, see Recommendation 17) and the estimated formaldehyde levels in ambient air.

Formaldehyde concentrations in indoor air are generally higher than outdoor levels. Formaldehyde levels in established conventional homes and buildings are generally low at average levels of 15-30 ppb. However, limited monitoring data indicate that mobile homes and possibly relocatable buildings have higher levels of formaldehyde [average of 29 ppb with a range from 8 to 175 ppb in occupied caravans; average of 100 ppb with a range from 10 to 855 ppb in unoccupied caravans; average of 710 ppb with a range from 420 to 830 ppb in relocatable offices (1992 data)]. This is primarily due to the higher usage of products that emit formaldehyde in these buildings, relatively low ventilation rate and/or small internal volume, and other potential sources of formaldehyde such as from combustion of gas used in cooking and refrigeration. There is a potential risk of sensory irritation for people living in these types of buildings, but the risk of nasal cancer is estimated to be low.

Due to public concern of childhood chemical exposure and cancers, together with the findings of relatively high levels of formaldehyde in mobile homes and relocatable buildings, a worst-case scenario risk estimation incorporating higher exposures during childhood has been conducted using the CIIT model. The worst-case scenario was
identified to be children who live in mobile homes and spend all their schooling time in relocatable classrooms up to 17 years of age. The predicted additional risk of respiratory tract cancer for a full 80-year lifetime, including childhood exposure to formaldehyde under the worst-case scenario is low, at 0.45 in a million.

The general population may also come into skin contact with formaldehyde solutions due to its use in a wide range of cosmetics and consumer products. However, the majority of the products contain formaldehyde at low concentrations (< 0.2%). Because formaldehyde solutions may induce skin sensitisation and even very low concentrations of formaldehyde in solution may elicit a dermatological reaction in individuals who have been sensitised, dermal exposure should be minimised or prevented wherever possible.

**Occupational exposure and health risks**

Occupational exposure during importation, transportation and storage of formaldehyde is limited, except in cases of accidental spills or leaks of the chemical. The principle occupational exposure route for formaldehyde is inhalation. Workers may be exposed to formaldehyde vapours during resin manufacture, product formulation, and end use. During repackaging, formulation and end use of formaldehyde products, workers are likely to be exposed by skin and eye contact during handling of formaldehyde solutions, such as in manual operations and cleaning of equipment.

The risk characterisation identified concerns in a number of use scenarios based on sensory irritation. The risk of sensory irritation in embalmers and workers in medicine-related industries, such as forensic/hospital mortuaries and pathology laboratories, is high due to high concentrations of formaldehyde products handled and relative long exposure durations. The risk of sensory irritation also exists during formaldehyde and formaldehyde resin manufacture (when formaldehyde vapour replacement occurs and where there is a need to break open or enter the enclosed system), product formulation (during raw material weighing and transfer, open mixing process, and equipment cleaning and maintenance), and end use (when formaldehyde product is heated and/or in contact with high humidity, use of formaldehyde resins that contain high levels of free formaldehyde, and during certain modes of application that may generate formaldehyde vapour e.g. spraying).

Skin sensitisation of workers can occur as a result of manual handling of formaldehyde products during formaldehyde and resin manufacturing, formulation, repackaging, and end uses. The likelihood of skin contact in some end use scenarios, such as spraying or brushing, is high. Because formaldehyde solutions may induce skin sensitisation and even very low concentrations of formaldehyde in solution may elicit a dermatological reaction in individuals who have been sensitised, dermal exposure should be minimised or prevented wherever possible.

The CIIT carcinogenic risk estimation of formaldehyde to humans indicates that the risk for respiratory tract cancer is low (less than 1 in a million) after 40 years repeated occupational exposure to δ 0.6 ppm formaldehyde. Limited monitoring data indicate that formaldehyde levels at the majority of workplaces are < 0.2 ppm. Consequently, the occupational risks for respiratory tract cancers after repeated exposure to formaldehyde by inhalation is likely to be low.

The occupational risks can be managed by a number of control measures to reduce workers’ exposure to formaldehyde, such as elimination, process improvements (e.g. use of an automated or enclosed system), effective ventilation, and proper use of personal protective equipment.

*Formaldehyde*
The current national exposure standard is 1 ppm 8h time-weighted average (TWA) and 2 ppm short-term exposure limit (STEL). It is recommended that the occupational exposure standard be lowered to 0.3 ppm 8h TWA and 0.6 ppm STEL. This recommended standard not only provides adequate protection against discomfort of sensory irritation (the health endpoint on which the proposed standard is set), but also provides a high level of protection for cancer.

Recommendations

The recommendations arising from the assessment of formaldehyde are made for occupational health, public health, and environmental protection. The critical issues that have been taken into consideration in formulating these recommendations are summarised in the preamble for each of these areas.

Recommendations for Occupational Health and Safety

Preamble

It is best occupational health and safety (OHS) practice to follow the hierarchy of controls when a risk assessment indicates a potential risk to workers’ health due to use of chemicals in the workplace.

The hierarchy of controls are:

1. Elimination
2. Substitution
3. Engineering controls
4. Safe work practices (Administrative practices)
5. Personal protective equipment

When deciding on the best way to control a risk, start at the top of the hierarchy of controls, i.e. investigate if the risk can be eliminated first, for example, by changing the way the work is done, or by using safer substances. This is the most effective way to control a hazard. If these methods are not possible, use engineering or administrative controls to reduce or minimise the risk. The final approach is to use appropriate personal protective equipment if the risk needs further control.

In addition, personal monitoring should be conducted where a workplace assessment indicates a potential risk to health due to exposure to hazardous chemicals, particularly, workplaces with possible high exposure to the chemical.

Based on the known hazards and risks of formaldehyde, the hierarchy of controls should be implemented to manage occupational exposure to formaldehyde.

Specifically for formaldehyde, it is noted that:

- The best available LOEL for non-cancer effects in humans is 0.5 ppm for sensory irritation;
- Formaldehyde in solution is a strong skin sensitiser;
- Formaldehyde may cause nasal cancer by inhalation;
The predicted risk for respiratory tract cancers is less than 1 in a million workers at occupational exposure levels \( \delta 0.6 \) ppm;

The occupational risk characterisation identified concerns in a number of use scenarios, particularly in embalming and medicine-related industries;

The current Australian occupational exposure standard is 1 ppm time-weighted average (TWA), and 2 ppm short-term exposure limit (STEL);

The following recommendations are made:

**Recommendation 1. Occupational hazard classification (OASCC)**

a) Based on the hazard assessment, formaldehyde should be classified as:

<table>
<thead>
<tr>
<th>Risk Phrase</th>
<th>Concentration Cut-off</th>
</tr>
</thead>
<tbody>
<tr>
<td>R23/24/25</td>
<td>toxic by inhalation, in contact with skin and if swallowed</td>
</tr>
<tr>
<td>R34</td>
<td>causes burns</td>
</tr>
<tr>
<td>R43</td>
<td>may cause sensitisation by skin contact</td>
</tr>
<tr>
<td>R49</td>
<td>may cause cancer by inhalation (Carcinogen, Category 2)</td>
</tr>
</tbody>
</table>

Compared with the current hazard classification for formaldehyde in the *Hazardous Substances Information System* of the Office of the Australian Safety and Compensation Council (OASCC), only classification for carcinogenicity has been changed (from Category 3).

b) Based on the NOHSC’s *Approved Criteria for Classifying Hazardous Substances* (NOHSC, 2004), the appropriate risk phrases for mixtures containing formaldehyde are:

<table>
<thead>
<tr>
<th>Risk Phrase</th>
<th>Concentration Cut-off</th>
</tr>
</thead>
<tbody>
<tr>
<td>R49</td>
<td>( \varepsilon 0.1% ) to (&lt;0.2%)</td>
</tr>
<tr>
<td>R49, R43</td>
<td>( \varepsilon 0.2% ) to (&lt;3%)</td>
</tr>
<tr>
<td>R49, R43, R36/38, R20/21/22</td>
<td>( \varepsilon 3% ) to (&lt;10%)</td>
</tr>
<tr>
<td>R49, R43, R34, R20/21/22</td>
<td>( \varepsilon 10% ) to (&lt;25%)</td>
</tr>
<tr>
<td>R49, R43, R34, R23/24/25</td>
<td>( \varepsilon 25%)</td>
</tr>
</tbody>
</table>

**Key:**

- R20/21/22 Harmful by inhalation, in contact with skin and if swallowed
- R23/24/25 Toxic by inhalation, in contact with skin and if swallowed
- R34 Causes burns
- R36/38 Irritating to eyes and skin
- R43 May cause sensitisation by skin contact
- R49 May cause cancer by inhalation

It is recommended that this classification be included in the *Hazardous Substances Information System* (HSIS) as soon as possible.


2.1 It is recommended that OASCC (formerly NOHSC) lower the current occupational exposure standard for formaldehyde. Based on the hazard assessment for
formaldehyde, NICNAS recommends that the new standard be 0.3 ppm (0.36 mg/m³) 8h TWA and 0.6 ppm (0.72 mg/m³) STEL. The recommended new standard offers adequate worker protection for extended shifts. The documentation to support the recommended exposure standard is in Appendix 16, which will serve as an attachment in the OASCC Regulatory Impact Statement when the proposed exposure standard is released for public comment. The OASCC should consider the recommended exposure standard as a matter of priority, with a view to declaration of a new standard within 12 months.

Australian monitoring studies, whilst limited, indicate that in some sectors, particularly workplaces manufacturing pressed wood products and mortuary and forensic/hospital and pathology laboratories, exposure levels are likely to regularly exceed the proposed new health-based exposure standard. These data need to be considered by OASCC in their development of a new occupational exposure standard and the timing of its implementation, noting such issues will be subject to further consultation with stakeholders under the OASSC exposure standard setting process.

2.2 Anecdotal information provided to NICNAS indicates that, in practice, occupational exposure standards (TWAs and STELs) appear to be misinterpreted. For example, industry has advised that it is their understanding that workplaces need to operate at half the level of an exposure standard to ensure compliance with the standard. To address this, it is recommended that the OASCC and state and territory workplace safety authorities develop and disseminate clear guidance on the application of national exposure standards in the workplace.

Recommendation 3. Use of formaldehyde in spray and aerosol products (Industry)

It is recommended that activities involving spraying of formaldehyde or products containing formaldehyde only be carried out in a controlled manner using adequate engineering controls and other suitable protection. If such controls or protection cannot be provided for an activity, spraying should not be permitted.

Recommendation 4. Hazard communication (Industry)

It is recommended that suppliers and employers take note of the new hazard classification in regards to carcinogenicity (Category 2 - may cause cancer by inhalation) and amend Material Safety Data Sheets (MSDS), labels and training materials accordingly.

It is recommended that all manufacturers, suppliers and employers review their hazard communication, paying particular attention to the following points:

MSDS (see Sample MSDS, Appendix 14):
- correct identification of health hazards, especially skin sensitisation, corrosiveness, and carcinogenicity;
- correct information on the concentration cut-offs for mixtures containing formaldehyde;
- first aid advice, including the advice that vomiting should not be induced; and
- include the Australian occupational exposure standard.

Labels:
- correct signal word;
correct risk and safety phrases;
include emergency procedures; and
correct first aid statements.

Recommendation 5. Specific recommendations for the embalming industry (Industry)

It is recommended that the Australian Funeral Directors Association (AFDA) and the Australian Institute of Embalming (AIE), together with the registered training organisations for embalming industry, the Funeral Industry Development Australia (FIDA) and Mortuary and Funeral Educators (MFE), use the information in this report to 1) update information on formaldehyde in their training materials for embalmers; 2) develop a specific guideline for controlling non-infectious hazards such as hazardous chemicals (including formaldehyde) for embalmers. The development of any materials and guidelines should be in consultation with relevant stakeholders such as state/territory authorities and organisations representing the workers;

The following workplace controls are recommended:

- Employers of embalming industry should consider replacing high concentration formalin products with low concentrations or less hazardous or formaldehyde-free products, if available;
- Effective ventilation is a critical control measure for embalmers. It is recommended that the embalming industry ensure that a ventilation system is in place and is effective at maintaining exposure levels below the recommended national exposure standard of 0.3 ppm (TWA) and 0.6 ppm (STEEL); and
- Embalmers should pay particular attention to the type of personal protective equipment (PPE) used during embalming. Relevant Australian standards and/or guidance from manufacturers in selecting and use of PPE should be followed. Respirators should be used in situations where high formaldehyde levels and high frequency exposures may be encountered which may be above the occupational exposure standard, such as embalming post-mortem bodies;
- NICNAS will prepare a Safety Information Sheet in consultation with industry, organisations representing the workers and relevant state/territory government organisations, specifically for safe use of formalin products in the embalming industry. It is recommended that employer industry associations and unions distribute this information widely to their members and workers.

Recommendation 6. Specific recommendations for forensic/hospital mortuaries and pathology laboratories (Industry)

It is recommended that the Royal College of Pathologists of Australasia (RCPA), National Institute of Forensic Science (NIFS), Australian Forensic Medicine Managers Association (AFMMA), and other relevant associations and training organisations use the information in this report to 1) update information on formaldehyde in training materials for these industries; 2) develop a guideline for controlling hazardous workplace chemicals including
formaldehyde. The development of any materials and guidelines should be in consultation with relevant stakeholders such as state/territory authorities and organisations representing the workers;

- The following workplace controls are recommended:
  - Use of local exhaust ventilation at each specimen station;
  - Relocate specimen vats to areas with isolated ventilation or use local exhaust ventilation over the vats;
  - Avoid the need for dilution of concentrated formalin products by purchasing diluted formalin products;
  - Ensure effective ventilation, especially in areas where formaldehyde levels may be high, such as exhaust ventilation in storage areas, and down draught arrangements at dissection areas; and

- NICNAS will prepare a Safety Information Sheet in consultation with industry, organisations representing the workers and relevant state/territory government, specifically for safe use of formalin in forensic/hospital mortuaries and pathology laboratories. It is recommended that employer industry associations and unions distribute this information widely to their members and workers.

**Recommendation 7. Compliance with state and territory legislation (Government)**

It is recommended that state and territory OHS authorities review the compliance of workplaces with the workplace controls recommended in this report, including occupational exposure standard, MSDS and labels. Reviews should be conducted at an appropriate interval to allow for the adoption by industry of the recommended workplace controls, and should target industries with potential for high formaldehyde exposure, such as the embalming industry.

**Recommendation 8. Communication (Government and industry)**

NICNAS will prepare a Safety Information Sheet for formaldehyde in consultation with industry, organisations representing the workers, and relevant state/territory government, aimed primarily at workers in general who use formaldehyde products. It is recommended that state/territory jurisdictions and organisations representing the workers distribute this information widely.

**Recommendations for Public Health**

**Preamble**

Noting that:

- The best available LOEL for non-cancer effects in humans is 0.5 ppm for sensory irritation;
- Formaldehyde in solution is a strong skin sensitiser;
- Formaldehyde may cause nasal cancer by inhalation;
- Respiratory tract cancer risk estimates for the general public (including children) are low based on worst-case exposure scenarios;
Formaldehyde concentrations in indoor air are generally higher than outdoor levels;

Limited monitoring data indicate that mobile homes and possibly relocatable buildings have higher levels of formaldehyde, primarily due to use of large quantities of formaldehyde-emitting materials;

Currently there is no national indoor air standard or guidance value for formaldehyde;

The direct and indirect exposure of the general public via cosmetic and consumer products is expected to be widespread and repeated. Overseas countries, such as the European Union (EU), have restrictions on use of formaldehyde in cosmetic products; and

Based on the hazard profile of formaldehyde, it is prudent to eliminate or reduce formaldehyde exposure to the public wherever possible.

The following recommendations are made:

**Recommendation 9. Indoor air guidance value (Government)**

NICNAS recommends an indoor air guidance value of 80 ppb (sampling over a short duration). This guidance value is based on sensory irritation, an acute effect. Therefore, the sampling duration should be short (such as hourly). This value will provide guidance for the public and regulatory authorities so that the results of monitoring studies can be considered and action taken where appropriate.

This recommendation, together with the full report, will be forwarded to the Australian Government Department of the Environment and Heritage (DEH) and the Environment Protection and Heritage Council (EPHC) for consideration in setting an indoor air standard or guidance value for formaldehyde in the future.

**Recommendation 10. Standards Australia (Non-government organisation)**

It is recommended that Standards Australia

- adopt and/or develop a standard(s) for mobile homes and relocatable buildings which includes guidance on ventilation and use of pressed wood products that meet the revised Australian Standards in regards to formaldehyde emission limits;
- adopt and/or develop applicable method(s) for the sampling and analysis of formaldehyde in indoor air; and
- adopt international testing and labelling practices for assessing emissions of formaldehyde from materials, which allow for testing to low emission levels as provided in other countries such as Japan.

**Recommendation 11. Mobile home and relocatable building manufacturers (Industry)**

Manufacturers of mobile homes and relocatable buildings should aim to minimise levels of formaldehyde in indoor air. Recommendations include:

- design the structure to ensure that the recommended indoor air guidance value of 80 ppb is not exceeded;
- only use low formaldehyde-emitting pressed wood products, such as those that meet the Australian Standards for formaldehyde emission limits;
coat or laminate untreated surfaces with materials, such as vinyl or water-resistant coatings to reduce formaldehyde emission; and
ventilate the buildings well before delivery and use to ensure the recommended indoor air guidance value of 80 ppb is met.

Recommendation 12. Residents/occupants of mobile homes and relocatable buildings (The general public)
The following recommendations are for the general public and are particularly relevant to current residents/occupants of mobile homes and relocatable buildings:
- ensure adequate ventilation (exhaust ventilation, fans or window ventilation);
- exhaust all combustion appliances directly to the outdoors;
- purchase low formaldehyde-emitting pressed wood products, such as those that meet Australian Standards for formaldehyde emission limits;
- where possible/practicable, ensure that furniture and fittings are manufactured from materials that are low formaldehyde emitters;
- avoid smoking indoors; and
- avoid high room temperatures and high relative humidity wherever possible, such as through the use of air-conditioning.

Recommendation 13. Indoor air monitoring (Government, industry and research organisations)
In order to more accurately estimate the risks to the public from indoor air exposure to formaldehyde, indoor air monitoring data should be collected, focusing on the buildings with potentially high formaldehyde levels, such as mobile homes and relocatable buildings including classrooms.

Recommendation 14. Communication (Government and industry)
To raise consumer awareness, NICNAS will prepare an Information Sheet, in consultation with industry and other government departments, for distribution to mobile building owners/residents, state and private education departments/offices, and teaching unions. It is recommended that industry, local governments, and other relevant authorities distribute the information widely.

To facilitate consumer choice and use of safer products, low formaldehyde emitting products should be labelled accordingly.

Recommendation 15. Poison Scheduling (Government)
It is recommended that the National Drugs and Poisons Schedule Committee (NDPSC) consider amending the current scheduling for formaldehyde and paraformaldehyde taking note of the following:

1) the need to consider more restrictive categories given its potency of causing skin sensitisation and its classification for the workplace as a Category 2 carcinogen;

2) the need for more protective cut-off values for cosmetics and personal care products containing formaldehyde. The EU cut-off values are highlighted below as representing a potential best practice model and have the following restrictions:
Formaldehyde and paraformaldehyde (as a preservative) for cosmetic use:

- free formaldehyde at 0.2% or less in all cosmetic preparations [except oral hygiene preparations, nail hardeners and aerosol dispensers (sprays)];
- free formaldehyde at 0.1% or less in oral hygiene preparations;
- free formaldehyde at 5% or less in nail hardeners; and
- use of formaldehyde and paraformaldehyde in aerosol dispensers (sprays) is prohibited.

Recommendations 16. Utilisation of the health hazard assessment (Government)

It is recommended that other government organisations, such as Agricultural Pesticides and Veterinary Medicines Authority (APVMA) and Therapeutic Good Administration (TGA), take the findings of the human health hazard assessment into consideration in future work on formaldehyde or products containing formaldehyde, noting use of formaldehyde in therapeutic and agricultural and veterinary products.

Recommendations for Environmental Protection

Preamble

Noting that:

- The major environmental release of formaldehyde is into the atmosphere;
- Formaldehyde is a hazardous air pollutant otherwise known as an ‘air toxic’;
- The release and disposal of formaldehyde from industrial facilities are regulated by licence agreements; and
- Formaldehyde in ambient air is currently being investigated by the National Environment Protection Council (NEPC), as part of their Air Toxics National Environment Protection Measure (NEPM).

The following recommendations are made:

Recommendation 17. Ambient air standard (Government)

It is recommended that NEPC take the data and findings of this report into consideration when setting an ambient air standard for formaldehyde. Evaluation of the available data in this report indicates that an ambient air standard in the order of 80 ppb (sampling over a short duration) would be warranted.

Recommendation 18. Communication (Government)

It is recommended that the Australian Government Department of the Environment and Heritage update the National Pollutant Inventory (NPI) Fact Sheet for formaldehyde in accordance with the findings of this report.
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<td>ACGIH</td>
<td>American Conference of Governmental Industrial Hygienists</td>
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<td>ADG Code</td>
<td>Australian Dangerous Goods Code</td>
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<tr>
<td>AICS</td>
<td>Australian Inventory of Chemical Substances (NICNAS)</td>
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<td>APA</td>
<td>American Psychiatric Association</td>
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<td>APVMA</td>
<td>Australian Pesticides and Veterinary Medicines Authority</td>
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<td>Australian Standard</td>
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<td>ATP</td>
<td>Air Toxics Program</td>
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<td>ATSDR</td>
<td>Agency for Toxic Substances and Disease Registry (US)</td>
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<td>AWPA</td>
<td>Australian Wood Panel Association</td>
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<tr>
<td>BMD</td>
<td>benchmark dose analysis</td>
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<td>BCA</td>
<td>Building Code of Australia</td>
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<tr>
<td>bw</td>
<td>bodyweight</td>
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<td>cDNA</td>
<td>complementary deoxyribonucleic acid</td>
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<td>CHO</td>
<td>Chinese hamster ovary</td>
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<tr>
<td>CI</td>
<td>Confidence interval</td>
</tr>
<tr>
<td>CICAD</td>
<td>Concise International Chemical Assessment Document</td>
</tr>
<tr>
<td>CIIT</td>
<td>Chemical Industry Institute of Toxicology</td>
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<tr>
<td>CNS</td>
<td>central nervous system</td>
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<td>CSIRO</td>
<td>Commonwealth Scientific and Industrial Research Organisation</td>
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<td>DEH</td>
<td>Australian Government Department of the Environment and Heritage</td>
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<tr>
<td>DMDM Hydantoin</td>
<td>1,3-dihydroxymethyl-5, 5-dimethyl hydantoin</td>
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<tr>
<td>DNA</td>
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<td>DNPH</td>
<td>dinitrophenylhydrazine</td>
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<td>DPX</td>
<td>DNA protein cross-linking</td>
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<td>EA</td>
<td>Environment Australia (former name of the Australian Government Department of the Environment and Heritage)</td>
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<tr>
<td>EASE</td>
<td>estimation and assessment of substance exposure</td>
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<tr>
<td>EC</td>
<td>European Community, or European Commission</td>
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<tr>
<td>EC50</td>
<td>median effective concentration</td>
</tr>
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<td>ECETOC</td>
<td>European Centre for Ecotoxicology and Toxicology of Chemicals</td>
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<td>EHC</td>
<td>Environmental Health Criteria</td>
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<td>EINECS</td>
<td>European Inventory of Existing Commercial Chemical Substances</td>
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<td>EL50</td>
<td>effective loading rate resulting in 50% effect</td>
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<td>EU</td>
<td>European Union</td>
</tr>
<tr>
<td>FDR</td>
<td>fecundability density ratio</td>
</tr>
<tr>
<td>FEFR</td>
<td>Forced expiratory flowrate</td>
</tr>
<tr>
<td>FEV_{1.0}</td>
<td>forced expiratory volume in one second</td>
</tr>
<tr>
<td>FID</td>
<td>Flame ionisation detection</td>
</tr>
<tr>
<td>g</td>
<td>gram</td>
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<tr>
<td>GC</td>
<td>gas-chromatography</td>
</tr>
<tr>
<td>GC-MS</td>
<td>gas-chromatography/mass spectrometry</td>
</tr>
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<td>GHS</td>
<td>Globally Harmonised System for Health and Environmental Hazard Classification and Communication</td>
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<tr>
<td>GLP</td>
<td>good laboratory practice</td>
</tr>
<tr>
<td>h</td>
<td>hour</td>
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<tr>
<td>HAP</td>
<td>hazardous air pollutant</td>
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<tr>
<td>HPLC</td>
<td>high-performance liquid chromatography</td>
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<td>HPV</td>
<td>high production volume</td>
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<td>Full Form</td>
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<tr>
<td>HSIS</td>
<td>Hazardous Substances Information System</td>
</tr>
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<td>HUD</td>
<td>US Department of Housing and Urban Development</td>
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<td>HVICL</td>
<td>High Volume Industrial Chemical List</td>
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<td>IARC</td>
<td>International Agency for Research on Cancer</td>
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<tr>
<td>IC(NA) Act</td>
<td><em>Industrial Chemicals (Notification and Assessment) Act 1989 (Cwlth)</em></td>
</tr>
<tr>
<td>IDLH</td>
<td>immediately dangerous to life and health</td>
</tr>
<tr>
<td>IgE</td>
<td>immunoglobulin-E</td>
</tr>
<tr>
<td>ip</td>
<td>Intraperitoneal</td>
</tr>
<tr>
<td>IPCS</td>
<td>International Programme on Chemical Safety</td>
</tr>
<tr>
<td>IUPAC</td>
<td>International Union of Pure and Applied Chemistry</td>
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<tr>
<td>ISO</td>
<td>International Organization for Standardization</td>
</tr>
<tr>
<td>kcal</td>
<td>kilocalorie</td>
</tr>
<tr>
<td>kg</td>
<td>kilogram</td>
</tr>
<tr>
<td>Koc</td>
<td>organic carbon partition coefficient</td>
</tr>
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<td>Kow</td>
<td>octanol/water partition coefficient</td>
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<td>kPa</td>
<td>kilo Pascal</td>
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<tr>
<td>L</td>
<td>litre</td>
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<tr>
<td>LC50</td>
<td>median lethal concentration</td>
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<td>LD50</td>
<td>median lethal dose</td>
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<tr>
<td>LEV</td>
<td>local exhaust ventilation</td>
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<tr>
<td>LOAEC</td>
<td>lowest-observed-adverse-effect concentration</td>
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<td>LOAEL</td>
<td>lowest-observed-adverse-effect level</td>
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<td>LOEC</td>
<td>lowest-observed-effect concentration</td>
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<td>LOEL</td>
<td>lowest-observed-effect level</td>
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<td>LVL</td>
<td>laminated veneer lumber</td>
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<td>m</td>
<td>metre</td>
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<td>MAK</td>
<td>maximale arbeitsplatz-konzentration (maximum workplace concentration)</td>
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<tr>
<td>MCOD</td>
<td>multiple cause of death</td>
</tr>
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<td>MDF</td>
<td>medium density fibreboard</td>
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<td>MEL</td>
<td>Maximum exposure limit</td>
</tr>
<tr>
<td>mg</td>
<td>milligram</td>
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<tr>
<td>mg/kg bw/d</td>
<td>milligram per kilogram bodyweight per day</td>
</tr>
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<td>min</td>
<td>minute</td>
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<td>mL</td>
<td>millilitre</td>
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<tr>
<td>MN</td>
<td>micronucleation</td>
</tr>
<tr>
<td>mRR</td>
<td>meta-relative risk</td>
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<tr>
<td>MS</td>
<td>mass spectrometry</td>
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<td>MSDS</td>
<td>material safety data sheet</td>
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<td>MUF</td>
<td>melamine urea formaldehyde</td>
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<td>MUPF</td>
<td>melamine urea phenol formaldehyde resins</td>
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<td>NA</td>
<td>not available</td>
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<td>NDPSC</td>
<td>National Drugs and Poisons Schedule Committee</td>
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<td>NEPM</td>
<td>National Environmental Protection Measure</td>
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<td>NHMRC</td>
<td>National Health and Medical Research Council</td>
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<td>NICNAS</td>
<td>National Industrial Chemicals Notification and Assessment Scheme</td>
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<td>no-observed-effect level</td>
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<td>NPI</td>
<td>National Pollutant Inventory</td>
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<td>NSW</td>
<td>New South Wales</td>
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<td>NTP</td>
<td>National Toxicology Program</td>
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<td>NOHSC</td>
<td>National Occupational Health and Safety Commission</td>
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<td>Acronym</td>
<td>Description</td>
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<tr>
<td>OASCC</td>
<td>Office of the Australian Safety and Compensation Council</td>
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<td>OECD</td>
<td>Organisation for Economic Cooperation and Development</td>
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<td>OHS</td>
<td>occupational health and safety</td>
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<tr>
<td>OR</td>
<td>odds ratio</td>
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<td>OSB</td>
<td>oriented strand board</td>
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<td>OSHA</td>
<td>Occupational Safety and Health Administration (USA)</td>
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<tr>
<td>P</td>
<td>p value</td>
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<td>P_{trend}</td>
<td>p value for trend analysis</td>
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<td>PAA</td>
<td>Plywood Association of Australia</td>
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<tr>
<td>PACIA</td>
<td>Plastics and Chemicals Industry Association</td>
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<td>Pb</td>
<td>particleboard</td>
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<td>PCMR</td>
<td>proportionate cancer mortality ratio</td>
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<td>PEC</td>
<td>predicted environmental concentration</td>
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<td>PEL</td>
<td>permissible exposure limit</td>
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<td>PEFR</td>
<td>peak expiratory flow rate</td>
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<td>PMR</td>
<td>proportionate mortality ratio</td>
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<td>PNEC</td>
<td>predicted-no-effect concentration</td>
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<tr>
<td>ppb</td>
<td>parts per billion</td>
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<td>PPE</td>
<td>personal protective equipment</td>
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<tr>
<td>ppm</td>
<td>parts per million</td>
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<td>ppt</td>
<td>Parts per trillion</td>
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<td>QLD</td>
<td>Queensland</td>
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<td>RD50</td>
<td>depression of the respiratory rate by 50%</td>
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<td>REL</td>
<td>reference exposure level</td>
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<td>RR</td>
<td>relative risks</td>
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<td>RTECS</td>
<td>Registry of Toxic Effects of Chemical Substances (US)</td>
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<td>SCE</td>
<td>sister chromatid exchange</td>
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<td>Abbreviation</td>
<td>Full Form</td>
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<td>SIAR</td>
<td>SIDS Initial Assessment Report</td>
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<td>Screening Information Data Set</td>
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<td>SMRs</td>
<td>standardised mortality ratios</td>
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<td>STEL</td>
<td>short-term exposure limit</td>
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<td>SUSDP</td>
<td>Standard for the Uniform Scheduling of Drugs and Poisons</td>
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<tr>
<td>TLV</td>
<td>Threshold Limit Value</td>
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<td>TWA</td>
<td>time-weighted average</td>
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<td>UF</td>
<td>urea formaldehyde resin</td>
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<td>UFFI</td>
<td>urea formaldehyde foam insulation</td>
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<td>UK HSE</td>
<td>United Kingdom Health and Safety Executive</td>
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<td>°</td>
<td>degree</td>
</tr>
<tr>
<td>µg</td>
<td>microgram</td>
</tr>
</tbody>
</table>
## Glossary

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute exposure</td>
<td>A contact between an agent and a target occurring over a short period of time, generally less than a day. (Other terms such as “short-term exposure” and “single dose” are also used.)</td>
</tr>
<tr>
<td>Adverse effect</td>
<td>Change in the morphology, physiology, growth, development, reproduction, or life span of an organism, system or (sub) population that results in an impairment of functional capacity, an impairment of the capacity to compensate for additional stress, or an increase in susceptibility to other influences.</td>
</tr>
<tr>
<td>Agent</td>
<td>A chemical, biological, or physical entity that contacts a target.</td>
</tr>
<tr>
<td>Analysis</td>
<td>Detailed examination of anything complex, made in order to understand its nature or to determine its essential features</td>
</tr>
<tr>
<td>Assessment</td>
<td>Evaluation of appraisal of an analysis of facts and the inference of possible consequences concerning a particular object or process.</td>
</tr>
<tr>
<td>Assessment endpoint</td>
<td>Quantitative/qualitative expression of a specific factor with which a risk may be associated as determined through an appropriate risk assessment.</td>
</tr>
<tr>
<td>Background level</td>
<td>The amount of an agent in a medium (e.g., water, soil) that is not attributed to the source(s) under investigation in an exposure assessment. Background level(s) can be naturally occurring or the result of human activities. (Note: natural background is the concentration of an agent in a medium that occurs naturally or is not the result of human activities).</td>
</tr>
<tr>
<td>Biomarker/biological marker</td>
<td>Indicator of changes or events in biological systems. Biological markers of exposure refer to cellular, biochemical, analytical, or molecular measures that are obtained from biological media such as tissues, cells, or fluids and are indicative of exposure to an agent.</td>
</tr>
<tr>
<td>Bounding Estimate</td>
<td>An estimate of exposure, dose, or risk that is higher than that incurred by the person with the highest exposure, dose, or risk in the population being assessed. Bounding estimates are useful in developing statements that exposures, doses, or risks are &quot;not greater than&quot; the estimated value.</td>
</tr>
<tr>
<td>Chronic exposure</td>
<td>A continuous or intermittent long-term contact between an agent and a target. (Other terms, such as “long-term exposure,” are also used.)</td>
</tr>
<tr>
<td>Concentration</td>
<td>Amount of a material or agent dissolved or contained in unit quantity in a given medium or system.</td>
</tr>
<tr>
<td>Contact volume</td>
<td>A volume containing the mass of agent that contacts the exposure surface</td>
</tr>
<tr>
<td>Term</td>
<td>Definition</td>
</tr>
<tr>
<td>-----------------------------</td>
<td>----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Dose</td>
<td>Total amount of an agent administered to, taken up or absorbed by an organism, system or (sub) population.</td>
</tr>
<tr>
<td>Dose-effect relationship</td>
<td>Relationship between the total amount of an agent administered to, taken up or absorbed by an organism, system or (sub) population and the magnitude of a continuously-graded effect to that organism, system or (sub) population. Related terms: Effect Assessment, Dose-Response Relationship, Concentration-Effect Relationship.</td>
</tr>
<tr>
<td>Dose-related effect</td>
<td>Any effect to an organism, system or (sub) population as a result of the quantity of an agent administered to, taken up or absorbed by that organism, system or (sub) population.</td>
</tr>
<tr>
<td>Dose-response</td>
<td>Relationship between the amount of an agent administered to, taken up or absorbed by an organism, system or (sub) population and the change developed in that organism, system or (sub) population in reaction to the agent. Synonymous with Dose-response relationship. Related Term: Dose-Effect Relationship, Effect Assessment, Concentration-Effect Relationship.</td>
</tr>
<tr>
<td>Dose-response assessment</td>
<td>Analysis of the relationship between the total amount of an agent administered to, taken up or absorbed by an organism, system or (sub)population and the changes developed in that organism, system or (sub)population in reaction to that agent, and inferences derived from such an analysis with respect to the entire population. Dose-Response Assessment is the second of four steps in risk assessment. Related terms: Hazard Characterisation, Dose-Effect Relationship, Effect Assessment, Dose-Response Relationship, Concentration-Effect Relationship.</td>
</tr>
<tr>
<td>Dose-response curve</td>
<td>Graphical presentation of a dose-response relationship.</td>
</tr>
<tr>
<td>Dose-Response Relationship</td>
<td>Relationship between the amount of an agent administered to, taken up or absorbed by an organism, system or (sub) population and the change developed in that organism, system or (sub) population in reaction to the agent. Related Terms: Dose-Effect Relationship, Effect Assessment, Concentration-Effect Relationship.</td>
</tr>
<tr>
<td>Effect</td>
<td>Change in the state or dynamics of an organism, system or (sub) population caused by the exposure to an agent.</td>
</tr>
<tr>
<td>Effect assessment</td>
<td>Combination of analysis and inference of possible consequences of the exposure to a particular agent based on knowledge of the dose-effect relationship associated with that agent in a specific target organism, system or (sub) population.</td>
</tr>
<tr>
<td>Expert judgement</td>
<td>Opinion of an authoritative person on a particular subject.</td>
</tr>
<tr>
<td>Exposure</td>
<td>Concentration or amount of a particular agent that reaches a target organism, system or (sub) population in a specific frequency for a defined duration.</td>
</tr>
<tr>
<td>Exposure assessment</td>
<td>Evaluation of the exposure of an organism, system or (sub) population to an agent (and its derivatives). Exposure Assessment is the third step in the process of Risk Assessment.</td>
</tr>
<tr>
<td>Exposure concentration</td>
<td>The exposure mass divided by the contact volume or the exposure mass divided by the mass of contact volume depending on the medium.</td>
</tr>
<tr>
<td>Exposure duration</td>
<td>The length of time over which continuous or intermittent contacts occur between an agent and a target. For example, if an individual is in contact with an agent for 10 minutes a day, for 300 days over a one-year time period, the exposure duration is one year.</td>
</tr>
<tr>
<td>Exposure frequency</td>
<td>The number of exposure events in an exposure duration.</td>
</tr>
<tr>
<td>Exposure mass</td>
<td>The amount of agent present in the contact volume. For example, the total mass of residue collected with a skin wipe sample over the entire exposure surface is an exposure mass.</td>
</tr>
<tr>
<td>Exposure model</td>
<td>A conceptual or mathematical representation of the exposure process.</td>
</tr>
<tr>
<td>Exposure pathway</td>
<td>The course an agent takes from the source to the target.</td>
</tr>
<tr>
<td>Exposure period</td>
<td>The time of continuous contact between an agent and a target.</td>
</tr>
<tr>
<td>Exposure route</td>
<td>The way an agent enters a target after contact (e.g., by ingestion, inhalation, or dermal absorption).</td>
</tr>
<tr>
<td>Exposure scenario</td>
<td>A set of conditions or assumptions about sources, exposure pathways, amount or concentrations of agent(s) involved, and exposed organism, system or (sub) population (i.e. numbers, characteristics, habits) used to aid in the evaluation and quantification of exposure(s) in a given situation.</td>
</tr>
<tr>
<td>Exposure surface</td>
<td>A surface on a target where an agent is present. Examples of outer exposure surfaces include the exterior of an eyeball, the skin surface, and a conceptual surface over the nose and open mouth. Examples of inner exposure surfaces include the gastro-intestinal tract, the respiratory tract and the urinary tract lining. As an exposure surface gets smaller, the limit is an exposure point.</td>
</tr>
<tr>
<td>Fate</td>
<td>Pattern of distribution of an agent, its derivatives or metabolites in an organism, system, compartment or (sub) population of concern as a result of transport, partitioning, transformation or degradation.</td>
</tr>
<tr>
<td>Term</td>
<td>Definition</td>
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</tr>
<tr>
<td>Guidance value</td>
<td>Value, such as concentration in air or water, which is derived after allocation of the reference dose among the different possible media (routes) of exposure. The aim of the guidance value is to provide quantitative information from risk assessment to the risk managers to enable them to make decisions. (See also: reference dose)</td>
</tr>
<tr>
<td>Hazard</td>
<td>Inherent property of an agent or situation having the potential to cause adverse effects when an organism, system or (sub) population is exposed to that agent.</td>
</tr>
<tr>
<td>Hazard assessment</td>
<td>A process designed to determine the possible adverse effects of an agent or situation to which an organism, system or (sub) population could be exposed. The process includes hazard identification and hazard characterization. The process focuses on the hazard in contrast to risk assessment where exposure assessment is a distinct additional step.</td>
</tr>
<tr>
<td>Hazard characterization</td>
<td>The qualitative and, wherever possible, quantitative description of the inherent properties of an agent or situation having the potential to cause adverse effects. This should, where possible, include a dose-response assessment and its attendant uncertainties. Hazard Characterisation is the second stage in the process of Hazard Assessment, and the second step in Risk Assessment. Related terms: Dose-Effect Relationship, Effect Assessment, Dose-Response Relationship, Concentration-Effect Relationship.</td>
</tr>
<tr>
<td>Hazard identification</td>
<td>The identification of the type and nature of adverse effects that an agent has inherent capacity to cause in an organism, system or (sub) population. Hazard identification is the first stage in hazard assessment and the first step in process of Risk Assessment.</td>
</tr>
<tr>
<td>Intake</td>
<td>The process by which an agent crosses an outer exposure surface of a target without passing an absorption barrier, i.e. through ingestion or inhalation.</td>
</tr>
<tr>
<td>Measurement of end-point</td>
<td>Measurable (ecological) characteristic that is related to the valued characteristic chosen as an assessment point.</td>
</tr>
<tr>
<td>Medium</td>
<td>Material (e.g., air, water, soil, food, consumer products) surrounding or containing an agent.</td>
</tr>
<tr>
<td>Microenvironment</td>
<td>The rate at which the medium crosses the outer exposure surface of a target, during ingestion or inhalation.</td>
</tr>
<tr>
<td>Reference dose</td>
<td>An estimate of the daily exposure dose that is likely to be without deleterious effect even if continued exposure occurs over a lifetime. Related term: Acceptable Daily Intake.</td>
</tr>
<tr>
<td>Response</td>
<td>Change developed in the state or dynamics of an organism, system or (sub) population in reaction to exposure to an agent.</td>
</tr>
<tr>
<td><strong>Risk</strong></td>
<td>The probability of an adverse effect in an organism, system or (sub)population caused under specified circumstances by exposure to an agent.</td>
</tr>
<tr>
<td>-----------</td>
<td>----------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td><strong>Risk analysis</strong></td>
<td>A process for controlling situations where an organism, system or (sub)population could be exposed to a hazard. The Risk Analysis process consists of three components: risk assessment, risk management and risk communication.</td>
</tr>
<tr>
<td><strong>Risk assessment</strong></td>
<td>A process intended to calculate or estimate the risk to a given target organism, system or (sub)population, including the identification of attendant uncertainties, following exposure to a particular agent, taking into account the inherent characteristics of the agent of concern as well as the characteristics of the specific target system. The Risk Assessment process includes four steps: hazard identification, hazard characterization (related term: dose-response assessment), exposure assessment, and risk characterization. It is the first component in a risk analysis process.</td>
</tr>
<tr>
<td><strong>Risk characterization</strong></td>
<td>The qualitative and, wherever possible, quantitative determination, including attendant uncertainties, of the probability of occurrence of known and potential adverse effects of an agent in a given organism, system or (sub)population, under defined exposure conditions. Risk Characterization is the fourth step in the Risk Assessment process.</td>
</tr>
<tr>
<td><strong>Risk communication</strong></td>
<td>Interactive exchange of information about (health or environmental) risks among risk assessors, managers, news media, interested groups and the general public.</td>
</tr>
<tr>
<td><strong>Risk estimation</strong></td>
<td>Quantification of the probability, including attendant uncertainties, that specific adverse effects will occur in an organism, system or (sub)population due to actual or predicted exposure.</td>
</tr>
<tr>
<td><strong>Risk evaluation</strong></td>
<td>Establishment of a qualitative or quantitative relationship between risks and benefits of exposure to an agent, involving the complex process of determining the significance of the identified hazards and estimated risks to the system concerned or affected by the exposure, as well as the significance of the benefits brought about by the agent. It is an element of risk management. Risk Evaluation is synonymous with Risk-Benefit evaluation.</td>
</tr>
<tr>
<td><strong>Risk management</strong></td>
<td>Decision-making process involving considerations of political, social, economic, and technical factors with relevant risk assessment information relating to a hazard so as to develop, analyse, and compare regulatory and non-regulatory options and to select and implement appropriate regulatory response to that hazard. Risk management comprises three elements: risk evaluation; emission and exposure control; risk monitoring.</td>
</tr>
<tr>
<td><strong>Risk monitoring</strong></td>
<td>Process of following up the decisions and actions within risk management in order to ascertain that risk containment or reduction with respect to a particular hazard is assured. Risk monitoring is an element of risk management.</td>
</tr>
<tr>
<td>Safety</td>
<td>Practical certainty that adverse effects will not result from exposure to an agent under defined circumstances. It is the reciprocal of risk.</td>
</tr>
<tr>
<td>Safety factor</td>
<td>Composite (reductive) factor by which an observed or estimated no-observed-adverse effect level (NOAEL) is divided to arrive at a criterion or standard that is considered safe or without appreciable risk. Related terms: Assessment Factor, Uncertainty Factor.</td>
</tr>
<tr>
<td>Source</td>
<td>The origin of an agent for the purposes of an exposure assessment.</td>
</tr>
<tr>
<td>Subchronic exposure</td>
<td>A contact between an agent and a target of intermediate duration between acute and chronic. (Other terms, such as “less-than-lifetime exposure” are also used.)</td>
</tr>
<tr>
<td>Target</td>
<td>Any biological entity that receives an exposure or a dose (e.g., a human, human population or a human organ).</td>
</tr>
<tr>
<td>Threshold</td>
<td>Dose or exposure concentration of an agent below that a stated effect is not observed or expected to occur.</td>
</tr>
<tr>
<td>Time-averaged exposure</td>
<td>The time-integrated exposure divided by the exposure duration. An example is the daily average exposure of an individual to carbon monoxide. (Also called time-weighted average exposure.)</td>
</tr>
<tr>
<td>Tolerable daily intake</td>
<td>Analogous to Acceptable Daily Intake. The term Tolerable is used for agents which are not deliberately added such as contaminants in food.</td>
</tr>
<tr>
<td>Toxicity</td>
<td>Inherent property of an agent to cause an adverse biological effect.</td>
</tr>
<tr>
<td>Uncertainty</td>
<td>Imperfect knowledge concerning the present or future state of an organism, system or (sub) population under consideration.</td>
</tr>
<tr>
<td>Uncertainty factor</td>
<td>Reductive factor by which an observed or estimated no-observed-adverse-effect level (NOAEL) is divided to arrive at a criterion or standard that is considered safe or without appreciable risk. Related terms: Assessment Factor, Safety Factor.</td>
</tr>
</tbody>
</table>
1. Introduction

1.1 Declaration

The chemical formaldehyde (CAS No 50-00-0) was declared a Priority Existing Chemical for full assessment under the Industrial Chemicals (Notification and Assessment) Act 1989 (the Act) on 5 March 2002. It was nominated by the public, unions and non-government organisations for assessment due to its adverse effects and widespread use. In addition, there were indications of a need to review the occupational exposure standard and develop a National Environmental Protection Measure (NEPM) for formaldehyde.

1.2 Objectives

The objectives of this assessment were to:

- characterise the properties of formaldehyde;
- determine the uses of formaldehyde in Australia;
- determine the extent of environmental, public and occupational exposure to formaldehyde;
- characterise the intrinsic capacity of formaldehyde to cause adverse effects on humans and the environment;
- characterise the risk to humans and the environment resulting from exposure to formaldehyde; and
- determine the extent to which any risk can be minimised.

1.3 Sources of information

Information for the assessment was obtained from various sources including industry, literature searches, site visits, all levels of governments, and other organisations, such as research institutes and overseas regulatory authorities.

Industry

In accordance with the Act, manufacturers and importers of formaldehyde were required to apply for assessment and supply relevant information. Data supplied by applicants included:

- quantity of the chemical and/or products containing the chemical manufactured and/or imported;
- quantity of the chemical formulated into products;
- uses of the chemical and products containing the chemical;
- methods used in handling, storing, manufacturing and disposal of the chemical and products containing the chemical;
- information on human and environmental exposure to the chemical;
Material Safety Data Sheet (MSDS) and labels; and

contact details of their customers.

The National Industrial Chemical Notification and Assessment Scheme (NICNAS) conducted a questionnaire survey (the NICNAS survey) in October 2002 to investigate the use patterns, occupational exposure levels, control technologies and environmental exposure to formaldehyde. Randomly selected formulators and end users of formaldehyde products participated in the NICNAS survey. Further details are provided in Section 7.3.

A number of industry associations were also contacted and provided relevant information. A list of all companies, associations and individuals consulted during this assessment is provided in Appendix 1.

**Literature review**

A number of overseas peer-reviewed assessment reports on formaldehyde are available (see Section 2.4). The major source of information on the health effects of formaldehyde for this assessment was the Concise International Chemical Assessment Document (CICAD) for formaldehyde, published under the International Programme on Chemical Safety (IPCS, 2002). To enhance the efficiency of the NICNAS assessment and provide transparency, not all primary sources of data in the CICAD were evaluated. However, relevant studies published since the cited reviews were identified (up to July 2004) and assessed on an individual basis.

**Site visits**

Information on methods of use and potential for workers’ exposure was also obtained through a number of site visits. The site visits included formaldehyde and formaldehyde resin manufacturers, a wood panel plant, funeral homes, pathological laboratories and film processing plants.

**1.4 Peer review**

During all stages of preparation, the report has been subject to internal peer review by NICNAS and the Australian Government Department of the Environment and Heritage (DEH). Selected parts of the report were also externally peer reviewed by independent experts from Australia and overseas.
2. Background

2.1 Introduction

Formaldehyde is a naturally occurring, volatile organic compound which is ubiquitous in the environment. It is formed primarily by the combustion of organic materials and by a variety of natural and anthropogenic activities.

Formaldehyde is the product of many natural processes, such as forest and bush fires, animal wastes, microbial products of biological systems, and plant volatiles. In water, it is also formed by the irradiation of humic substances by sunlight. As a metabolic intermediate, formaldehyde is present at low levels in most living organisms. It is emitted by bacteria, algae, plankton, and vegetation as well.

Anthropogenic sources of formaldehyde from combustion processes account directly or indirectly for most of the formaldehyde entering the environment. Direct combustion sources include power plants, incinerators, refineries, wood stoves, kerosene heaters, and cigarettes. Formaldehyde is also produced indirectly by photochemical oxidation of hydrocarbons or other formaldehyde precursors that are released from combustion processes. Other anthropogenic sources of formaldehyde in the environment include industrial on-site uses and off-gassing from building materials and consumer products.

Secondary formation of formaldehyde occurs in the atmosphere through the photochemical oxidation of natural and anthropogenic volatile organic compounds in the air, such as methane, isoprene, and pollutants from mobile and stationary sources, such as alkanes, alkenes, aldehydes and alcohols.

2.2 Global production

Since 1889 in Germany, formaldehyde has been produced commercially by the catalytic oxidation of methanol. Various manufacturing methods were used in the past, but only two are widely used today: the silver catalyst and metal oxide catalyst processes (IARC, 1995). Formaldehyde is used predominately in the production of resins, followed by fertilizer production, and for various other purposes, such as preservatives and disinfectants. Formaldehyde can be used in a variety of industries, including the medical, detergent, cosmetics, food, rubber, metal, wood, leather, petroleum, and agricultural industries, and as a hydrogen sulfide scavenger in oil operations.

Because of its low cost and high purity, formaldehyde has become one of the most important industrial and research chemicals in the world. The global production of formaldehyde in 1999 (the most recent figure) was estimated 5 to 6 million tonnes (Asia: 1 to 1.5 million tonnes, North America: 1 to 1.5 million tonnes, Western Europe: 2 to 2.5 million tonnes) (OECD, 2002). A global production figure of 12 million tonnes in 1992 was reported by IARC (1995). Formaldehyde is listed on the Organisation for Economic Cooperation and Development’s (OECD) List of High Production Volume (HPV) chemicals, i.e. production volume of 1000 tonnes or more in at least one OECD country (OECD, 2004).
2.3 Australian perspective

In Australia, consistent with overseas use, formaldehyde is mainly used in the manufacture of formaldehyde-based resins, which are widely used in a variety of industries, predominately the wood industry. Formaldehyde is on the 2003 Australian High Volume Industrial Chemical List (HVICL) compiled by NICNAS (NICNAS, 2002), which means it is an industrial chemical that had a combined annual import and manufacturing quantity of 1000 tonnes or more during 2001-2002. The total quantity of formaldehyde manufactured and imported is detailed in Sections 7.1 and 7.2.

Concerns have been expressed by the public and several organisations over its widespread use and adverse health effects, including its sensitisation potential and carcinogenicity.

Formaldehyde is listed in the OASCC’s Hazardous Substances Information System (DEWR, 2004) and in Schedules 2 and 6 of the Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) (NDPSC, 2005). It is also listed in the Australian Code for the Transport of Dangerous Goods by Road and Rail (FORS, 1998) as a dangerous good. An Australian occupational exposure standard for formaldehyde has been established (DEWR, 2004).

2.4 Assessments by other national or international bodies

Formaldehyde has been assessed by several national and international bodies, who have reviewed and evaluated data pertaining to the health and/or environmental hazards posed by the chemical. Of these, the most noteworthy are:

- International Agency for Research on Cancer examined a number of recent epidemiology studies on carcinogenicity (IARC, 2004a). It concluded that the carcinogen classification for formaldehyde be upgraded from probable human carcinogen (Category 2A) to known human carcinogen (Category 1) based on evidence that exposure to formaldehyde may cause nasopharyngeal cancer in humans (more details in Section 11.6). IARC has also reviewed formaldehyde on a number of previous occasions (IARC, 1987, 1995);

- Concise International Chemical Assessment Document (CICAD) No. 40: Formaldehyde, published by the International Programme on Chemical Safety (IPCS, 2002);

- A Screening Information Data Set (SIDS) Initial Assessment Report (SIAR) prepared by the German BMU (Bundesministerium für Umwelt, Naturschutz und Reaktorsicherheit) was agreed at the Organization for Economic Cooperation and Development (OECD) 14th SIDS Initial Assessment Meeting (SIAM) in March 2002 (OECD, 2002). It concluded that further work on the environmental exposure assessment was needed;

- US Agency for Toxic Substances and Disease Registry (ATSDR) report (ATSDR, 1999); and

3. Applicants

Following the declaration of formaldehyde as a Priority Existing Chemical, the following companies or organisations applied for assessment of this chemical.

In accordance with the *Industrial Chemicals (Notification and Assessment) Act 1989*, NICNAS provided the applicants with a draft copy of the report for comment during the correction and variation phases of the assessment. The applicants were as follows:

- **A.S. Harrison & Co Pty Ltd**
  PO Box W2
  Warringah Mall NSW 2100

- **ACE Chemical Company**
  119A Mooringe Avenue
  Camden Park SA 5038

- **Agent Sales and Services Pty Ltd**
  32 Charles St
  South Perth, WA 6151

- **AGFA-Gevaert Ltd**
  PO Box 48
  Nunawading VIC 3131

- **Akzo Nobel Pty Ltd**
  51 McIntyre Road
  Sunshine VIC 3020

- **Amtrade International Pty Ltd**
  PO Box 6421 St Kilda Road
  Central Post Office VIC 8008

- **Ashland Pacific Pty Ltd**
  PO Box 162
  Chester Hill NSW 2162

- **Asia Pacific Specialty Chemicals Ltd**
  PO Box 232
  Seven Hills NSW 1730

- **Australian Council of Trade Unions**
  393 Swanston Street
  Melbourne VIC 3000

- **Australian Plantation Products and Paper Industry Council**
  Level 3, Tourism House
  Barton, ACT 2600

- **Australian Wood Panels Association**
  33 Bambury St
  Fingal Head, NSW 2487

- **BASF Australia Ltd**
  Box 4705
  Melbourne VIC 3001

- **Bayer Australia, SP/CH Business Group**
  633-647 Springvale Rd
  Mulgrave North VIC 3170

- **BetzDearbon Australia**
  69-77 Williamson Rd
  Ingelburn NSW 2565

- **Biolab (Aust) Pty Ltd**
  2 Clayton Rd
  Clayton, VIC 3168

- **Bio Scientific Pty Ltd**
  PO Box 78
  Gymea NSW 2227

- **Campbell Brothers Ltd**
  PO Box 118
  Newport VIC 3015

- **Campbell Cleantec Ltd**
  PO Box 490
  Sumner Park BC QLD 4074
Canpoint International Pty Ltd
72 Tennyson Rd
Mortlake, NSW 2137

Carter Holt Harvey Panels
L6, Tower A, Zenith Centre
821 Pacific Highway
Chatswood, NSW 2067

CHT Australia Pty Ltd
33 Elliott Rd
Dandenong Vic 3175

Ciba Specialty Chemicals
235 Settlement Road
Thomastown VIC 3074

Clariant (Australia) Pty Ltd
PO Box 23
Chadstone VIC 3148

Colgate Palmolive Pty Ltd
GPO Box 3964
Sydney NSW 2001

Cytec Australia Holdings
PO Box 7215
Baulkham Hills BC NSW 2153

Du Pont (Australia) Ltd
49-59 Newtown Road
Wetherill Park NSW 2164

Dynea WA Pty Ltd
PO Box 1298
Bunbury WA 6231

Ecolab Pty Ltd
6 Hudson Avenue
Castle Hill NSW 2154

Gumfighters
Suite 68, 89-97 Jones St
Ultimo, NSW 2007

Gunnersen Timbermark Pty Ltd
112 Salmon St
Port Melbourne, VIC 3207

H B Fuller Company
Australia Pty Ltd
PO Box 4202
Dandenong South, VIC 3164

H Treval & Son Pty Ltd
157 Kingsgrove Rd
Kingsgrove, NSW 2208

Halex Flooring Products Pty Ltd
2/73 Zenith Rd
Dandenong VIC 3175

Hexion Specialty Chemicals Pty Ltd
2-8 James Street
Laverton North VIC 3026

ISP (Australasia) Pty Ltd
PO Box 6564
Silverwater NSW 1811

International Sales and Marketing Pty Ltd
262 Highett Road
Highett VIC 3190

International Trade Strategies Pty Ltd
Level 2, 60 Collins St
Melbourne, Vic 3000

Jayco Corporation Pty Ltd
252-254 Frankston-Dandenong Rd
Dandenong, Vic 3175

Kodak (Australasia) Pty Ltd
PO Box 90
Coburg VIC 3058

Lomb Scientific (Aust) Pty Ltd
PO Box 2223
Taren Point NSW 2229

Manildra Flour Mills (Manufacturing) Pty Ltd
PO Box 72
Auburn, NSW 2144

Merck Pty Ltd
207 Colchester Rd
Kilsyth VIC 3137

Novek Synthetics
102a Winbourne Rd
Hazelbrook, NSW 2779
Nowra Chemical Manufacturers Pty Ltd
112 Albatross Rd
Nowra 2541

Nuplex Industries (Aust) Pty Ltd
49-61 Stephen Road
Botany NSW 2019

Orica Australia Pty Ltd
1 Nicholson Street
Melbourne VIC 3001

PCA Hodgson Chemicals Pty Ltd
19-25 Anne Street
St Mary NSW 2760

Plywood Association of Australia
13 Dunlop St
Newstead, QLD 4006

Professional Compounding Chemists of Australia Pty Ltd
Suite 2, 1371 Botany Rd
Botany, NSW 2019

ProSciTech
PO Box 111
Thuringowa QLD 4817

Redox Chemicals Pty Ltd
Locked Bag 60
Wetherill Park NSW 2164

RH Minter Pty Ltd
17 Park Road
Oakleigh VIC 3166

Sigma Aldrich Pty Ltd
2/14 Anella Ave
Castle Hill, NSW 2154

Standards Australia
GPO Box 5420
Sydney NSW 2001

Sulzer Medica Pty Ltd
Level 5, 384 Eastern Valley Way
Chatswood, NSW 2067

Swift and Company Ltd
PO Box 689
Mulgrave VIC 3170

The Structural Adhesive Company Pty Ltd
116 Kitchener Rd
Ascot, QLD 4007

Thor Specialties
GPO Box 3124
Wetherill Park NSW 2164

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4. Chemical Identity and Composition

4.1 Chemical name (IUPAC)
Methanal

4.2 Registry numbers
Formaldehyde is listed on the Australian Inventory of Chemical Substances (AICS) as formaldehyde.
- CAS number: 50-00-0
- EINECS number: 200-001-8
- UN numbers: 2209 for non-flammable formaldehyde solutions ($\leq$25%) 1198 for flammable formaldehyde solutions

4.3 Other names
Formaldehyde solution
Formaldehyde gas
Formalin
Formalith
Formol
Formic aldehyde
Methaldehyde
Methyl aldehyde
Methylene oxide
Morbicid
Oxomethane
Oxymethylene
Paraform

4.4 Molecular formula
CH$_2$O
4.5 Structural formula

\[
\begin{align*}
\text{H} & \\
\text{|} & \\
\text{C = O} & \\
\text{|} & \\
\text{H}
\end{align*}
\]

4.6 Molecular weight

30.03

4.7 Composition of commercial grade product

Pure formaldehyde is not commercially available. Formaldehyde is generally available as a 37% to 54% (by weight) aqueous solution, known as formalin. To reduce the intrinsic polymerisation of formaldehyde, stabilisers, such as methanol and various amine derivatives, are added to the solution (IPCS, 2002; IARC, 1995). Methanol concentrations can be as high as 15% (by weight). The concentrations of other stabilisers can be in the order of several hundred mg/mL (IPCS, 1989). Formaldehyde is marketed in a solid form as trioxane (CH₂O)₃ and its polymer paraformaldehyde, with 8 to 100 units of formaldehyde (IPCS, 2002; IARC, 1995).
5. Physical and Chemical Properties

This section covers physical and chemical properties for both gaseous formaldehyde gas and formalin (37% formaldehyde solution).

5.1 Physical state

At room temperature, formaldehyde is a colourless gas with a pungent, irritating odour. The odour threshold of formaldehyde varies widely, ranging from 0.05 to 1 ppm. However, for most people the odour threshold is in the 0.5 to 1 ppm range (OECD, 2002).

5.2 Physical and chemical properties

The physical and chemical properties of gaseous formaldehyde and formalin (37% formaldehyde solution) are summarised in Table 5.1. The values in the following text and in Table 5.1 are cited from the CICAD (IPCS, 2002), unless otherwise stated.

Gaseous formaldehyde

Formaldehyde gas is highly reactive, highly flammable and can form explosive mixtures in air. It presents a fire hazard when exposed to flame or heat. At temperatures greater than 150°C, formaldehyde decomposes to methanol and carbon monoxide (IPCS, 1989). It readily undergoes polymerisation. Formaldehyde polymers or products containing formaldehyde polymers can decompose to release significant amounts of gaseous formaldehyde when overheated.

Formaldehyde gas is readily soluble in water, alcohol, and other polar solvents. It can exist as methylene glycol, polyoxymethylene and hemiformal in solutions. Formaldehyde is a reactive aldehyde that undergoes a number of self-association reactions. For example, at concentrations above 30% the polymer precipitates. The chemical species produced when formaldehyde associates with water may have different properties from those of the pure monomolecular substance. These associations tend to be more prevalent at higher concentrations of formaldehyde. Therefore, the properties described at high concentrations may not be relevant for more dilute concentrations.

Formalin

Formalin without methanol has a flash point of 83 to 85 °C and is combustible. Formalin can be a flammable liquid when the formaldehyde or methanol concentration is high. Formalin may become cloudy on standing, especially at cool temperatures, and form paraformaldehyde at very low temperatures. It slowly oxidizes in air to formic acid and is sensitive to light. It is easily hydrated and polymerised if not stabilised (Keith and Walters, 1992).
Formalin is a strong reducing agent, especially in the presence of alkalis. It is incompatible with ammonia, alkalis, bisulfides, iron preparations, iodine, phenols, potassium permanganate, tannin and salts of copper, iron, and silver. It combines directly with albumin, casein, gelatin, agar and starch to form insoluble compounds. It reacts violently with hydrogen peroxide, magnesium carbonate, nitromethane, perchloric acid and aniline, and performic acid and also reacts with strong oxidizers and acids. Reactions with nitrous oxides (nitrogen dioxide)
become explosive at 180 °C. It is corrosive to carbon steel as well as copper and its alloys (Keith and Walters, 1992).

Paraformaldehyde emits formaldehyde gas when it is heated to decomposition. It is also hydrolysed by hot water and alkali forming formaldehyde. It behaves like methanol-free formaldehyde of the same concentration once it dissolves in water (Lewis, 1996).

5.3 Conversion factors

The conversion factors for formaldehyde at 25 °C are:

1 ppm = 1.2 mg/m³
1 mg/m³ = 0.83 ppm
6. Methods of Detection and Analysis

6.1 Characterisation

Formaldehyde can be characterised by a number of methods including spectrophotometry, high performance liquid chromatography (HPLC), colorimetry, fluorimetry, polarography, gas chromatography (GC) using flame ionisation detection (FID), and infrared detection. Methods based on spectrophotometry are the most widely used, and have sensitivities of 8 to 25 ppb (10 to 30 $\times 10^{-6}$ g/m$^3$). HPLC is another method commonly used and has a detection limit of 1.7 ppb (2 $\times 10^{-6}$ g/m$^3$). The most sensitive method of detection is flow injection, with a detection limit of 9 ppt (0.011 $\times 10^{-6}$ g/m$^3$).

Information on methods of detection and analysis for formaldehyde in various media is abundant and has been summarised in a number of reviews (IPCS, 2002; ATSDR, 1999; IARC, 1995; IPCS, 1989). For all methods, organic and inorganic chemicals, such as sulphur dioxide, other aldehydes and amines, can cause interference. Therefore, the method of sampling and the treatment of the sample before analysis are important factors in the accuracy of the determination.

This section focuses on the methods commonly used in Australia for detecting formaldehyde in the atmosphere of workplaces, ambient air, indoor air and emissions from products releasing formaldehyde, such as wood and textiles. Methods of detection in other media, such as water and in biological samples, are also briefly discussed.

6.2 Atmospheric monitoring methods

6.2.1 In the workplace

For personal monitoring during full shifts or tasks, workers are equipped with a sampler (tube or badge) placed in the breathing zone. For area monitoring, the tube or badge is placed at a fixed location in the workplace environment. Tubes are connected to a portable metering pump, whereas badges sample the air by diffusion. At the end of the sampling period, the tube or badge is sealed and transferred to a laboratory, where the chemical is liberated from the absorbent and quantified using different analytical methods. The result is expressed as ppm or mg/m$^3$ over the duration of the sampling period. The analytical detection limit depends on the airflow across the absorbent and the duration of the sampling period.

The US National Institute of Occupational Safety and Health (NIOSH) methods (NIOSH, 1994) are commonly used in Australia. They are summarised in Table 6.1.
### Table 6.1: NIOSH methods of detection for formaldehyde (NIOSH, 1994)

<table>
<thead>
<tr>
<th>Method Number</th>
<th>Sampling method</th>
<th>Analytical method</th>
<th>Limit of detection</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>3500</td>
<td>Filter and impingers</td>
<td>Visible absorption spectrometry</td>
<td>0.02 ppm for an 80L air sample</td>
<td>The most sensitive method of the NIOSH methods; Best suited for static samples.</td>
</tr>
<tr>
<td>2541</td>
<td>Solid sorbent tube</td>
<td>GC, FID</td>
<td>0.24 ppm for an 10L air sample</td>
<td>Suitable for the simultaneous determinations of acrolein and formaldehyde; suited for personal samples.</td>
</tr>
<tr>
<td>2016</td>
<td>Cartridge</td>
<td>HPLC, UV detection</td>
<td>0.021 ppm for an 15L air sample</td>
<td>Can be used for both TWA and STEL measurements.</td>
</tr>
</tbody>
</table>

TWA, time weighted average; STEL, short-term exposure limit.

Several other atmospheric monitoring methods for detecting formaldehyde in the workplace are summarised in the CICAD (IPCS, 2002). These include some methods that have been used in Australia, such as use of a formaldehyde passive sampler/monitor followed by chromotropic acid test (detection limit of 0.083 ppm) and gas tube detectors with infrared analysers (detection limit of 0.33-0.42 ppm).

Instantaneous measurement of the concentration of airborne formaldehyde, such as by direct read, hand-held electronic formaldehyde devices, is also used in Australia. For example, formaldehyde meters and Interscan machines provide instantaneous readings.

The sensor of formaldehyde meters is an electro-chemical cell which contains electrodes that are used for temperature compensation and to improve the selectivity. The sensor response is linear with the concentration of formaldehyde in air. Two filters are used to eliminate interferences. Measurements are first made with a filter that permits determination of the background or baseline. Insertion of a second filter then permits the measurement of formaldehyde. The limit of detection is 0.01 ppm.

The Interscan machine is an electrochemical gas detector operating under diffusion-controlled conditions. Gas molecules from the sample are adsorbed on an electrocatalytic-sensing electrode, after passing through a diffusion medium, and are electrochemically reacted at an appropriate sensing electrode potential. This reaction generates an electric current directly proportional to the gas concentration. This current is converted to a voltage for meter or recorder readout. The limit of detection is 0.01 ppm.

### 6.2.2 In the environment

The methods commonly used for measuring the concentration of formaldehyde in ambient air fall into the following two categories (EA, 2001):
Discrete air sampling with subsequent laboratory analysis;  
Continuous or semi-continuous in-field analysis.

The most widely used method for discrete air sampling involves the collection of air into a stainless steel canister over a predetermined period of time, such as 24 hours, followed by GC or GC-MS analysis. Discrete sampling methods determine average pollutant levels over the sample collection time.

A commonly used continuous in-field analysis method uses an optical remote sensing system to determine the concentration of the chemical by means of the differential absorption of transmitted light by gaseous compounds along the light path. The system consists of a light transmitter and sensor placed at a given distance apart at the monitoring site. Alternatively, the concentration in air can be analysed by semi-continuous gas chromatography. Samples are collected directly onto solid absorbents, desorbed thermally onto the GC column and analysed while the next sample is collected. Compared with discrete sampling method, continuous or semi-continuous methods enable more detailed information about concentration variations.

The analytical limit of detection of the above methods typically ranges from 1.3 to 0.1 ppb. All of the methods allow for the simultaneous determination of several other gaseous air pollutants in the same sample.

In addition, several other methods of detection for measuring ambient air formaldehyde levels are available including:

- United States Environmental Protection Agency (US EPA), Method TO5, *Determination of Aldehydes and Ketones in Ambient Air Using High Performance Liquid Chromatography (HPLC)* (US EPA, 1988a);

A recent NEPM document (NEPC, 2004) recommended use of two other US EPA testing methods:

- US EPA Compendium Method TO-11A, *Determination of Formaldehyde in Ambient Air Using Adsorbent Cartridge Followed by High Performance Liquid Chromatography* (active sampling methodology) (US EPA, 1999a);

### 6.2.3 Indoor air

Formaldehyde concentrations in indoor air can be measured by either active or passive sampling using a sampler to collect the formaldehyde followed by analysis using a number of methods. The use of passive sampling techniques should be fully verified by active means.
Currently, there is an Australian Standard for testing formaldehyde in indoor air, AS 2363.6-1995, *Methods for the Sampling and Analysis of Indoor Air. Method 6: Determination of Formaldehyde –Impinger Sampling- Chromotropic Acid Method* (Standards Australia, 1995). However, there are problems with use of chromotropic acid due to interferences and quality-related issues. There are more suitable methods including active collection onto DNPH, which are analysed via HPLC or GC/MS or equivalent analytical methods. The US EPA methods discussed above (TO5, TO11, TO-11A, and TO-15) are also suitable for measuring indoor air formaldehyde.

There are a number of International Organization for Standardization (ISO) documents on indoor air formaldehyde testing. They are:

- ISO 16000-3 *Indoor air - part 3: Determination of formaldehyde & other carbonyl compounds - Active sampling method.* (based on US EPA method TO-11A) (ISO, 2001)
- ISO 16000-4 *Indoor air - part 4: Determination of formaldehyde - Diffusive sampling method. (i.e. passive sampling with badges)* (ISO, 2004b).

The Standards Australia Indoor Air Committee advised that the Committee would be considering these ISO methods along with other methods such as the US EPA methods when determining suitable testing methods for indoor air formaldehyde in the future.

Methodology for the simultaneous sampling of a number of indoor airborne aldehydes including formaldehyde is also available. A recent paper investigated detecting indoor air formaldehyde using a direct reading device (Suzuki, 2003). However, this method has certain limitations and serves mainly for screening purposes.

### 6.2.4 Off-gas monitoring from wood products

Four methods have been developed to measure formaldehyde emissions from wood products and details have been summarised in recent reviews (IPCS, 2002; IARC, 1995).

The Standards Australia has published a number of methods for the measurement of formaldehyde emission from particleboard, fibreboard and medium density fibreboard (MDF). A summary of these standards is provided in Table 6.2. Standard testing methods for formaldehyde emissions from plywood (AS/NZS 2098.11:2004) and laminated veneer limber (AS/NZS 4357.4:2004) products are currently being considered by the Standards Australia.
Table 6.2: Standards Australia methods for the measurement of formaldehyde emissions from wood-based products

<table>
<thead>
<tr>
<th>Method</th>
<th>Sampling Matrix</th>
<th>Principle</th>
<th>Emission Rate</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Desiccator</td>
<td>Particleboard and</td>
<td>Emission of formaldehyde is determined by placing test pieces of known surface area in a desiccator, at a controlled temperature, and measuring the quantity of emitted formaldehyde absorbed in a specific volume of water during 24 h using a spectrophotometer.</td>
<td>mg/L</td>
<td>AS/NZS 4266.16: 2004</td>
</tr>
<tr>
<td>method</td>
<td>fibreboard</td>
<td></td>
<td></td>
<td>(Standards Australia/New Zealand, 2004a)</td>
</tr>
<tr>
<td>Perforator</td>
<td>Particleboard and</td>
<td>Formaldehyde is extracted from test pieces by means of boiling toluene and then transferred into distilled or demineralised water. A sample of the water is then analysed photometrically by the acetylacetone method.</td>
<td>mg/100g</td>
<td>AS/NZS 4266.15: 1995</td>
</tr>
<tr>
<td>method</td>
<td>medium density fibreboard</td>
<td></td>
<td></td>
<td>(Standards Australia/New Zealand, 1995)</td>
</tr>
</tbody>
</table>

6.3 Biological monitoring

The concentration of formaldehyde in biological samples, such as blood and breath, has been used in attempts to monitor workers’ exposure (ATSDR, 1999). Formic acid or formate, a metabolite of formaldehyde, has been measured in workers’ urine and blood. However, it has been suggested exposure to formaldehyde cannot be adequately assessed by these methods because formaldehyde is rapidly metabolised and is highly reactive. Therefore, it is unlikely to be present in samples. Urinary formate levels are also an unreliable biomarker as formate is a metabolite of many other substances.

6.4 Water

Methods for the collection and determination of formaldehyde in atmospheric water, drinking water and fog water have been summarised by ATSDR (1999). These methods are similar to those for ambient air described above. The methods for formaldehyde in drinking water and fog water rely on the formation of the DNPH derivative followed by HPLC. The method for measuring formaldehyde in atmospheric water relies on the reaction of formaldehyde in atmospheric water with diketone (2,4-pentanediione) and ammonium acetate to form a fluorescent derivative that is measured spectrophotometrically in a flow injection analysis system.
6.5 Soil

One method for measuring formaldehyde in soil has been reported (Klasco, 2003). The soil is dried by addition of magnesium sulfate. Freon 113 is then used to extract the formaldehyde and the sample is scanned with a spectrophotometer. The concentration is determined from a calibration curve.
7. Manufacture, Importation and Use

7.1 Manufacture

Formaldehyde is manufactured in Australia by catalytic oxidation of methanol. Two methods are used; one uses a silver catalyst and the other a metal oxide catalyst. As formaldehyde is produced in gas form, it is absorbed into water during manufacture. The aqueous solutions are called formalin and the concentrations of formaldehyde in formalin range from 37% to 54%. Four companies manufacture formaldehyde at five sites around Australia. Information on the location of the plants, manufacturing techniques and the formaldehyde concentrations in formalin produced are summarised in Table 7.1.

Table 7.1: Manufacturers of formaldehyde in Australia

<table>
<thead>
<tr>
<th>Company</th>
<th>Location</th>
<th>State</th>
<th>Manufacture technique</th>
<th>% Formaldehyde in formalin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Woodchem</td>
<td>Oberon</td>
<td>NSW</td>
<td>Metal oxide catalyst</td>
<td>37</td>
</tr>
<tr>
<td>Orica</td>
<td>Deer Park</td>
<td>VIC</td>
<td>Metal oxide catalyst</td>
<td>54</td>
</tr>
<tr>
<td>Hexion</td>
<td>Laverton</td>
<td>VIC</td>
<td>Silver catalyst</td>
<td>54</td>
</tr>
<tr>
<td>Hexion</td>
<td>Gibson Island</td>
<td>QLD</td>
<td>Silver catalyst</td>
<td>50</td>
</tr>
<tr>
<td>Dynea</td>
<td>Dardanup</td>
<td>WA</td>
<td>Silver catalyst</td>
<td>37</td>
</tr>
</tbody>
</table>

Some manufacturers also dilute the 50% and 54% formalin to as low as 26% for use or sale.

The quantities of formaldehyde manufactured (calculated as 100% formaldehyde) for calendar years 2000 to 2002 are shown in Figure 7.1. The information was provided by the four manufacturers. Approximately 50 000 tonnes of formaldehyde are manufactured annually.

The formaldehyde manufacturers advised that over 80% of formalin production is used in resin manufacture on site. The remainder is supplied to local formulators or end users and small amounts are exported overseas.

Paraformaldehyde is not manufactured in Australia.

Manufacturing process

Formaldehyde manufacture involves a series of continuous, enclosed processes designed to facilitate the oxidation of methanol over a catalyst. The processes for the two manufacturing methods used in Australia are similar and are shown in Figure 7.2.
Figure 7.1: Quantities of formaldehyde manufacture in Australia

Figure 7.2: Formaldehyde manufacturing process
Raw materials used in formaldehyde manufacture are methanol, water, air and catalysts. Liquid methanol is fed into a vaporising chamber where it is mixed with water and air (oxygen). The contents of the chamber are maintained at a desired temperature range through the addition of steam. The vaporised methanol is then directed to the top of an exothermic reaction chamber. The reaction generates heat that is used to sustain the temperature of the catalyst and generate steam for use in resin manufacture. Hot gaseous formaldehyde is cooled as it exits the reaction chamber. It is then passed to absorption towers where formaldehyde is absorbed into recirculating water. By careful control of temperature and/or flows into the absorber tower the required concentration of formalin is achieved in the base of the tower. Formalin is then passed through a distillation tower where any remaining methanol is removed. Decanting of formalin is via pump and closed pipe system to either storage tanks on site or loaded to tankers or drums for road transport.

Most of the gas exiting the top of the absorber tower is recycled through the process again. This lowers the oxygen level of the gas stream so that it can be maintained below the explosive range for the methanol/air mix. Exhaust gases pass over a catalytic converter to minimise emissions of formaldehyde, methanol and by-products that remain. The whole manufacturing process is controlled by a computer system operated by workers in a control room.

The metal oxide process involves the oxidation of vaporised methanol using air whereas the silver catalyst process involves partial oxidation and dehydrogenation of vaporised methanol in air using steam and granulated silver. Table 7.2 shows the similarities and differences between these two manufacturing techniques.

<table>
<thead>
<tr>
<th>Table 7.2: Comparison between silver catalyst process and metal oxide process (Kroschwitz &amp; Howe-Grant, 1994; IARC, 1995)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>No. of reactions</strong></td>
</tr>
<tr>
<td><strong>Reaction type</strong></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td><strong>Reaction</strong></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td><strong>Temperature in reaction chamber</strong></td>
</tr>
<tr>
<td><strong>Pressure</strong></td>
</tr>
<tr>
<td><strong>By-products formed</strong></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
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<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>
7.2 Importation

Information on the quantities of formaldehyde imported was provided by importers of formalin and products/mixtures containing formaldehyde, for the years 2000 and 2001. Predicted quantities for the year 2002 were also provided. Furthermore, as paraformaldehyde can be a significant source of formaldehyde, imported quantities of paraformaldehyde for the same periods were provided.

The reported quantities of imported formaldehyde are listed in Table 7.3. The amount of formaldehyde (calculated as 100%) was estimated by multiplying the volume of formalin or product by the % of formaldehyde in the formalin/product. The quantity of imported formaldehyde is approximately 76 to 109 tonnes per annum.

Table 7.3: Importation quantities of formaldehyde

<table>
<thead>
<tr>
<th></th>
<th>2000 (tonnes)</th>
<th>2001 (tonnes)</th>
<th>2002* (tonnes)</th>
<th>% Formaldehyde</th>
</tr>
</thead>
<tbody>
<tr>
<td>Formalin</td>
<td>36</td>
<td>45</td>
<td>60</td>
<td>16% - 40%</td>
</tr>
<tr>
<td>Formaldehyde</td>
<td>14</td>
<td>18</td>
<td>24</td>
<td></td>
</tr>
<tr>
<td>(calculated as 100%)</td>
<td>(calculated as 100%)</td>
<td>(calculated as 100%)</td>
<td>(calculated as 100%)</td>
<td></td>
</tr>
<tr>
<td>Formaldehyde products</td>
<td>4500</td>
<td>4200</td>
<td>4400</td>
<td>0.0002% - 40%</td>
</tr>
<tr>
<td>Formaldehyde</td>
<td>95</td>
<td>58</td>
<td>61</td>
<td></td>
</tr>
<tr>
<td>(calculated as 100%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Formaldehyde</td>
<td>109</td>
<td>76</td>
<td>85</td>
<td></td>
</tr>
<tr>
<td>(calculated as 100%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Estimated figures

Formalin is imported in packaging of various sizes including 220 kg drums, 20 L drums, 22 kg carboys, 2.5 L bottles, 500 mL bottles and 10 mL ampoules. Imported formalin is transported in pallets in full container loads or on trucks mainly by road. The majority of imported formalin is used in resin manufacture and as laboratory reagents.

The information provided to NICNAS indicates that more than 250 formaldehyde-containing products, such as formaldehyde resins, film processing products, surface coating products, and preservatives, are imported. The concentrations of formaldehyde in the imported products vary widely, however, the majority of them are less than 1%. Imported products are either further incorporated into end products or used directly by end users. Some end use products containing formaldehyde are imported and sold directly to the general public. Examples include cosmetics products and other consumer products, such as fabric softener, surface liquid cleaners and dishwashing liquids.

Paraformaldehyde is imported as prills or powder in 25 kg bags. The concentrations of formaldehyde in these prills/powder range from 81% to 99%.
The total reported importation of paraformaldehyde is shown in Figure 7.3 and is approximately 700 tonnes per year. It was reported that most imported paraformaldehyde is used in resin manufacture.

**Figure 7.3: Importation of paraformaldehyde**

![Bar chart showing importation of paraformaldehyde](image)

*Estimated figure

7.3 Use

Formalin is either used by manufacturers/importers, and/or supplied to formulators to produce intermediate or end products, or sold directly to end users. A similar distribution pattern exists for imported products containing formaldehyde. The distribution chains vary as repackaging and reselling may occur as intermediate steps.

Information on uses of formalin and products containing formaldehyde in Australia was provided by industry and also obtained by site visits and a questionnaire survey (the NICNAS survey). The NICNAS survey attempted to reach users of formaldehyde through the distribution chains. The information collected by the NICNAS survey included product details, description of formulation/use processes, use of personal protective equipment, current controls and potential release to environment. A copy of the NICNAS survey form for formulators and manufacturers of formaldehyde products is provided in Appendix 2. The NICNAS survey form was modified for repackers, resellers and end users of formaldehyde. The formulators and end users were randomly selected from customer lists provided by importers and manufacturers, covering as many industry sectors as possible. However, the profile of users contacted during the NICNAS survey might not be fully representative of an industry sector, as response rate to the NICNAS survey was about 60% after a follow up attempt. Moreover, operation processes vary from site to site.

Formalin is used as a raw material for the manufacture of formaldehyde-based resins, which are widely used in a variety of industries, predominately the wood industry.
Formalin is also used directly or in blends, typically in the following industries:

- Forensic/hospital mortuaries and pathology laboratories;
- Embalming;
- Photographic film processing;
- Leather tanning;
- Sanitising treatment;
- Lubricant;
- Analytical laboratories;
- Fumigation;
- Personal care products; and
- Consumer products.

As paraformaldehyde has similar applications to formalin, the uses of paraformaldehyde are not specifically described in this section.

Formaldehyde has some other applications in Australia, including poultry shed disinfections, sheep foot rot treatments and uses of formaldehyde products as biocides and preservatives for non-industrial applications, such as pharmaceutical products. These applications are not considered in this assessment, as they are not as defined as ‘industrial uses’ by the Industrial Chemicals (Notification and Assessment) Act 1989 (Cwlth).

7.3.1 Formulation of formaldehyde products

The majority of formalin is used in the production of formaldehyde resins. Formalin and/or formaldehyde-containing products are also used as raw materials in blends to formulate non-resin industrial and/or consumer end products.

Resin manufacture

All formaldehyde manufacturers use the majority of the formalin they produce to manufacture formaldehyde resins. The total formaldehyde resins manufactured by the four companies are approximately 266 600 tonnes in calendar year 2000, 342 200 tonnes in 2001 and 257 300 tonnes in 2002 (estimation). Some importers of formalin or paraformaldehyde, and formulators who purchase formalin or paraformaldehyde locally, also manufacture formaldehyde resins. The total quantity of formaldehyde resins manufactured in Australia cannot be estimated as not all formulators were identified during the assessment. The types of resins that are manufactured in Australia include urea formaldehyde, melamine formaldehyde, phenol formaldehyde resins and combination of these resins, such as melamine urea formaldehyde resins.

The resin making process involves the reaction of formaldehyde with other reactants, such as urea, melamine and phenol or combinations of these reactants. The manufacture of resins is a batch process and conducted in enclosed systems. The manufacturing process varies from site to site. Typically, formalin is transferred through a fixed piping system and charged into resin reactors. Manual
charging of formalin from drums occurs at some smaller resin manufacturing sites. In the situation that paraformaldehyde is used, it is charged manually from sealed bags into the reactor. Each batch typically takes about 8 to 12 hours, but can vary from 5 to 30 hours depending on the technical grade of the resin. Decanting of the resins is via a closed pipe system to storage tanks on site from which it is pumped to drums, bulk containers or bulk tankers for road transport. Some workplaces decant the resins manually into 8 to 200 L drums.

The typical resin manufacture process is summarised in Figure 7.4. The majority of the formaldehyde resins contain < 0.2% free formaldehyde, but some contain > 0.2% depending on the applications of the resins. For example, some fibreglass resins contain up to 13% free formaldehyde.

Solid phenol formaldehyde resin powder is also manufactured in Australia. The molten phenol formaldehyde resin is dropped from the reactor onto a cooling floor where it becomes a brittle solid, which is then manually broken into lumps. The lumps are subsequently blended with curing agents and ground to a powder which is then packed in 15 kg or 700-800 kg bags for sale. The resin powder does not contain any free formaldehyde and is used as a binder in the manufacture of abrasive products, such as grinding wheels, brake components (for example, brake linings), and refractory products. These products are typically compression moulded and then heat cured.

**Formulation of formaldehyde products (other than resins)**

Both formalin and products containing formaldehyde are used to formulate a large number of end products that are used in various industries. In general, formulation is a batch process, in which measured amounts of formaldehyde or product containing formaldehyde and other components are added to mixing vessels and blended to form end products. The product is then transferred to containers and dispatched to customers. However, the blending processes vary from site to site. A number of examples have been selected from the industry submissions and the NICNAS survey, and are presented in Table 7.4, to illustrate the differences in formulation processes.

In general, manual processes occur in small batch productions, such as formulation of anti-graffiti wall sealer. Typically, formalin or product containing formaldehyde is decanted into a vessel for weighing before being poured into an open tub and stirred. Decanting is done with a small jar and funnel. Equipment is cleaned manually between different products with either water or cleaning solvents.

For larger-scale production, such as detergents and disinfectants formulations, formalin or product containing formaldehyde is either directly poured into a mixing tank using a drum lifter or is transferred via a transfer pump. Other ingredients are then added, followed by mechanical stirring. For some formulations, formalin or product containing formaldehyde is premixed with other ingredients before adding into the main mixing vessel. The mixing operation is usually conducted under closed or partially closed conditions and the final product is pumped into drums for transport to customers. Decanting is usually an automated process. Table 7.4 shows that the duration and frequency of the formulation process vary largely depending on a number of factors, such as customer orders, batch sizes and properties of ingredients.
Figure 7.4: Typical resin manufacture process
Table 7.4: Examples of formulation processes for formaldehyde products

<table>
<thead>
<tr>
<th>Product formulated</th>
<th>% FA in raw material</th>
<th>% FA in end product</th>
<th>Work process</th>
<th>Duration</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fixative solutions</td>
<td>37</td>
<td>4-32</td>
<td>E O N NR A NR</td>
<td>&gt;0.5 h</td>
<td>NR</td>
</tr>
<tr>
<td>Embalming fluids</td>
<td>37</td>
<td>20-30</td>
<td>M O Y NA M M</td>
<td>6-8 h</td>
<td>20</td>
</tr>
<tr>
<td>Film processing</td>
<td>37</td>
<td>10.4</td>
<td>E E N M A E</td>
<td>1 h</td>
<td>5</td>
</tr>
<tr>
<td>Preservative fluid</td>
<td>37</td>
<td>4</td>
<td>E NA N M M M</td>
<td>5 min</td>
<td>1</td>
</tr>
<tr>
<td>Leather tanning</td>
<td>37</td>
<td>&lt;1%</td>
<td>E E Y M M E</td>
<td>2.5-10 h</td>
<td>240</td>
</tr>
<tr>
<td>Anti-graffiti wall sealer</td>
<td>37</td>
<td>0.6</td>
<td>M O N M M M</td>
<td>1-2 h</td>
<td>2</td>
</tr>
<tr>
<td>Biocides</td>
<td>37</td>
<td>&lt;0.6</td>
<td>E E N M A M</td>
<td>6 h</td>
<td>200</td>
</tr>
<tr>
<td>Textile treatment</td>
<td>37</td>
<td>&lt;0.5</td>
<td>E E N M M M</td>
<td>2-3 d</td>
<td>72</td>
</tr>
<tr>
<td>Surfactants</td>
<td>37</td>
<td>&lt;0.2</td>
<td>E O N M Semi-A M</td>
<td>12 h</td>
<td>260</td>
</tr>
<tr>
<td>Consumer products</td>
<td>37</td>
<td>&lt;0.2</td>
<td>O O N M M M</td>
<td>2-4 h</td>
<td>208</td>
</tr>
<tr>
<td>Disinfectant</td>
<td>37</td>
<td>&lt;0.2</td>
<td>E PE Y NR A E</td>
<td>0.4-2 h</td>
<td>240</td>
</tr>
<tr>
<td>Detergents</td>
<td>3-21</td>
<td>&lt;0.2</td>
<td>E PE Y M A E</td>
<td>0.5-3 h</td>
<td>240</td>
</tr>
<tr>
<td>Scour pads</td>
<td>3</td>
<td>&lt;0.2</td>
<td>E PE N NA A M</td>
<td>2 h</td>
<td>100</td>
</tr>
<tr>
<td>Furniture lacquer</td>
<td>&lt;3</td>
<td>&lt;0.2</td>
<td>M PE N NR M M</td>
<td>4 h</td>
<td>6</td>
</tr>
<tr>
<td>Paints</td>
<td>0.7-3</td>
<td>&lt;0.2</td>
<td>M O N M M M</td>
<td>1-3 d</td>
<td>100</td>
</tr>
</tbody>
</table>

FA, formaldehyde; NR, not reported; NA, not applicable; E, enclosed process; PE, partially enclosed process; O, open process; A, automated process; M, manual; N, no; Y, yes.
<table>
<thead>
<tr>
<th>Product</th>
<th>Package size</th>
<th>Repackaged size</th>
<th>Work process</th>
<th>Duration</th>
<th>Frequency (day/year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Formalin (40%)</td>
<td>200 L drum</td>
<td>20 L, 5 L, 2.5 L, 500 mL bottle</td>
<td>Drums are transferred to packing area by a forklift truck. A worker connects a hose to a tap on the drum and formalin is transferred into smaller containers by gravity. The bulk storage tank is dedicated to formalin only and is not cleaned on a regular basis.</td>
<td>0.1 h</td>
<td>2</td>
</tr>
<tr>
<td>Formalin (37%)</td>
<td>Bulk tank</td>
<td>20 L, 200 L drums, 1000 L bulk box</td>
<td>Formalin is pumped from the bulk storage tank into various size containers through an enclosed tubing system. Caps are manually screwed on and the containers are taken away using forklift to storage area. The bulk storage tank is dedicated to formalin only and is not cleaned on a regular basis.</td>
<td>2 h</td>
<td>200</td>
</tr>
<tr>
<td>Formaldehyde product</td>
<td>205 L drum</td>
<td>Various sizes</td>
<td>Drums are transferred to packing area on a pallet via a forklift truck. A worker inserts a drum pump into the drum opening and product is transferred by weight into various smaller containers. Caps are manually screwed on and the containers are taken away using forklift to storage areas.</td>
<td>1 h</td>
<td>8</td>
</tr>
<tr>
<td>Formaldehyde product</td>
<td>200 L drum</td>
<td>20 L plastic pail</td>
<td>Drums are transferred to packing area on a pallet via a forklift truck. A drum pump is manually inserted into the drum opening and product is transferred by weight into 20 L plastic pails. Pails are packed onto a disposable wooden pallet, steel banded and shrink wrapped prior to transport.</td>
<td>3 h</td>
<td>2</td>
</tr>
<tr>
<td>Paraformaldehyde</td>
<td>25 kg paper bag</td>
<td>3 kg paper bag</td>
<td>Bags are opened and tipped into a 200 L bin by hands. Workers scoop out the powder and weigh them into 3 kg paper bags. Paper bags are glued shut and vacuum packed into plastic bags which are then packed in boxes and stored on pallets before transport.</td>
<td>8 h</td>
<td>40</td>
</tr>
</tbody>
</table>
7.3.2 Repackaging

Repackaging of both manufactured and imported formalin and products containing formaldehyde occurs in Australia. The package sizes before and after repackaging vary greatly and repackaging processes differ from company to company. Again, several examples of the repackaging processes have been selected from the industry submissions and the NICNAS survey and are presented in Table 7.5. Most repackaging of formalin or product containing formaldehyde is from 200 L drums to smaller containers, such as 5 L and 20 L containers. They are decanted into smaller containers either through a pump (enclosed process) or fed via gravity. Repackaging is usually not a continuous operation and the duration and frequency of the operation vary from site to site.

Formalin is also repacked from large storage tanks. The material is pumped into the storage tanks and transferred into various size containers using a pump and an enclosed tubing system.

Manual and open repackaging processes were reported during repackaging paraformaldehyde powder (see Table 7.5). It is assumed that enclosed processes may also occur in Australia.

7.3.3 End use of formaldehyde products

Formaldehyde resins

The uses of formaldehyde resins are diverse in Australia. Reported industrial uses include:

- manufacture of pressed wood products and their applications;
- paper treating and coating;
- textile treatments;
- foundry industry;
- fibreglass industry;
- composites construction;
- foam insulation;
- lighter manufacture; and
- anti-graffiti wall sealer.

Manufacture of pressed wood products

Pressed wood products are sheet materials in which wood is predominant in the form of strips, veneers, chips, strand or fibres. The categories usually recognised within this group of panel materials are:

- particleboard, including wood particleboard (chipboard), flaxboard and cement-boned particleboard;
- fibreboard, including medium density fibreboard (MDF);
oriented strand board (OSB); and

- plywood, including blockboard and laminboard.

**Particleboard and fibreboard manufacture and their applications**

The majority of the formaldehyde resins are used as adhesives in the production of particleboard and MDF in the timber industry. The types of formaldehyde resins used in this industry include urea, phenol, melamine formaldehyde resins and some combination of these resins, such as melamine urea and melamine urea phenol formaldehyde resins. The concentrations of free formaldehyde in the resins used in this industry range from < 0.2% to 0.5%. Information from the Australian Wood Panel Association (AWPA) indicates that 932 000 m$^3$ MDF and 965 000 m$^3$ particleboard were manufactured using formaldehyde resins in year 2001-2002. However, no information is available for the total consumption of each type of formaldehyde resins in this industry. AWPA represents all particleboard and MDF manufacturers in Australia. Information from Australian Customs indicates that approximately 233 000 m$^3$ wood panel products were imported in Australia in financial year 2001-2002.

Figure 7.5 is a flow diagram showing the typical process of particleboard and MDF manufacture, which is a continuous process. The formaldehyde resins are charged into storage tanks and injected and mixed with refined wood fibre through an enclosed system. The particleboard and MDF are rolled and pressed in a semi-enclosed area during the hot press stage (the temperature is 160°C to 200 °C) where resins set.

These wood panel products have both industrial and do-it-yourself (DIY) applications for decorative, structural and industrial purposes, such as shelving. Decorative applications include furniture, shelving, panelling/partitioning, mouldings and doors. Examples of structural applications are domestic and commercial flooring, access flooring, concrete formwork and exterior signs.

**Manufacture of plywood and its applications**

Formaldehyde resins containing < 0.2% to up to 5% free formaldehyde are used in the manufacture of plywood and associated structural veneer based products, such as laminated veneer lumber (LVL). The types of plywood products used in Australia include structural plywood, concrete formwork plywood, marine plywood, exterior and interior plywood, and overlaid and composite plywood.

Phenol formaldehyde resin, which is the predominate resin (approximately 88%) used in this industry, is used for bonding structural, exterior and marine plywood and structural LVL. Urea and melamine urea formaldehyde resins are usually used for interior and some formply products. According to the information from the Plywood Association of Australia (PAA), 189 533 m$^3$ of plywood and LVL were produced in the year 2001-2002 with total consumption of 3340 tonnes phenol formaldehyde and 500 to 850 tonnes of urea formaldehyde resins. PAA represents manufacturers who produce approximately 98% of plywood and LVL in Australia. PAA advised that Australian-made plywood occupies 55% of the Australian market. Information from Australian Customs indicates that approximately 74 000 m$^3$ plywood products were imported in Australia in financial year 2001-2002.
Figure 7.5: Simplified flow chart of typical particleboard and MDF manufacture

Particleboard

LOGS/CHIPS (RADIATA)

PROCESSED TO FINE FLAKES

DRIER

BLENDER

MAT FORMATION

HOT PRESS (160-200°C)

COOL PANELS

SANDING

CUT TO SIZE

MDF

LOGS/CHIPS

DIGESTER (2-3 MINUTES) -- BAR STEAM PRESS

UF/MUF/ PF/MUPF

REFINER (FIBERIZES WOOD)

Resin Wax Catalyst

BLOWLINE

DRIER

HOT PRESS (160-200°C)

COOL PANELS

SANDING

CUT TO SIZE

UF, urea formaldehyde resin; MUF, melamine urea formaldehyde resin; PF, phenol formaldehyde resin; MUPF, melamine urea phenol formaldehyde resins
Plywood/LVL manufacturing processes are similar throughout Australia. Formaldehyde resins are delivered in tankers and transferred into a holding tank from where they are pumped into enclosed mixing vessels and mixed with extenders (wheat flour), fillers (shell flour) and water. The mixed resin is then pumped into glue spreaders and applied to the veneer using rubber rollers or pressurised curtain coaters, which is an open process. The spread packs of veneer are then cold pressed and finally hot pressed at about 140 °C, where the formaldehyde resins are set.

Plywood and associated structural veneer based products are used in a number of areas:

- Residential buildings including mobile homes, such as caravans and manufactured homes. Residential building applications include LVL framing, flooring, bracing, plywood webbed beams, roofing, cladding, interior wall and ceiling linings, plywood in domestic wet areas;
- Building components for commercial and industrial structures including relocatable buildings (classrooms, offices etc.). Structural LVL and plywood components for commercial and industrial structures include flooring, stressed skin panels, beams, arches, gussets, portal frames, and bracing walls;
- Material handling, such as pallets, shelving, containers, bins and transport equipment;
- Construction on site applications, such as structural ramps, overhead protection barriers, runways etc.; and
- DIY in a wide range of projects, such as flooring, wall and ceiling lining, boat building.

**Paper treating and coating**

Urea and melamine resins containing up to 1.5% free formaldehyde are used in paper treating and coating. Paper treating is an automatic, continuous process involving two resin stages. In the first resin stage, urea resin is pumped from storage tanks to an automatic closed batching station where additives, water and a catalyst are added to help with paper saturation and promote curing in later drying and laminating processes. This mixture is pumped into the first stage bath where the paper for impregnation is automatically fed by rollers through the bath at a speed of approximately 40 meters per minute and is impregnated as it passes through the bath. The bath is open at the top. The paper then passes into a closed oven with temperatures ranging from 120°C to 170°C for drying.

In the second resin stage, melamine resin is pumped into an automatic closed batching station and mixed with a release agent and a catalyst. This mixture is then pumped into a second stage resin application station where the paper (after the first resin stage) is fed through the rollers and is coated with the resin mixtures automatically. The coated paper then goes into the second drier. Finally, the paper is automatically cut to length and stacked in plastic wrapped packs for shipment.
**Textile treatment**

The formaldehyde resin products used in the textile industry include printing inks, dyes and textile finishing products. The concentrations of free formaldehyde in these products are generally < 2%.

Textile printers use formaldehyde resins as a cross-linking agent in acrylic binder systems for pigment printing of polyester/cellulose or synthetic materials. The formaldehyde resin is diluted with water and mixed with print paste for approximately 10 minutes in a vat by either manual stirring or mechanical mixing. Typically 1% to 3% of the resin product is used in the print paste depending on the depth of shade of the print required. The print paste is then transferred onto the fabric using a print screen (flat bed printer). The print is generally cured at 150 °C for up to 3 minutes to cross link the acrylic resin binder.

At large textile dyeing enterprises, formaldehyde resin is pumped from drums into a large storage/dispensing vessel and then transferred to the dyeing equipment where the product is diluted at a rate of 1-2 g/L. The temperature inside the dyeing machine is about 100 °C. The product is rinsed off after dyeing and the water goes to trade waste. The operation is a daily activity and manual processes occur at some smaller sites.

Formaldehyde resins are used as cross-linking agents for cotton fabric and other cellulosics to produce a finish that resists hydrolysis and is inert, durable and unaffected by heat or bleach. Formaldehyde resin is poured into an open tank and diluted with water to ratios of 1:10 to 1:20. Textile finishing processes include padding, drying, and curing. The padding is normally done by immersing the fabric in the resin aqueous solution, followed by squeezing it between two rollers, and finally drying and curing. The durations of the padding vary depending on the type of fabrics.

**Foundry industry**

Formaldehyde resins are used as a sand binder to coat sand which is then used in core making for casting operations in the foundry industry.

At sand coating sites, the resin is pumped into a mixer at a rate of 1% to 1.2% resin by weight of sand. At some sites, the resin is decanted from drums manually into a measuring cup and then poured into a mixing vessel. Mixing normally takes about 5 minutes and the coated sands are then decanted into bags ready for core making at foundries. This is a batch operation and the frequency of the operation varies from site to site.

At foundry sites, a variety of iron castings are produced for the automotive industry. Foundry using sand as the moulding material consists of six basic processes: pattern making, core making, moulding, metal melting and pouring, and casting cleaning (fettling). Core making is the process of creating solid shapes from sand using a variety of binding system. These solid shapes, called ‘cores’, determine the internal cavities of the casting. Hot, warm and cold box core making techniques are used in the foundry. About 90% of the cores are produced by hot and warm box technologies, using urea formaldehyde resin, phenol, and furfuryl alcohol systems. The hot box resin system contains typically 5% to 6% free formaldehyde in the resin, whilst the warm box typically contains
2% to 3% free formaldehyde. Typically, the sand coated with formaldehyde resins is blown into a hot mould (with temperatures around 110 °C) where formaldehyde resin melts and functions as a bonding agent to make cores. At larger enterprises, sand coating and core making occurs in an enclosed system. Drums containing formaldehyde resins are connected to an automatic dosage system, which supplies a set dosage of the resin into core making machines.

**Fibreglass industry**

Formaldehyde resins containing up to 13% free formaldehyde are used as fire resistant laminates in the fibreglass industry, such as manufacture of fireproof hubcaps used in the mining industry. Formaldehyde resin is diluted with up to 40% water before it is mixed with other ingredients by manual stirring. The mixture is applied to a mould using mop rollers or bristle rollers. The mould is then put in an oven at temperatures up to 60 °C for about 12 hours, where the resin is cured.

Formaldehyde resins are also used as bonding resins to make glass fibre materials for use in the building industry. The concentration of free formaldehyde in the resins is about 1%. The resin and other ingredients are diluted with water and mixed in an open tank. The mixture is sprayed onto the glass fibres, which then pass through an oven (temperature 220°C to 300°C) where the resin is cured.

**Composite construction industry**

Formaldehyde resins containing about 3% free formaldehyde are used for the manufacture of composite parts that are used in the automotive industry, especially racing car parts. These parts are made of a few layers of either fibreglass or carbon fibre clothes coated with formaldehyde resins. The resin is mixed manually with a hardener in a ratio of 20:3. A worker applies the blend onto each fibreglass or carbon fibre cloth sheet using a brush, before piling several sheets together to make a mat. The mat is then moulded into the shape of a car part. Depending on the application of the part, it is either left at room temperature or gradually heated up to 250 °C in an oven for 1 to 2 days when the resin is cured.

**Foam insulation**

Formaldehyde resins containing up to 5% free formaldehyde are used to make foam insulation for industries, such as the floral industry. Formaldehyde resins are pumped into a mixing bowl and blended with other ingredients for about 5 minutes in an open system. The blend is then tipped into a mould and baked under 45 °C in an oven for about 90 minutes to make solid foams. The foam is then cut and processed into various shapes and sizes to sell to wholesale companies.

**Firelighter manufacture**

Formaldehyde resins containing up to 1% free formaldehyde are used in firelighter manufacture. The resin is pumped from a refrigerated storage tank into an enclosed mixing tank and mixed with other ingredients. The resin accounts for approximately 11% of the total mixture. The mixed product is then automatically deposited into trays, which are then wrapped and boxed approximately one minute after initial deposit into tray. Firelighter manufacture is a daily operation.
Anti-graffiti wall sealer

The product is a low gloss resin containing up to 1% free formaldehyde. The product is stirred manually prior to use and during application. It is applied at a rate of not less than 200 mL/m² using airless spray equipment. For porous surfaces, such as blockwork, an application rate of up to 400 mL/m² may be necessary to ensure total saturation.

Formaldehyde products other than resins

Forensic/hospital mortuaries, pathology laboratories and other medicine-related uses

Formalin is used as a fixative in many medicine-related industries. The most commonly used solutions are neutral buffered formalin solutions containing 4% formaldehyde. The solutions are either purchased from suppliers already in aliquot containers/specimen jars or made on site by diluting concentrated formalin solutions containing 20% to 32% formaldehyde. The dilution process varies depending on the quantities used. Where large quantities are used, such as some forensic or hospital mortuaries and anatomy laboratories, the concentrated formalin solution is manually poured into an enclosed mixing system, diluted with water in ratios of 1:5 to 1:8 and mixed with other ingredients. These aqueous solutions are stored in enclosed large tanks (up to 1000 L) and are automatically decanted into smaller containers before end use. The aqueous solutions are manually dispensed into specimen jars and used for fixing human tissues and organs after autopsy. At workplaces where small quantities of formalin solutions are used, such as pathology laboratories, concentrated formalin solutions are diluted manually with water using measurement equipment and funnels.

The neutral buffered formalin solutions already aliquoted into specimen jars are used in hospitals and doctors’ rooms for preserving human tissues from biopsy. The specimen jars are sealed and sent to pathology laboratories. In pathology laboratories including histopathology laboratories, human tissues are taken out of the specimen jars and accessioned (‘cut-up’) to certain sizes or shapes which are then placed on a tray that goes through a processing machine (‘processor’). Accession is undertaken manually on benches equipped with ‘down draught’ extraction systems. The processor has a number of containers holding different chemical liquids including neutral buffered formalin solution, which needs to be topped up regularly (up to once a day in large laboratories). During the topping up, the container is taken out of the processor and the solution is poured in using a funnel. After the processing, the specimens are waxed and cut to prepare slices for microscopic observations.

In anatomical pathology laboratories, the corpse is transported to the cadaver preparation laboratory and kept in cold storage until embalming. The embalming procedure is conducted by laboratory technicians and formalin solutions containing 10% to 13% formaldehyde are used. The procedure is similar with that described for embalming in funeral homes below. The embalmed bodies are then used by students and prosectors for examination and dissection involving cutting and removing tissues to reveal anatomical features for further study or examination. In addition to intact cadavers, separated limbs and organs, such as the brain, lungs, and kidneys are stored in the dissection laboratory in different sized containers filled with solutions containing 1.5% to 5% formaldehyde. These
containers are distributed around the dissection laboratory and specimens are often used in classes for wet specimen observation. A stainless steel trap with a waste shredder is used for disposal of old biopsy specimens and the accompanying formalin solutions.

The 4% buffered formalin solution is also used for transporting explanted orthopaedic prostheses, which have been removed from a patient by a surgeon. The solution is stored in a ‘Histological Retrieval Kit’ containing a number of small plastic bottles of various sizes for different sized explants. One kit usually has a total of approximately 0.75 L of the formalin solution. The kits are supplied to hospital staff who sterilise the explant and transfer it to the selected container. It is then sealed for transport to overseas for investigations.

Other medicine-related uses include sterilisation of dialysis machines in hospital dialysis units. Formalin (40%) is added to the dialysis machines for approximately 15 minutes. The solution becomes diluted as water is also flushed through the machines. The solution is fed into a small open stainless steel drain when it is pumped out of the machines.

**Embalming at funeral homes**

Formalin is used extensively as a preservative fluid during embalming in the funeral industry. It is used as an arterial, internal cavities, and hypodermic injection fluid and on surface packs. The concentrations of formaldehyde in the products range from <10% to 40%. Information from the Australian Funeral Director Association (AFDA) indicates that approximately 30% to 40% of deceased bodies are embalmed in Australia for various purposes, such as allowing long distance transportation of bodies, particularly by airplanes, allowing more time for the planning and arrangement of the funeral, and allowing the body to be viewed under optimal conditions. The degree of body embalming varies.

A typical embalming procedure involves cleansing and disinfections of body surfaces and orifices, arterial embalming, cavity embalming, and supplemental embalming. Formalin products containing <10% of formaldehyde are usually used for cleansing and disinfections of body surfaces and orifices, destroying maggots and vermin, and spray to preserve, disinfect and deodorise external body surfaces.

Arterial embalming is a process whereby a disinfecting and preserving fluid is injected into a large artery and then blood is flushed out of the circulatory system by opening a vein. One or more points may be used for arterial injection depending on the circumstances. One point injection is usually sufficient in the case of natural death where no post-mortem is performed. Cavity embalming is a process by which the contents of hollow organs in the abdomen and thorax are aspirated by means of a trocar (a metal tube with a sharp point) inserted through the abdominal wall and this is followed by the injection of cavity fluid. For arterial/internal cavities injections, products containing greater than 10% formaldehyde are diluted with warm water, in dilution ranges of 1:10 to 1:33.

For areas that have not received arterial fluid or received insufficient amounts of preservative solution during arterial injection, supplemental embalming is conducted. This process includes hypodermic and surface embalming. Hypodermic embalming is the sanitation and preservation of a local area by subcuticular injection of a suitable solution. The solution may be injected by a
hypodermic needle, syringe, or an infant trocar attached by tubing to a pressurised embalming machine. Surface embalming applies surface packs to external skin, such as bedsores, ulcers, burned areas, gangrenous areas and decomposed tissue, or to internal surfaces, such as within the thoracic or abdominal cavity of an autopsied body. This form of formaldehyde products, such as gel and semi-viscous, contains approximately 15% to 18% formaldehyde.

In the case of embalming a post-mortem body, the procedure is more complicated due to disruption of normal anatomy and sometimes the resultant inaccessibility of vessels. Excised visceras are often contained in a plastic bag placed in the body cavity at the time of autopsy. This bag is removed and the visceras are washed in water and placed in a covered bucket, either with formalin (37%) or treated with paraformaldehyde powder (containing up to 99% formaldehyde) for at least 30 minutes. For arterial injections, a six-point injection, comprising 2 carotid arteries (neck), 2 femoral arteries (thigh) and 2 auxiliary arteries (shoulder), is usually undertaken. The cranial, thoracic and abdominal cavities are aspirated and dried and the internal walls may be coated with gel products. Next the bag containing the treated visceras is sealed and replaced in the body cavity. Alternatively, the organs are replaced loose and packed with granular paraformaldehyde. Paraformaldehyde is also used to absorb moisture in incisions, lacerations and wounds.

Considerable leakage can occur through severed blood vessels in the head and a pool of arterial fluid can build up in the open abdominal cavity. Blood and excess formalin solutions go to a draining system connected to the embalming table. Infectious waste is placed in labelled plastic bags and disposed by incineration in a facility approved by the State Environment Protection Authority (EPA). The transport of the waste is required to comply with the relevant EPA regulations.

After embalming, the embalming room and equipment are cleaned. The embalming table/trolley is washed and disinfected after each use. All tubing used are washed by flowing water and then flushed with disinfectant. Floors are cleaned using detergent and hot water. Equipment cleaning and sterilisation are undertaken by autoclaving (a process which uses steam under increased pressure to destroy all organisms), chemical disinfectants, or boiling.

The handling of formalin products in the funeral industry is usually carried out by embalmers.

**Photographic film processing**

Products containing formaldehyde are used in the photographic industry as a preservative/stabiliser/replenisher in final baths to prevent deterioration of image quality on colour negative and colour reversal films. They are also used as a hardener in final baths to prevent damage to the gelatine emulsion coating of black and white films during machine processing.

Formaldehyde products containing high concentrations of formaldehyde (20% to 35%) are used in final baths of some specialised film processing, such as aerial film processing. The products are in 9L or 19 L plastic drums and carried from the storage area to the film processing area. Workers open the cap and insert a tube into the drum. The product is pumped into the bottom of an enclosed wash tank (final bath) in an enclosed machine. Water is injected at the same time to dilute the solution. The formaldehyde concentration in the working solution is
< 1%. The aerial film goes through the final bath before passing a dryer (at 140 °C) and being developed. The wash goes to drain after use. The empty drums are sent to landfill or rinsed with water for re-use.

Most commercial film processing sites use enclosed machines (processors) that have a final bath tank specifically for formaldehyde aqueous solutions. The concentrations of formaldehyde in the solutions range from 0.1% to 15%. The solution is poured into the tank and diluted with water in the required ratios ranging from 1:100 to 1:1000. Typically, the processors are operated for an average of 4 or 5 hours a day, 5 days a week. The final bath is replenished about 1 to 2 times a week. The waste generated during film processing either goes to drain or is collected in a container for disposal.

Manual film processing also occurs at some workplaces (for example, quality control trials at aerial film companies) or at homes where people do their own film processing. Solution containing 10% formaldehyde is diluted at a ratio of 1:40 and poured into a deep tray where negative or film paper is merged to develop photos in a dark room.

**Leather and fur tanning**

Formalin containing 37% formaldehyde is used as a cross-link agent in fur tanning processes. Workers dilute the formalin solution at a ratio of 1:10. The working solution is then added manually to an enclosed processing drum. This operation takes about 5 minutes. Furs are added into the drum and mechanically rotated for 18 to 24 hours. The solution is drained before furs are removed manually to an open tub. The tanned furs then go through drying, staking and other numerous processes. The NICNAS survey data indicates that formalin is used occasionally in fur tanning, for example, one leather processing company uses it 6 times a year.

Products containing 10% to 15% formaldehyde are used daily in general leather tanning. The processes are similar with the fur tanning, except the addition of the product from intermediate bulk container to the processing drum is via an enclosed system.

Information from the Department of Textile and Fibre Technology (Leather Research Centre) of Commonwealth Scientific and Industrial Research Organisation (CSIRO) (CSIRO, 2004) indicates that a limited number of leather tanning companies use formalin.

**Sanitising treatment**

Formalin containing 37% to 40% formaldehyde is used as an additive to sanitise water treatment plants. The formalin is manually measured and poured into a water holding tank to make a 1% formaldehyde solution. The diluted solution is then pumped through the water pipe system for cleaning. This operation is undertaken occasionally, for example, one company conducts the treatment about twice a year.

Products containing up to 10% formaldehyde are also used to sanitise bins and digest portable toilet contents. For the bin disinfectants, product is usually diluted at ratios of 1:6 to 1:10 and added manually to sanitary bins. Toilet sanitizers are poured into portable toilets at a rate of 20 to 50 mL product per 5 L of holding
tank capacity per week. For recirculating toilets, 200 mL product is needed for initial charge. The waste goes to sewage systems.

**Lubricant products**

Some industrial lubricants contain > 0.2% formaldehyde as a preservative. For example, conveyor lubricant (0.3% formaldehyde) is used to provide lubrication and equipment protection for conveyor belts made of steel and plastic. Before use, the product is manually poured into a big container diluted with water to 0.1%. The diluted product is continuously dispersed onto the conveyor belt through an enclosed automatic system.

**Laboratory reagents**

Analytical grade formalin and paraformaldehyde powder/prill are commonly used in research laboratories as reagents. The concentrations of formaldehyde in formalin products range from 0.2% to 40%. The paraformaldehyde powder/prill contains 95% to 97% formaldehyde. Most of the analytical grade products are supplied to laboratories as imported/formulated. Some importers repackaged the products before selling to either distributors or end users including commercial enterprises, such as contract and company in-house analytical laboratories, universities and government laboratories. Quantities imported are relatively small. The average importation quantity for the calendar years 2000 to 2002 was 1100 L formalin products and 150 kg paraformaldehyde prills per year. Information on the quantities of analytical grade formalin formulated in Australia is not available.

**Fumigation**

Paraformaldehyde, in granular form, is used for fumigation of sterile areas, such as pharmaceutical plants. Workers transfer the paraformaldehyde granules into gas generators, which contain silicone oil. The paraformaldehyde granules are placed on the top of silicone oil. The oil is heated and the formaldehyde gas generated is released into the air at a dispensing rate of 10 g/m³. The activation of the fumigation generators is remote controlled and the gas generation continues for 3 hours. No access is allowed to the area for 30 hours after the fumigation and the air conditioning is initiated 8 hours after the fumigation and remains on for at least 28 hours. Air monitoring is conducted and must be less than 0.2 ppm before access is allowed. The residue in the generators is tipped into a waste drum and sent to an approved waste destruction company. The operation is run 1 to 2 times a year.

**Products containing < 0.2% free formaldehyde**

Industry uses numerous end products containing < 0.2% free formaldehyde (see Table 7.6).

**Cosmetics and consumer products containing formaldehyde**

Formaldehyde functions as a drying agent, surfactant or preservative in cosmetics and consumer products, such as homecare products and household cleaning products. Table 7.7 lists reported products containing formaldehyde.
<table>
<thead>
<tr>
<th>Product</th>
<th>Use</th>
</tr>
</thead>
</table>
| Adhesive products               | □ Formaldehyde functions as a biocide in water based adhesives and sealants which are used in insulation and construction industry, hardware and DIY soft floor adhesives  
|                                 | □ Use of starch adhesives to manufacture corrugated boards that are used in packaging industry to increase water resistance properties  
|                                 | □ Laminating paper  
|                                 | □ Bonding of paper when manufacturing industrial paper bags  
|                                 | □ Trim adhesives for automobile industry  
| Surface coating products        | □ Coating cookware, bake ware, scissors, photocopy rollers and other surfaces where non-stick, low friction qualities are required  
|                                 | □ Thermosetting coating in coil and automotive steel coating industry  
|                                 | □ Coating cans  
|                                 | □ As preservatives (biocide) in paints/printing inks  
| Concrete admixtures             | □ Enhance properties, such as flow, setting times and strengths of the plastic and/or hardened concrete  
| Cementitious compounds          | □ Cement containing compounds are used as concrete repair and levelling or as grouts  
| Cross linker products           | □ Rubber, emulsion polymers, paper filter, paint, adhesive, textiles  
| Metal treatment products        | □ Metal plating, such as Nickel plating  
|                                 | □ As a biocide in metal working fluids  
| Fire barrier & caulk            | □ Caulk for fire-rated walls  
| Carpet protector                | □ Mill applied carpet protection  
| Rubbing compound                | □ Removal of colour sanding scratches leaving minimal swirl marks while polishing  
| Floor finish products           | □ Used to seal and polish floors in large areas, such as supermarket and nursing homes  
| Industrial cleaning products/ disinfectants/ sterilsers | □ Industrial laundry and housekeeping products, floor cleaner, carpet cleaner, truck wash liquid, dishwasher detergents |
Table 7.7: Reported cosmetics and consumer products containing formaldehyde

<table>
<thead>
<tr>
<th>Cosmetics and personal care products</th>
<th>Shampoos and conditioners</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Shower gels</td>
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<tr>
<td></td>
<td>Liquid hand soaps</td>
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<tr>
<td></td>
<td>Cream cleansers</td>
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<tr>
<td></td>
<td>Skin moisturiser</td>
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<tr>
<td></td>
<td>Toothpastes</td>
</tr>
<tr>
<td></td>
<td>Nail hardeners</td>
</tr>
<tr>
<td>Household cleaning products</td>
<td>Sink detergent</td>
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<tr>
<td></td>
<td>Toilet cleaner</td>
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<tr>
<td></td>
<td>Stainless steel cleaner</td>
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<tr>
<td></td>
<td>Glass cleaner</td>
</tr>
<tr>
<td></td>
<td>Leather cleaner</td>
</tr>
<tr>
<td></td>
<td>Laundry liquid cleaners/sprays</td>
</tr>
<tr>
<td></td>
<td>Surface liquid cleaners</td>
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<tr>
<td></td>
<td>Floor cleaner</td>
</tr>
<tr>
<td></td>
<td>Rinse aid</td>
</tr>
<tr>
<td></td>
<td>Carpet cleaners</td>
</tr>
<tr>
<td></td>
<td>Dishwashing liquids</td>
</tr>
<tr>
<td>Homecare products</td>
<td>Fabric conditioners/softeners</td>
</tr>
<tr>
<td></td>
<td>Fabric wash</td>
</tr>
<tr>
<td></td>
<td>Wool wash</td>
</tr>
</tbody>
</table>

Concentrations of formaldehyde in cosmetics and consumer products are generally less than 0.2%. Reported products containing > 0.2% formaldehyde include concentrated fabric softener (0.3%), concentrated detergent (0.3%), concentrated dishwashing liquids (0.6%), and nail hardeners (up to 1%).

**Formaldehyde donor products**

Products designed to slowly release formaldehyde during use are used in Australia. 1,3-dihydroxymethyl-5, 5-dimethyl hydantoin (usually called DMDM Hydantoin) is the most commonly used chemical to release formaldehyde in this type of product. According to the industry submissions, approximately 16 000 kg DMDM Hydantoin was imported in the year 2000. Small amounts of other formaldehyde releasing chemicals/products, such as imidazolidinyl urea and tris-(hydroxy methyl) nitromethane, are also imported.

Formaldehyde releasing chemicals and/or products containing formaldehyde-releasing chemicals are used as preservatives for the control of bacteria and fungi in water-based solutions and for the long-term preservation of starch solutions including both industrial products and a wide range of consumer products, mainly cosmetics and toiletry products. Use of formaldehyde-releasing-chemicals as hardeners in the manufacture of phenolic based refractory binders and as a biocide in industrial emulsions, such as for aluminium rolling, are also reported.

The free formaldehyde content in DMDM Hydantoin is usually up to 2%. DMDM Hydantoin is typically used at a concentration of 0.2% in personal care products. Therefore, the concentrations of free formaldehyde in the end products are much less than 0.2%. However, information from suppliers indicates that the content of DMDM Hydantoin in final products can be up to 40% in some industrial products.
Once in contact with water in a mixer, DMDM hydantoin releases a molecule of formaldehyde. Rate of release can be controlled by pH adjustment or temperature. This results in an equilibrium state in the product where 0.2% Hydantoin molecules co-exist with free formaldehyde molecules at a very low concentration. If the product encounters any bacterial activity, these free molecules of formaldehyde are consumed against the bacterial cells. This will again result in the replenishment of formaldehyde molecules in the product from the donor molecule till equilibrium is reached. Over a period of time all formaldehyde from the donor molecule is used up in preserving the product against microbes.

7.4 Export

Formaldehyde manufactured in Australia is generally not exported. One of the formaldehyde and resin manufacturers reported an export of approximately 75 tonnes formaldehyde resins per year to New Zealand.
8. Environmental Release, Fate and Effects

Formaldehyde occurs naturally in the atmosphere and biosphere, where it is released through a variety of biological and chemical processes. The most important process responsible for natural background concentrations of formaldehyde in the environment is the photochemical oxidation of atmospheric methane. Other processes responsible for release of formaldehyde to nature are reactions of hydroxide radicals (OH) with terpenes and isoprene emitted from the foliage of plants, direct emission of formaldehyde during decomposition of organic matter (Martin et al. 1999), photochemical production of formaldehyde in snowpack, and direct emissions from algae living in the snow (Sumner and Shepson, 1999). Formaldehyde occurs naturally in plants and animals (IARC 1995).

A wide range of human domestic and industrial activities is responsible for both direct and indirect releases of formaldehyde into the atmosphere from diffuse and point sources. Emission from fuel combustion is perhaps the single most important anthropogenic source of atmospheric formaldehyde, with formaldehyde being released directly or subsequently formed by oxidation of higher alkanes, hydrocarbons, or other precursors, released from combustion processes (Lowe et al. 1980). Release of formaldehyde into the atmosphere or aquatic environment may also occur during its manufacture, or when used as an intermediate in manufacturing, and during use of products containing formaldehyde.

8.1 Release

8.1.1 Emissions to the atmosphere

Recent data from the Australian National Pollution Inventory (NPI) database for emissions of formaldehyde indicate that almost all formaldehyde is released to the atmosphere, with total emissions estimated to be 7150 tonnes for the year 2002-2003 compared with 6600 tonnes for the year 2001-2002.

Figure 8.1 is a summary of the atmospheric emissions estimates by source category. For the estimation methods, refer to the relevant NPI Emissions Estimation Technique Manuals, which are available on the NPI website (NPI, 2005a). The estimates include aggregated emissions estimates reported by state government departments and data reported by industry from individual industrial facilities (labelled ‘industry emissions’). Aggregated emissions are derived from domestic, mobile and non-industrial facilities, and from smaller industrial facilities not meeting the thresholds criteria for industry reporting, while industry emissions are derived from a large number of industrial activities emitting above-threshold levels of formaldehyde. The threshold criteria is use of ≥10 tonnes of formaldehyde per year, where “use” is defined as the handling, manufacture, import, processing, coincidental production, or other uses.
Figure 8-1: Annual formaldehyde atmospheric emissions for (a) 2001-2002 and (b) 2002-2003 (NPI).

The NPI data indicate that most of the atmospheric emissions of formaldehyde occur through combustion processes from diffuse sources. The primary combustion activities are burning of domestic fuel and transportation. The domestic fuel category includes burning solid and liquid fuels and gas for domestic heating and cooking, and lawn mowing. The transportation category includes emissions from motor vehicles, rail transport, recreational boating, commercial shipping, and air transport.
Formaldehyde emissions from industrial facilities are predominantly point source emissions including both direct emissions of vapour and emissions from fuel combustion. According to the NPI estimates, point source emissions from industrial activities contributed about 16% of the total formaldehyde emissions for 2001-2002 (1085 of 6600 tonnes) and around 14% for 2002-2003 (1022 of 7150 tonnes).

Miscellaneous combustion and miscellaneous activities also contribute diffuse and point source emissions of formaldehyde. The miscellaneous combustion category includes burning of vegetation for fuel reduction, regeneration, agricultural management, and wildfires, in addition to fuel combustion from sub-reporting threshold industrial and commercial facilities, and cigarette smoking. Miscellaneous activities include direct vapour emissions and fuel combustion from use of domestic and commercial aerosols, operation of agricultural machinery, and contributions from the operation of schools, laundries, bakeries, pubs and other small business enterprises.

### 8.1.2 Emissions to water and soil

Emissions of formaldehyde to water and soil may be expected to occur via sewage treatment facilities during manufacture of formaldehyde and formaldehyde products and during use of products containing formaldehyde, including consumer products.

However, formaldehyde emissions to water and soil are significantly less than emissions to the air. Emissions data from the NPI indicate only about 1000 kg of formaldehyde was released into water and/or onto land from point sources in the reporting year 2001-2002 and only 5 kg in 2002-2003. No distinction was made between amounts released to soil and that to water.

Formaldehyde is present in low concentrations (the majority < 0.2%) in a wide variety of consumer products. These products include household cleaning products, such as dishwashing liquids, disinfectants, fabric conditioners, and cosmetics products, such as shampoos, conditioners, and shower gels etc. (Section 7.3.3). Many of these products are released directly into wastewater streams during their use, and hence are a diffuse source of formaldehyde, which may contribute to formaldehyde levels in water.

Formaldehyde emissions to soils are most likely to occur through disposal of solid wastes containing formaldehyde. A number of companies indicated that they disposed of small amounts of solid waste containing formaldehyde (mainly solidified resin waste and sludge from on-site treatment facilities) into landfill.

### 8.2 Fate

This section summarizes the environmental fate of formaldehyde, emphasizing the atmospheric fate, as more than 99% of formaldehyde is released to air, with only small amounts being released to water and soil. The information is derived from the published literature and a number of peer-reviewed reports on formaldehyde. The latter include US EPA (1993), IPCS (1989), IPCS (2002), and the Canadian Priority Substance List report (Environment Canada, 2001). Data cited from existing reports are referenced as such and not necessarily by the original authors of the particular studies.
8.2.1 Atmosphere

In the atmosphere, formaldehyde has a high degree of chemical reactivity and is capable of undergoing a wide variety of chemical reactions (Section 5). However, the major mechanism of destruction of formaldehyde is by photolysis. Less important removal mechanisms are reactions with photochemically produced OH radicals and other trace substances, including nitrate (NO₃) and hydroperoxyl (HO₂) radicals, hydrogen peroxide (H₂O₂), ozone (O₃), and chlorine (Cl₂), and all classes of hydrocarbon pollutants (Atkinson, 1990).

The oxidation of formaldehyde with OH radicals proceeds primarily by H-atom abstraction, forming formyl (HCO) radicals, which then rapidly react with O₂ to form carbon dioxide (CO₂) and hydroperoxyl (HO₂) radicals. Other products formed during these reactions include water, formic acid, carbon monoxide (CO), and hydroperoxyl/formaldehyde (HCO₃) adduct (US EPA, 1993).

During direct photolysis, formaldehyde absorbs UV radiation from below 290 nm to about 340 nm. The dominant photolytic pathway produces stable molecular hydrogen (H₂) and carbon monoxide (Atkinson et al., 1990; Lowe et al., 1980). A second photolytic pathway produces an HCO radical and a hydrogen atom, both of which react quickly with oxygen to form hydroperoxyl radicals and carbon monoxide (US EPA, 1993).

Formaldehyde is an important precursor in smog formation in the urban atmosphere, where it reacts with nitrogen oxides and other compounds to eventually form ozone, peroxyacetyl nitrate and other compounds.

The daytime half-life of formaldehyde in ambient air is generally short. The calculated half-life of formaldehyde with respect to photolysis is about 4 hours, and to reactions with OH radicals is 1.2 days. Reactions with NO₃ radicals and O₃ are slower, with the half-life times for NO₃ reactions of 80 days, and for ozone reactions of > 4.5 years (Atkinson, 2000; US EPA, 1993).

The atmospheric residence time of formaldehyde varies with the availability of hydroxyl and nitrate radicals to react with formaldehyde, which is principally controlled by the season, time of day, intensity of sunlight, temperature and cloud cover. Table 8.1 provides the calculated atmospheric residence times (in hours) of formaldehyde, taking into account gas-phase reactions with OH, NO₃, and H₂O, photolysis, in-cloud reactions with OH, and wet and dry deposition (US EPA, 1993).

During the day, reaction with hydroxyl radicals is an important removal process of formaldehyde when their concentration is high. At night, reaction with nitrate radicals is an important (although slower) removal process, particularly in polluted urban areas where the concentration of nitrate radicals is high (Atkinson, 2000; IPCS, 2002). In the absence of nitrogen dioxide, the half-life of formaldehyde is approximately 50 min during the daytime. In the presence of nitrogen dioxide, this drops to about 35 min (IPCS, 1989). In winter on clear days, residence times of formaldehyde will be longer than in summer because the intensity of sunlight is lower.

Because of its high water solubility, formaldehyde is efficiently transferred into clouds and rain, where it can react with aqueous hydroxyl radicals in the presence of oxygen to produce formic acid and hydroperoxide. The formic acid may then
be removed in rainfall. Small amounts of formaldehyde may also be removed by dry deposition. The atmospheric residence time of formaldehyde under rainy conditions ranges from minutes in cold climates to a few hours in warm climates (Atkinson, 2000; US EPA, 1993). Table 8.1 shows that wet deposition results in significantly more rapid removal rates of formaldehyde during winter on rainy days.

Table 8.1: Seasonal and diurnal variations in the atmospheric residence times of formaldehyde (US EPA, 1993)

<table>
<thead>
<tr>
<th>Weather conditions</th>
<th>Time of Day</th>
<th>Atmospheric residence times (hours)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>New York</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Summer</td>
</tr>
<tr>
<td>Clear sky</td>
<td>Day</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Night</td>
<td>20-110</td>
</tr>
<tr>
<td></td>
<td>Average</td>
<td>5</td>
</tr>
<tr>
<td>Cloudy sky</td>
<td>Day</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Night</td>
<td>18-50</td>
</tr>
<tr>
<td></td>
<td>Average</td>
<td>9</td>
</tr>
<tr>
<td>Rainy</td>
<td>Day</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Night</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Average</td>
<td>3</td>
</tr>
</tbody>
</table>

### 8.2.2 Water

Formaldehyde is highly water soluble, with a solubility of up to 550 g/L at 25°C. Concentrations as high as 95% formaldehyde in water are obtainable if suitable temperatures are maintained and methanol and other substances are added as stabilizers (IPCS, 1989). The concentrations of formaldehyde in formalin solutions manufactured in Australia range from 37% to 54%. In dilute aqueous solutions, formaldehyde exists almost exclusively in the hydrated gem-diol form \([\text{CH}_2\text{O} + \text{H}_2\text{O} \leftrightarrow \text{CH}_2(\text{OH})_2]\), while at higher concentrations formaldehyde forms other species, such as methylene glycol, polyoxymethylene and hemiformals (Environment Canada, 1985; Dong & Dasgupta, 1986).

Most aqueous formaldehyde released into water is expected to remain dissolved in the aquatic compartment where it would enter sewage treatment facilities. While the vapour pressure of formaldehyde indicates a high volatility (516 kPa at 25°C), the Henry’s Law Constant (0.022-0.034 Pa.m³/mol) indicates only a moderate volatility from water (Mensink et al., 1995).

Limited degradation data are available. It is expected that formaldehyde will be degraded relatively rapidly in sewage treatment plants and in surface water. Formaldehyde does not contain any hydrolysable groups, and hence hydrolysis will not be a degradation pathway. However, at low concentrations, formaldehyde is readily biodegradable, with 90% degradation reported in a closed bottle test (at 2-5 mg/L) after 28 days (Gerike & Gode, 1990). Howard et al. (1991) estimate 57% to 99% removal from sewage treatment plants with secondary treatment. The aqueous anaerobic half-life times are predicted to be from 1 to 7 days in unacclimated sludge. The estimated half-life times in surface water are 24-168 hours, and in groundwater are 48 to 336 hours (Howard et al., 1991).
8.2.3 Soil and sediment

Limited data are available about the fate of formaldehyde in soil and sediment. Formaldehyde is formed in the early stages of decomposition of plant residues in soils and is degraded by soil bacteria such that accumulation in soil does not occur (IPCS, 1989). The high water solubility and low partition coefficient (maximum Log Kow of 0.35) indicates a low potential for adsorption onto suspended sediments in the soil solution or in aqueous environments. Aqueous solutions of formaldehyde released into soil through spills or disposal would be expected to infiltrate into the soil, from where it may leach into surface and ground water. However, since formaldehyde is susceptible to biodegradation by a range of micro-organisms, it is expected to be readily degraded, and not accumulate. Howard et al. (1991) estimates a soil half-life of 24 to 168 hours, based on the estimated aqueous aerobic biodegradation half-lives.

8.2.4 Biota

Formaldehyde occurs naturally in plants and animals, and is readily metabolised by organisms. The measured Log Kow indicates a low potential for bioaccumulation. This is confirmed by negative results of bioaccumulation studies with shrimp and fish showing no bioaccumulation of formaldehyde (OECD, 2002). A bioconcentration factor of 0.19 has been calculated based on a log octanol/water partition coefficient of 0.65 (IPCS, 2002).

8.3 Effects on organisms in the environment

The ecotoxicity data presented here are summarized from existing reports on formaldehyde, on-line computer databases, and the published literature. Due to the large volume of data, for example, 655 records in the US EPA ECOTOX (US EPA, 2002) database, predominantly for aquatic organisms, not all studies have been evaluated.

A recent paper by Hohreiter and Rigg (2001) highlights the poor reliability and quality of much of existing data on the aquatic toxicity of formaldehyde (the same can be said for the terrestrial toxicity data). The main criticisms were a lack of analytical confirmation of the concentrations of formaldehyde (most endpoints being reported as nominal concentrations), and the lack of GLP compliance (many of the studies were conducted prior to the introduction of GLP). A further criticism was the lack of available chronic toxicity data. Where possible, any anomalous or unreliable data are indicated.

8.3.1 Aquatic organisms

Fish

The US EPA ECOTOX database (US EPA, 2002) lists acute toxicity endpoints of formaldehyde for a large number of fish species. Many of these endpoints appear to be derived from non-standard tests. The 96-hour test data show that formaldehyde is practically non-toxic to fish, with most species listed having lethal concentration (LC50) values above 100 mg/L. The lowest recorded 96-hour LC50 in the database is 1.51 mg/L for Bluegill sunfish (Lepomis macrochirus). However, the original source of the latter endpoint is uncertain. The reference
indicates the data is from the Environmental Effects Database, Office of Pesticide Programs of US EPA.

The Hohreiter and Rigg review (2001) suggests that acute toxicity endpoints do not vary greatly between fish species. In their review of the most reliable existing data, striped bass (Morone saxatilis) is indicated to be the most sensitive fish species, with adjusted mean (to formaldehyde concentration) 96-h LC50 values of 16.9 mg/L (range 7.26 mg/L to 24.44 mg/L, of 13 endpoints). The most resistant fish species to formaldehyde are rainbow trout, with LC50 values of 58.7 mg/L, and Atlantic salmon with LC50 values of 69.8 mg/L.

Fajer-Ávila et al. (2004) have recently reported a study of the effects of formalin on bullseye puffer fish (Sphoeroides annulatus Jenyns, 1843). The replicated static study determined a 72-hour LC50 of 79 mg/L based on measured concentrations. The study also reported sublethal effects, including immobility and slow reaction to external stimulation, in the concentration range 24 mg/L to 103 mg/L. At concentrations above 75 mg/L, the test fish showed glassy exophthalmic eyes with an opaque film after 13 hours and haemorrhages in fins and eyes by 20 hours. Effects on the epithelial structure and mucous cell densities in rainbow trout (Oncorhynchus mykiss) have also been reported at concentrations between 50 ppm and 300 ppm (Buchmann et al., 2004).

Recent replicated static renewal studies with 7-day-old fathead minnow (Pimephales promelas) and conducted according to GLP indicated 96-hour LC50 and median effective concentration (EC50) (lethality and behavioural effects) values of 27.2 mg/L (Hohreiter & Rigg, 2001).

**Amphibians**

The responses of various species of amphibians are similar to those of fish, with median acute LC50 ranging from 10 mg/L to 20 mg/L for a 72-hour exposure. For example, leopard frog tadpoles (Rana pipiens) had a 72-hour LC50 value of 8.7 mg/L, and toad larvae had a 72-hour LC50 value of 18.6 mg/L. The available data indicate that tadpoles are more sensitive to formaldehyde than most species of fish and aquatic invertebrates. No data are available on long-term aquatic studies (IPCS, 1991; Hohreiter and Rigg, 2001).

**Aquatic invertebrates**

Unlike fish, aquatic invertebrates show a wide range of responses to formaldehyde. Available acute toxicity endpoints [adjusted by Hohreiter and Rigg (2001) to reflect formaldehyde content] indicate a range of 96-hour LC50 values between 0.42 mg/L for the seed shrimp (Cypridopsis sp.) and 337 mg/L for backswimmers (Notonecta sp.). Data in the US EPA ECOTOX database (US EPA, 2002) show EC50 values for mussels (Mytilus edulis) ranging between 5 mg/L and 60 mg/L. Hohreiter and Rigg (2001) list adjusted endpoints for molluscs (Corbicula and Helisoma sp) of between 35 mg/L and 50 mg/L.

The above data indicate that the seed shrimp is the most sensitive organism. However, Hohreiter and Rigg (2001) believe this endpoint (attributed to Bills et al. 1977) is anomalous. More recent replicated tests, performed under standard conditions with analytical confirmation of nominal formaldehyde concentrations, indicate much higher 96-hour EC50 values of 54.4 mg/L to 68.6 mg/L for Cypridopsis. The NOEC is 18.8 mg/L (measured) for both survival and
reproduction, and the LOEC is 50 mg/L. The most sensitive species attained from the most reliable endpoint for invertebrates reviewed in Hohreiter and Rigg (2001) is 5.8 mg/L for Daphnia pulex (96-hour EC50).

Available data indicate formaldehyde is slightly to moderately toxic to Daphnia. In the US EPA ECOTOX database (US EPA, 2002), the 48-hour EC50 values reported for the water flea (Daphnia magna) ranged between 14 mg/L and 58 mg/L. Recent replicated tests reported by Hohreiter and Rigg (2001) showed comparable values, with 48-hour static acute LC50 values of 9.45 mg/L for Ceriodaphnia dubia and 14.75 mg/L for Daphnia pulex.

Chronic toxicity of formaldehyde to Ceriodaphnia dubia in two 7-day tests for immobility and mortality gave NOEC and LOEC values of 3.0 mg/L and 6.0 mg/L, and 1.0 mg/L and 3.0 mg/L, respectively. The geometric mean of each test provide two chronic values of 4.24 mg/L and 1.73 mg/L, respectively (Hohreiter and Rigg, 2001).

Algae and aquatic plants

Only a limited number of studies have been carried out to evaluate the toxicity of formaldehyde to aquatic plants. In general, these data suggest that formaldehyde is slightly to moderately toxic to aquatic plants. However, much of the data is difficult to evaluate owing to the non-standard test methods used. The SIAR (OECD, 2002) indicates the toxic threshold (192 hours) of formaldehyde to Scenedesmus quadricauda in a static cell multiplication inhibition test using an aqueous solution of formaldehyde (35% solution) is 0.88 mg/L. The toxic threshold is defined in the cited investigation as the concentration of the test substance causing 3% inhibition of cell multiplication compared to untreated controls. The IPCS Report (1991) lists a 24-hour LC50 value of 0.4 mg/L for Scenedesmus sp. The US EPA ECOTOX database (US EPA, 2002) lists the following LOEC and NOEC values for algae: Blue-green algae (Microcystis aeruginosa) = 0.39 mg/L, Brown algae (Phyllospora comosa) = 0.1 mg/L to 10 mg/L; and Green algae (Scenedesmus quadricauda) = 0.3 mg/L to 2.5 mg/L. Most of these data are for 4 to 8 day tests, and are therefore not standard endpoints.

Hohreiter and Rigg (2001) did not estimate a final value for aquatic plants because most of the data they reviewed did not meet US EPA requirements. However, they believe that criteria protecting aquatic animals should also adequately protect aquatic plants.

8.3.2 Terrestrial organisms

Relatively few data are available on the toxicity of formaldehyde to terrestrial organisms. The US EPA ECOTOX database (US EPA, 2002) lists only 11 records for terrestrial organisms including plants, and with only two studies on birds. For the majority of these records, no endpoints are reported.

The studies on birds indicate that formaldehyde is practically non-toxic to Mallard duck (Anas platyrhynchos) and Northern bobwhite quail (Colinus virginianus), with the Mallard having an 8-day LC50 > 5000 ppm, and the Northern bobwhite having an 8 day LC50 > 5000 ppm and a 14 day LD50 of 790 mg/kg.
Several studies cited in the CICAD (IPCS, 2002) indicate potentially adverse effects on terrestrial plants after exposure to formaldehyde in air and fog. Bean plants (*Phaseolus vulgaris*) exposed to formaldehyde in air at concentrations between 65 ppb to 365 ppb for up to 4 weeks exhibited no short-term effects, but showed an imbalance in shoot and root growth, which could increase the vulnerability of plants to environmental stresses, such as drought.

Plants exposed to formaldehyde in fog water for 40 days (4.5 hour/night, 3 nights/week) at concentrations equivalent to 18 µg/m³ and 54 µg/m³ (14.9 ppb and 44.8 ppb) showed a range of potentially adverse effects. Rapeseed (*Brassica rapa*) exhibited a reduction in leaf area, leaf and stem dry weight, and flower and seedpod numbers, while slash pine (*Pinus elliotti*) exhibited an increase in needle and stem growth. Wheat (*Triticum aestivum*) and aspen (*Populus tremuloides*) exposed to formaldehyde in fog during the study exhibited no effects.

Pollen germination has been shown to be sensitive to some air pollutants. Pollen grains of *Lilium longiflorum*, sown in a straight line on a culture medium, were exposed separately for one, two, and five hours to formaldehyde gas at concentrations of 0.44 mg/m³ (0.35 ppm) and 2.88 mg/m³ (2.3 ppm). Grains exposed to the lower concentration for five hours showed a significant reduction in pollen-tube length, whereas a one- or two-hour exposure time had no effect. Pollen grains exposed to formaldehyde concentrations of 2.88 mg/m³ showed a decrease in tube length after one hour of exposure (IPCS, 1989).

### 8.3.3 Micro-organisms

Formaldehyde is toxic to a range of micro-organisms and is known to kill viruses, bacteria, fungi, and parasites when used at relatively high concentrations. Consequently, it has long been employed as a disinfectant and parasiticide in many industries. For example, in Australia, formaldehyde is commonly used for the control of fungal infections, protozoan and metazoan ectoparasites in aquaculture systems, and as a general disinfectant in animal husbandry situations.

Unicellular micro-organisms, such as algae and protozoa appear to be most sensitive to formaldehyde, with acute lethal concentrations ranging from 0.3 mg/L to 22 mg/L. Various species of microscopic fungi including *Aspergillus*, *Scopulariopsis* and *Penicillium crustosum* are also sensitive to formaldehyde gas, with 100% of spores exposed to 2 ppm of gaseous formaldehyde reported to be killed within 24 hours (IPCS, 1989).

A few studies summarized in the IPCS (1989) report indicate formaldehyde can negatively impact soil microbial biomass and activity. One study reports that formaldehyde was able to inhibit the enzyme which catalyses deamination of the amino acid L-histidine, an important nitrogen source for plants and microbes. Another study reported a significant reduction in bacterial populations in soils near industrial sites polluted with formaldehyde and in soils on sites using urea-formaldehyde fertilizers. Several studies also cited in the IPCS report (IPCS, 1989) indicated some strains of bacteria (e.g. *Psuedomonas*) are able to utilize formaldehyde as a carbon source.

Sewage micro-organisms were inhibited at 30 mg/L in a Closed Bottle test suggesting that sewage treatment plant performance would only be impaired at relatively high concentrations of formaldehyde (Gerike and Gode, 1990).
There is some evidence that certain soil mesofauna may be adversely affected by formaldehyde. The IPCS (1989) report indicated that nematodes in peat were killed by application of formalin (37% formaldehyde solution) at 179 mL/m³. However, in another study, cereal cyst nematode populations significantly increased following soil treatment with formalin, presumably due to suppression of fungal parasites, which attack the nematodes.

### 8.3.4 Summary

For aquatic organisms (Table 8.2), the available data indicate daphnia to be the most sensitive species, with EC50 of 5.8 mg/L. The most sensitive fish species is striped bass, with mean LC50 values of 16.9 mg/L. The responses of various species of amphibians are similar to those of fish, with LC50 ranging from 10 mg/L to 20 mg/L. While no EC50 endpoints are available, the data suggest that formaldehyde is only slightly to moderately acutely toxic to aquatic plants and algae.

**Table 8.2: Summary of the most sensitive aquatic species to formaldehyde based on acute toxicity endpoints**

<table>
<thead>
<tr>
<th>Aquatic organisms</th>
<th>Species</th>
<th>Endpoint</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fish</td>
<td>Stripped bass (<em>Morone saxatilis</em>)</td>
<td>96-h LC50 = 16.9 mg/L</td>
</tr>
<tr>
<td>Amphibians</td>
<td><em>Rana pipiens</em></td>
<td>72-h LC50 = 8.7 mg/L</td>
</tr>
<tr>
<td>Aquatic invertebrates</td>
<td><em>Daphnia pulex</em></td>
<td>96-h EC50 = 5.8 mg/L</td>
</tr>
<tr>
<td>Molluscs</td>
<td><em>Corbicula sp</em></td>
<td>96-h EC50 = 35 mg/L</td>
</tr>
<tr>
<td>Algae</td>
<td>Freshwater green algae</td>
<td>No reliable data</td>
</tr>
</tbody>
</table>

For terrestrial organisms (Table 8.3), the available data indicate that formaldehyde is practically non-toxic to birds exposed to formaldehyde in food. Formaldehyde in air and fog water has potentially adverse effects on some plant species when exposed. The lowest effect concentration of formaldehyde in air was 65 ppb and 14.9 ppb in fog. Gaseous formaldehyde also kills the spores of microscopic fungi within 24 hours at concentrations of 2 ppm. Pollen grains of *Lilium longiflorum*, exposed to 0.35 ppm of formaldehyde gas showed a significant reduction in pollen-tube length after 5 hours. Pollen grains exposed to formaldehyde concentrations of 2.3 ppm showed a decrease in tube length after 1 hour of exposure.
<table>
<thead>
<tr>
<th>Terrestrial organisms</th>
<th>Effects</th>
<th>Endpoint</th>
</tr>
</thead>
<tbody>
<tr>
<td>Northern bobwhite quail ((Colinus virginianus))</td>
<td>14-d LD50</td>
<td>790 mg/kg</td>
</tr>
<tr>
<td>Bean plants ((Phaseolus vulgaris))</td>
<td>Imbalance in shoot and root growth after up to 4 weeks exposure</td>
<td>65 ppb (fog)</td>
</tr>
<tr>
<td>Rapeseed ((Brassica rapa))</td>
<td>Reduction in leaf area, leaf and stem dry weight, and flower and seedpod numbers</td>
<td>14.9 ppb (fog)</td>
</tr>
<tr>
<td>Lilium longiflorum</td>
<td>Reduction in pollen tube length after 5 hours</td>
<td>0.35 ppm (gas)</td>
</tr>
<tr>
<td>Microscopic fungi ((Scopulariopsis and Penicillium))</td>
<td>100% mortality in 24 hours</td>
<td>2 ppm (gas)</td>
</tr>
</tbody>
</table>
9. Kinetics and Metabolism

9.1 Absorption

Inhaled formaldehyde is mostly deposited and readily absorbed in the regions of the upper respiratory tract with which it comes into initial contact, owing to its high water solubility and reactivity with biological macromolecules (Heck et al., 1983; Swenberg et al., 1983). A complex relationship between nasal anatomy, ventilation and breathing patterns (nasal or oronasal) determines where in the upper respiratory tract formaldehyde absorption occurs in species. In rodents, which are obligate nasal breathers, deposition and absorption occurs primarily in the nasal passage. In contrast, primates are oronasal breathers, and although absorption and deposition is likely to occur primarily in the oral mucosa and nasal passages it can also occur in the trachea and bronchus (Monticello et al., 1991). At the site of contact, formaldehyde has been shown to produce intra and intermolecular crosslinks with proteins and nucleic acids (Casanova et al., 1989; 1991).

There are no direct toxicokinetic studies on formaldehyde following oral or dermal administration. However, the use of physiochemical and toxicological data allows a qualitative assessment of the toxicokinetic behaviour of formaldehyde to be made for these routes of exposure. On the basis of its low molecular weight, high water solubility and moderate octanol/water partition coefficient (Log P) value, it is likely that significant absorption via the oral route would occur. These physiochemical characteristics of formaldehyde would also favour dermal absorption. The observation of skin sensitisation in animal studies (Section 10.3) indicates that such absorption can occur.

9.2 Distribution

No increase in formaldehyde concentration was seen in blood in humans, rats, and monkeys following exposure to concentrations of 1.9 ppm (2.3 mg/m³), 14.4 ppm (17.3 mg/m³) and 6 ppm (7.2 mg/m³) gaseous formaldehyde, respectively (IPCS, 2002). This has been attributed to the deposition of formaldehyde principally in the respiratory tract and its rapid metabolism (Heck et al., 1985; Casanova et al., 1988). The half-life in circulation has been shown to range from 1 to 1.5 minutes between animal species following intravenous administration (Rietbrock, 1969; McMartin et al., 1979). Such rapid metabolism would inhibit systemic distribution of formaldehyde.

9.3 Metabolism

Formaldehyde can be metabolised by a variety of pathways: (1) incorporation into the one-carbon pool pathway, (2) conjugation to glutathione then oxidation by formaldehyde dehydrogenase, and (3) oxidation by the peroxisomal enzyme catalase (Kallen & Jencks, 1966; Uotila & Koivusalo, 1974a; Waydhas et al., 1978).

Formaldehyde is rapidly metabolised to formate by a number of widely distributed cellular enzymes, the most important of which is formaldehyde
dehydrogenase that metabolises the formaldehyde-glutathione conjugate to formate. Formaldehyde dehydrogenase has been detected in human liver and red blood cells and a number of tissues in the rat including respiratory and olfactory epithelium, kidney and brain (Uotila & Koivusalo, 1974b; Casanova-Schmitz et al., 1984). Both formaldehyde and formate are incorporated into the one-carbon pathways involved in the biosynthesis of protein and nucleic acid via direct reaction with tetrahydrofolate. Formaldehyde can also be oxidised to formic acid by catalase, though this reaction probably represents a minor pathway for formaldehyde metabolism. Additionally, it should be noted that formaldehyde is itself formed endogenously during the metabolism of amino acids and xenobiotics (Johansson & Tjalve, 1978; Upreti et al., 1987).

9.4 Elimination and excretion

Due to the rapid metabolism of formaldehyde, much of the material is eliminated as carbon dioxide in expired air shortly after exposure, and as formate in urine (Keefer et al., 1987; Heck et al., 1983). Elimination of total radioactivity following exposure of rats to [14C]-formaldehyde indicated that 40% of the inhaled [14C] was excreted in expired air, 17% in urine and 5% in faeces. The rest of the radioactive label (35% to 39%) remained in the tissues and carcass, presumably as products of metabolic incorporation (Heck et al., 1983).
10. Effects on Laboratory Mammals and Other Test Systems

This chapter is a summary of the health effects of formaldehyde. It is mainly based on the Concise International Chemical Assessment Document (IPCS, 2002), the Toxicological Profile (ATSDR, 1999) and the SIDS Initial Assessment Report (OECD, 2002). Articles published post 1998 are summarised in this chapter.

10.1 Acute toxicity

Formaldehyde has been found to be moderately toxic in laboratory animals exposed via inhalation, dermal and oral routes. The acute toxicity of formaldehyde has been studied in several animal species and is summarised in Table 10.1.

<table>
<thead>
<tr>
<th>Route</th>
<th>Species</th>
<th>Measure</th>
<th>Result</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inhalation</td>
<td>Rat</td>
<td>LC50</td>
<td>480 ppm</td>
<td>Nagorny et al., 1979</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(4 hours)</td>
<td>(578 mg/m³)</td>
<td></td>
</tr>
<tr>
<td>Inhalation</td>
<td>Mouse</td>
<td>LC50</td>
<td>414 ppm</td>
<td>Nagorny et al., 1979</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(4 hours)</td>
<td>(497 mg/m³)</td>
<td></td>
</tr>
<tr>
<td>Oral</td>
<td>Rat</td>
<td>LD50</td>
<td>800 mg/kg bw</td>
<td>Smyth et al., 1941</td>
</tr>
<tr>
<td>Oral</td>
<td>Guinea-pig</td>
<td>LD50</td>
<td>260 mg/kg bw</td>
<td>Smyth et al., 1941</td>
</tr>
<tr>
<td>Dermal</td>
<td>Rabbit</td>
<td>LD50</td>
<td>270 mg/kg bw</td>
<td>Lewis &amp; Tatken, 1980</td>
</tr>
</tbody>
</table>

Clinical signs of toxicity, observed following single exposure of formaldehyde vapour at concentrations > 100 ppm (> 120 mg/m³) were hypersalivation, acute dyspnoea, vomiting, muscular spasms, and death (Skog, 1950; Horton et al., 1963; Bitron & Aharonson, 1978).

10.2 Corrosivity/Irritation

10.2.1 Skin and eye irritation

With the exception of a recently conducted eye irritation study by Maurer et al. (2001) summarised below, the limited data available for skin and eye irritation are from old briefly reported studies. These studies state that aqueous solutions of 0.1% to 20% formaldehyde were irritating to rabbit skin (NRC, 1981), and aqueous solutions of 5% and 15% formaldehyde were irritating to rabbit eyes (Carpenter and Smyth, 1946). In a mouse repeated dermal study (see Section 10.4.3), skin irritation was observed with 0.5% formaldehyde solution and above. No skin irritation was seen at 0.1% (Krivanek et al., 1983). The SIAR (OECD,
2002) and IPCS report (1989) concluded that although formaldehyde solution is known to be a primary skin and eye irritant in animals this is based on anecdotal evidence rather than robust animal studies. Skin irritation studies in animals using gaseous formaldehyde were not found.

In a recent well-reported study, Maurer et al. (2001) investigated the ocular irritation of formaldehyde solution in a series of experiments. In a low-volume eye test (LVET), 10 µl of 37% formaldehyde solution was applied directly to the cornea of 12 rabbits. Eyes were macroscopically examined to determine the degree and extent of irritation to the cornea, iris and conjunctiva at 3 hours post instillation and 1, 4, 7, 14, 21, 28 and 35 days after treatment. The maximum score obtainable was 110 (cornea = 80, iris = 10, conjunctiva = 20). Additionally, from this group of 12 rabbits, 3 animals were sacrificed at 3 hours, 1, 3 and 35 days post-instillation, and the eyes were removed, sectioned, stained and examined by light microscopy to determine the extent of corneal and conjunctiva changes (≤5% slight, 6% to 30% mild, 61% to 90% marked and 91% to 100% severe). Macroscopic observations showed that formaldehyde solution produced irritation of the cornea, conjunctiva and iris 3 hours after application. An irritation score of 53.5/110 was determined. This value increased to a maximum of 80.0/110 (time of scoring not reported). Microscopic examination indicated that severe irritation had occurred to the cornea and conjunctiva. Observations included erosion, denudation and oedema to the corneal and conjunctival epithelium. “Necrosis/loss” of corneal keratocytes was also observed 1 day after instillation in all 3 rabbits. At study termination on day 35, both macro- and microscopic examination revealed corneal irritation in all animals.

In a further experiment, Maurer et al. (2001) determined the initial corneal injury 3 hours and 1-day post-instillation of 10 µl of 37% formaldehyde solution by post-mortem quantitation of dead corneal epithelium and kerocytes, using a scanning laser confocal microscopy. Post mortem quantitation indicated that corneal injury extended deeply into the stroma, at times to 93.2% of the corneal thickness on day 1. Dead corneal epithelial cells and kerocytes were also observed on day 1.

10.2.2 Respiratory irritation

No internationally validated animal tests are currently available for this endpoint. Data are available from a study investigating effects on the mucociliary clearance and histopathological changes in Fischer 344 (F344) rats using light microscopy after a single 6-hour exposure to 0, 2, 6 or 15 ppm (0, 2.4, 7.2 or 18 mg/m³) gaseous formaldehyde (Morgan et al., 1986). At 15 ppm, slowing or cessation of mucus flow was detected in the nasal tract along with separation of epithelial cells and intravascular margination and local tissue infiltration by neutrophils and monocytes. No effects were seen at 2 or 6 ppm formaldehyde. However, in a study using electron microscopy to investigate histopathological changes in the nasal tract of F344 rats following a single exposure to formaldehyde (Monteiro-Riviere & Popp, 1986), loss of microvilli in ciliated cells, autophagic vacuoles in basal cells and cytoplasmic vacuoles in most cell types were seen at ≥6 ppm. Although altered cilia were seen at 0.5 ppm and 2.2 ppm (0.6 mg/m³ and 2.6 mg/m³), such changes were also occasionally reported in control animals. Consequently, it cannot be determined whether these findings at 0.5 ppm and 2.2 ppm are attributable to formaldehyde exposure or inter-animal variations.
In an Alarie assay in Swiss mice (Kane & Alarie, 1997), a 10-minute exposure to 3.1 ppm (3.7 mg/m$^3$) formaldehyde was calculated to depress the respiratory rate by 50% (RD50 value). Additionally, tracheal cannulation of mice was seen to produce a minimal decrease in respiratory rate; 4.2% compared to 54% in uncannulated controls. In a recent modified Alarie assay (Nielsen et al., 1999), respiratory patterns and parameters were continuously measured in BALB/c mice exposed (head only) to formaldehyde at concentrations ranging from 0.2 to 13 ppm (0.24 to 15.6 mg/m$^3$) for 30 minutes. A 10-minute RD50 of 4 ppm (4.8 mg/m$^3$) was calculated, which was reported to be due to irritation of the upper respiratory tract. At concentrations above the RD50 value both upper respiratory tract irritation and bronchoconstriction were involved in the decrease in respiratory rate.

10.3 Sensitisation

10.3.1 Skin

The skin sensitisation potential of formaldehyde solutions has been investigated in numerous studies in the guinea-pig and mouse. A positive response to formaldehyde solution was seen in a large number of these studies. For example, strong positive responses to formaldehyde solution were observed in well-conducted guinea-pig maximisation tests, a Buehler occluded patch test and murine local lymph node assays (Kimber et al., 1991; Hoechst, 1994; Hilton et al., 1996). The details of the studies were summarised in ATDSR (1999).

Furthermore, the cytokine secretion profile of formaldehyde was recently determined in mice and compared with that produced by a reference skin and respiratory sensitiser. Previous studies by the authors had shown that skin sensitisers stimulated a cytokine profile associated with the activation of T helper type 1 cells, compared to T helper type 2 cells for respiratory sensitisers. Topical exposure of mice to a 50% formaldehyde solution resulted in a cytokine secretion profile identical to that induced by the reference contact allergen (Dearman et al., 1999).

There is no evidence in inhalation studies with rats, mice, hamsters or monkeys that formaldehyde gas induces skin sensitisation.

10.3.2 Respiratory

No internationally validated animal test is currently available that allows prediction of the ability of a chemical to induce respiratory sensitisation. However, data are available from non-validated studies investigating this endpoint in mice and guinea pigs.

Formaldehyde was negative in immunoglobulin-E (IgE) tests in the mouse (Potter & Wederbrand, 1995; Hilton et al., 1996;) and guinea-pig (Lee et al., 1984). This predictive test method for assessment of respiratory sensitisation potential measures induced changes in serum concentration of IgE following topical exposure of mice to the test chemical. Furthermore, in a study investigating the cytokine secretion profile in mice (Dearman et al., 1999), topical exposure to formaldehyde did not induce a profile comparable to that of the reference respiratory sensitis (i.e. secretion of cytokines associated with selective activation of T helper type 2 cells).
Data is also available from studies that investigated whether pre-exposure to formaldehyde may enhance allergic responses to ovalbumin. Compared to controls, a statistically significant increase in specific anti-ovalbumin antibody levels were seen in mice exposed to 1.67 ppm (2.00 mg/m³) formaldehyde daily for 10 days (Tarkowski & Gorski, 1995), and guinea-pigs to 0.25 ppm (0.3 mg/m³) daily for 5 days (Riedel et al., 1996), prior to induction then bronchial challenge with ovalbumin.

### 10.4 Repeat dose toxicity

Repeated dose studies are available via the inhalation, oral, and dermal routes of exposure.

#### 10.4.1 Inhalation

For repeated inhalation exposure the database is extensive. Studies have generally been conducted in rats, though data are also available in mice, hamsters and monkeys. These studies clearly show that the target organ following formaldehyde exposure is the nasal tract, where effects observed have included alterations in mucociliary clearance, cell proliferation and histopathological changes to the nasal epithelium.

In the only study that investigated effects on the nasal mucociliary apparatus (Morgan et al., 1986), male F344 rats were exposed to 0, 0.5, 2, 6, or 15 ppm (0, 0.6, 2.4, 7.2 or 18 mg/m³) formaldehyde 6 hours/day, 5 days/week for up to 2 weeks. Inhibition of mucociliary clearance (i.e. reduced mucous flow rate) was observed at 6 ppm and above in the 9-day exposure group. The inhibitory effect of formaldehyde was mostly observed in the lateral aspect of the nasoturbinate and dorsal or medial aspects of the maxilloturbinate. No evidence of reduced mucous flow rate was seen at 2 ppm.

**Short-term and sub-chronic exposure studies**

In the rat, studies with exposure durations from 2 days to a lifetime are available. An overview of the results seen in short-term to sub-chronic exposure studies is presented below [see CICAD (IPCS, 2002) for a more detailed summary of the data].

In short-term to sub-chronic exposure studies with exposure periods of 6-8 hours/day, 5 days/week, conclusive evidence of squamous metaplasia and/or cell proliferation of the nasal epithelium were seen with light microscopy at \( \geq 3.2 \) ppm (3.8 mg/m³) formaldehyde for 2-3 days exposure (Swenberg et al., 1983; Monteiro-Riviere and Popp, 1986; Morgan et al., 1986; Cassee et al., 1996); \( \geq 5 \) ppm (6 mg/m³) formaldehyde in a 4-week study (Wilmer et al., 1987); \( \geq 6.2 \) ppm (7.4 mg/m³) formaldehyde in a 6-week study (Monticello et al., 1991); and \( \geq 3 \) ppm (3.6 mg/m³) formaldehyde in studies with exposure durations of approximately 13-weeks (Feron et al., 1988; Woutersen et al., 1987; Zwart et al., 1988; Wilmer et al., 1989; Casanova et al., 1994).

In these short-term to sub-chronic studies, the severity of histopathological changes was seen to increase with concentrations (e.g. in the study by Monticello et al. (1991). Epithelial cell vacuolar degeneration, individual cell necrosis, epithelial exfoliation and multifocal erosions were observed at \( \geq 10 \) ppm.
neurotoxic). Some studies (Wilmer et al., 1986; 1987) indicated that it is the concentration rather than the total dose (i.e. concentration x time of exposure) that determines the severity of this cytotoxicity.

In a rat study with a near continuous exposure period (i.e. 22 hours/day), hyperplasia and metaplasia were observed in the nasal epithelium following 3 consecutive days exposure to 3.1 ppm (3.7 mg/m³) formaldehyde (Reuzel et al., 1990).

In a recent study, a decrease in testicular zinc (52% - 65%) and copper concentrations (40-68%), increase in testicular iron concentrations (17% – 76%) and reductions in body weight gain (38% – 87%) were seen in male Wistar rats exposed to 10.2 or 20.3 ppm (12.2 or 24.4 mg/m³) formaldehyde gas 8 hours/day. 5 days/week for 4 and 13 weeks compared to controls (Ozen et al., 2002; exposure concentrations confirmed by personnel communication). The effects seen on these testicular trace elements are considered a secondary non-specific consequence of marked general toxicity, seen as growth retardation.

Data are also available from short-term to sub-chronic studies in other species. Hyperplasia of the nasal epithelium was seen in mice exposed to 15 ppm (18 mg/m³) gaseous formaldehyde 6 hours/day for 3 consecutive days (Swenberg et al., 1986). In a 13-week mouse study (Maronpot et al., 1986), minimal squamous metaplasia was observed in the nasal tract of 1/10 males, but absent in females, exposed to 4 ppm (4.8 mg/m³) formaldehyde 6 hours/day 5 days/week. Data are also available in the monkey. Histopathological changes in the nasal cavity and upper portion of the respiratory tract (trachea and bronchial bifurcation) were seen in male rhesus monkeys exposed to 6 ppm (7.2 mg/m³) formaldehyde 6 hours/day 5 days/week for 1 or 6 weeks (Monticello et al., 1989). A comparative study of the effects of near continuous exposure to formaldehyde (i.e. 22 hours/day 7 days/week) for 26 weeks is available in cynomologus monkeys, F344 rats and Syrian hamsters (Rusch et al., 1983). Comparable effects were seen between F344 rats and cynomologus monkeys at 3 ppm (3.6 mg/m³) formaldehyde. In contrast, no conclusive evidence of histopathological changes in the respiratory tract was observed in hamsters at 3 ppm. Together, the data from these two studies suggests that rats and monkeys may be equally susceptible to epithelial damage from formaldehyde exposure, but a wider regional distribution of formaldehyde occurs in the upper respiratory tract of (rhesus) monkeys than in rats.

Although no obvious clinical signs of neurotoxicity or histopathological changes in the brain have been observed in rodent inhalation studies, a recent sub-chronic inhalation study is available investigating the effect of formaldehyde on behaviour in male and female Wistar rats (Pitten et al., 2000). Compared to controls, exposure to 2.6 or 4.6 ppm (3.1 or 5.5 mg/m³) formaldehyde 10 min/day, 7 days/week for 13 weeks was seen to produce a statistically significant increase in the time to find the food, and number of mistakes made in a maze. However, the small group sizes (13-14/dose), assessment of a single neurobehavioural trait and absence of dose-response relationship for observed effects prevent any reliable conclusions being drawn from the data on the neurotoxic potential of formaldehyde.
Long-term exposure studies

Data are available from seven chronic inhalation studies in rodents. All these studies, which employed an exposure period of 6 hours/day 5 days/week, are presented below.

In a study by Kerns et al. (1983), F344 rats and B6C3F1 mice (approximately 120 per species per sex per concentration) were exposed to 0, 2, 5.6 or 14.3 ppm (0, 2.4, 6.7 or 17.2 mg/m³) formaldehyde for up to 24 months. In rats, rhinitis, epithelial dysplasia and squamous metaplasia of the nasal tract was observed at 2 ppm and above. In mice, histological changes were seen at 5.6 ppm and above, along with rhinitis in a “few” animals at 2 ppm (no further details available).

In a study by Appelman et al. (1988), male Wistar rats (40 per concentration) were exposed to 0, 0.1, 1 or 9.4 ppm (0, 0.12, 1.2 or 11.8 mg/m³) formaldehyde for 12 months. Rhinitis, hyperplasia and squamous metaplasia were observed in animals at 9.4 ppm only.

In a study by Woutersen et al. (1989), male Wistar rats (30 per concentration) were exposed to 0, 0.1, 1 or 9.8 ppm (0, 0.12, 1.2 or 11.8 mg/m³) formaldehyde for up to 28 months. At 9.8 ppm rhinitis, disarrangement of the olfactory epithelium, hyperplasia and squamous cell metaplasia were observed in the nasal tract. No histopathological changes were observed at 0.1 or 1.0 ppm.

In a study by Monticello et al. (1996), male F344 rats (90-150 per concentration) were exposed to 0, 0.7, 2, 6, 10 or 15 ppm (0, 0.84, 2.4, 7.2, 12 or 18 mg/m³) formaldehyde for up to 24 months, and effects determined at seven sites within the nasal tract: anterior lateral meatus, posterior lateral meatus, anterior mid-septum, posterior mid-septum, anterior dorsal septum, medial maxilloturbinate and maxillary sinus. At ≥6 ppm hyperplasia and squamous metaplasia were observed in the nasal tract, mainly at the anterior lateral meatus. No histopathological changes were observed in the nasal tract at 0.7 or 2 ppm.

In a study by Kamata et al. (1997), male F344 rats (36 per concentration) were exposed to 0, 0.3, 2.17 or 14.85 ppm (0, 0.36, 2.6 or 17.8 mg/m³) formaldehyde for up to 28 months. At ≥2.17 ppm a statistically significant increase in squamous metaplasia in the nasal tract was observed both in the presence and absence of epithelial hyperplasia. At 0.3 ppm, although not statistically significant, squamous metaplasia was seen in the absence (1/5 animals at 18 months) and presence of hyperplasia (1/5 animals at 24 months and 3/11 animals at 28 months). However, the small group sizes and number of animals at interim sacrifice limits the significance that can be attached to the results of this study.

Hyperplasia and squamous metaplasia were observed in the nasal tract of rats in studies by Sellakumar et al. (1985) and Holmstrom et al. (1989) that are of limited value as they only employed a single (high) exposure level; 14 and 12 ppm (16.8 and 14.4 mg/m³) formaldehyde, respectively.

In these studies no conclusive evidence of systemic toxicity following inhalation exposure to formaldehyde was seen. The principal non-neoplastic effect observed in animals after repeated inhalation exposure was histological changes at the site of contact (i.e. in the nasal tract) due to irritation. The available data provide a dose-response range for histopathological changes in the nasal tract of rats, with effects being seen at 2 ppm (2.4 mg/m³) and above. Overall, the data also indicate
similar effects are observed irrespective of exposure period. Although histopathological changes to the nasal tract were observed in rats at 0.3 ppm following 28 months exposure (Kamata et al., 1997), study limitations reduce the significance that can be attached to the data. Furthermore, no histopathological changes were seen at 0.7 and 1 ppm in studies of 24 and 28 months duration, respectively (Monticello et al., 1996; Woutersen et al., 1989). Consequently, a LOAEC of 2 ppm (2.4 mg/m³) is identified for histopathological changes to the nasal tract from an 18- and 24-month rat studies (Swenberg et al., 1980 and Kerns et al., 1983, respectively) with a NOAEC of 1 ppm (1.2 mg/m³) identified from a rat 28-month study (Woutersen et al., 1989).

10.4.2 Oral

Data are available from studies in rats and a dietary study in dogs. In short-term drinking water studies in rats, histopathological changes to the forestomach were seen at 125 mg/kg bw/day in a 28-day study following administration of formaldehyde solution (95% paraformaldehyde prill and 5% water) at dose levels of 5, 25, 125 mg/kg bw/d (Til et al., 1988). In contrast, a reduction in body weight gain was seen in a 13-week study [administering formaldehyde solution (95% paraformaldehyde prill and 5% water) in drinking water at 0, 50, 100, 150 mg/kg bw/d] at 100 mg/kg bw/day but no treatment-related histopathological changes were reported up to 150 mg/kg bw/day (Johannsen et al., 1986). A 28-day study is also available investigating the immunotoxicity of formaldehyde solution (28.44%) in male rats (Vargova et al., 1993). Animals were administered 0, 20, 40 or 80 mg/kg bw/day formaldehyde by gavage. Compared to controls, the only effects seen were a statistically significant increase in haematocrit concentration and decrease in body weight gain at $\geq 40$ and 80 mg/kg bw/day, respectively. However, the magnitude of changes were $< 10\%$ and are not considered biologically significant. Additionally, although lymph node weight was significantly increased at 80 mg/kg bw/day no histopathological changes were seen in the lymph node organs. Consequently, this study is not considered to provide conclusive evidence that formaldehyde possesses an immunosuppressive potential.

Data are also available from long-term drinking water studies in the rat. In a study by Tober et al. (1989), male and female Wistar rats (20 per sex per concentration) were administered formaldehyde solution in drinking water at concentrations of 0, 0.02, 0.1, 0.5% (approximately 0, 10, 50 or 300 mg/kg bw/day formaldehyde solution) for up to two years. However, the small group sizes employed and significant increase in mortality rate at the top dose (45% females and 55% males had died at 12 months) limits the value of this study for identification of a robust no-effect level. In contrast, a 2-year study by Til et al. (1989) was both well conducted and reported. In this study, groups of male and female Wistar rats (70 per sex per concentration) were administered formaldehyde solution (95% paraformaldehyde prill and 5% water) at dose levels of approximately 0, 1.2, 15 or 82 mg/kg bw/day in males and 0, 1.8, 21 or 109 mg/kg bw/day females for up to 2 years. At the top dose, histopathological changes including hyperplasia, hyperkeratosis, and focal ulceration of the forestomach epithelium, as well as focal atrophic gastritis, glandular hyperplasia and ulceration in the glandular stomach, were observed in both sexes. A reduction in body weight gain, liquid intake and an increased incidence in renal papillary necrosis were also seen in
both sexes at the top dose. As these findings were not seen in other studies they are considered likely to be a secondary consequence of the severe effects seen in the stomach. No treatment-related effects were seen in either sex in the mid and low dose groups.

In a 90-day oral study in dogs administering formaldehyde solution (95% paraformaldehyde prill and 5% water) in drinking water at 0, 50, 100 mg/kg bw/d (Johannsen et al., 1986), no treatment-related effects were reported up to 100 mg/kg bw/day. The absence of toxicity in both the dogs and rats in this study suggests that the target intakes may not have been achieved. Furthermore, it is not reported whether histopathological examination of the stomach was conducted in this study.

Therefore, from the available data there is no conclusive evidence of systemic toxicity following oral administration of formaldehyde. The principal non-neoplastic effect observed in animals after repeated oral dosing was irritation at the site of contact (i.e. fore- and glandular-stomach). From the available data, a NOAEL of 15 mg/kg bw/day and a LOAEL of 82 mg/kg bw/day were identified for histopathological changes to the stomach from a well-conducted 2-year oral study in the rat (Til et al., 1989).

10.4.3 Dermal

The limited data available on the repeat dermal toxicity of formaldehyde solution are from briefly reported mouse initiation/promotion studies (Krivaneck et al., 1983; Iversen, 1986). None of these studies showed evidence of systemic toxicity. The study by Krivanek et al. (1983) contained a briefly reported dose ranging test. Groups of female CD-1 mice (number/dose not reported) received 100 \( \times \)1 of a 10%, 2% or 1% formaldehyde solution in acetone (equivalent to 10, 2 and 1 mg) 5 days/week for 2 weeks or, 0.5% or 0.1% (equivalent to 0.5 or 0.1 mg) 5 days/week for 3 weeks. Skin irritation was observed at 0.5% and above, whose severity increased with concentration. Systemic toxicity was not seen at any dose level. However, the limited details provided prevent identification of a reliable NOAEL or LOAEL from this study.

10.5 Genotoxicity

10.5.1 In vitro studies

A large number of studies have been conducted in vitro with either gaseous or aqueous formaldehyde and a wide variety of endpoints assessed. An overview of these results is presented below [see IARC (1995) for a comprehensive summary of the available data].

The majority of Ames tests with *Salmonella typhimurium* produced a positive result in the absence of metabolic activation, as seen in more recent studies by Marnett et al. (1985) and Takahashi et al. (1985). Positive results, generally weaker, have also occasionally been reported in the presence of metabolic activation (Connor et al., 1983; Donovan et al., 1983; Pool et al., 1984; Schmid et al., 1986; Temcharoen & Thilly, 1983). Positive results have also been reported in the reverse mutation assay with *Escherichia coli* in the absence of metabolic activation (Takahashi et al., 1985; O’Donovan & Mee, 1993).
In mammalian cells, positive results have been reported in gene mutation assays in the absence of metabolic activation (Goldmacher & Thilly, 1983; Crosby et al., 1988; Liber et al., 1989). Furthermore, loss of heterozygosity analysis following a positive gene mutation assay in the absence of metabolic activation suggested that small-scale chromosomal deletion or recombination is the mechanism of mutation formation in mammalian cells in vitro (Speit and Merk, 2002). Additionally, increased incidences of chromosomal aberrations and SCE have been observed in the presence and absence of metabolic activation (Basler et al., 1985; Galloway et al., 1985; Natarajan et al., 1983; Schmid et al., 1986). Formaldehyde has also been reported to produce DNA damage (single strand breaks), and DNA-protein cross-links (DPX) in the absence of metabolic activation (Ross et al., 1981; Grafström et al., 1984; Grafström, 1990).

10.5.2 In vivo studies

In somatic cells

Data are available from a number of in vivo studies that are presented below. Some of these studies did not follow validated test methods with regard to the tissues examined or the exposure duration employed (i.e. prolonged).

In a bone marrow cytogenetic assay (Natarajan et al., 1983), no increased incidence in chromosome aberrations or micronuclei were seen in male and female CBA mice that received two intraperitoneal injections of formaldehyde solution (concentrations not stated) over 24 hours for total doses up to 25 mg/kg bw. Additionally, no increased incidence in chromosome aberrations was seen in spleen cells. In a further ip bone marrow study (Gocke et al., 1981), no significant increase was seen in micronuclei in male and female Sprague-Dawley following a single injection of formaldehyde solution (concentration not reported) up to 30 mg/kg bw. No information on cytotoxicity was reported for either of these studies.

In an inhalation bone marrow cytogenetic study by Kitaeva et al., 1990 (reported in Russian, summary from IPCS, 2002), a statistically significant increase in the proportion of cells with chromosomal aberrations (chromatid or chromosome breaks) were seen in female Wistar rats exposed to 0, 0.42, or 1.3 ppm (0, 0.50 or 1.56 mg/m³) gaseous formaldehyde for 4 hours/day for 4 months (0.7%, 2.4% and 4%, respectively). No further details are reported in the CICAD (IPCS, 2002). Whereas, no significant increase in chromosome aberrations was seen in the bone marrow of male Sprague-Dawley rats exposed up to 15 ppm (18 mg/m³) formaldehyde 6 hour/day, 5 days/week for 1 or 8 weeks (Dallas et al., 1992). A marginal but statistically significant increase in chromosome aberrations (predominantly chromatid breaks) was seen in pulmonary lavage macrophages in the same study at 15 ppm only following 1 and 8 weeks exposure (7.6% and 9.2%, respectively, compared to 3.5% and 4.8% in controls). No information on cytotoxicity was reported. In a further inhalation study (Kligerman et al., 1984), no significant increase in SCE or chromosome aberrations were seen in peripheral lymphocytes from male and female F344 rats exposed up to 15 ppm (18 mg/m³) formaldehyde 6 hour/day for 5 days. No information on cytotoxicity was reported.

Compared to controls, a statistically significant increase in the proportion of cells with micronuclei and nuclear anomalies (e.g. karyorrhexis, pyknosis, vacuolated
bodies) were observed in the stomach, duodenum, ileum and colon of male Sprague–Dawley rats after a single dose of 200 mg/kg bw formaldehyde solution by gavage (Migliore, et al., 1989). Although no statistically significant effect was seen on the mitotic index in formaldehyde treated rats, the observed increased incidences in micronuclei and nuclear anomalies were reported to clearly correlate with severe local irritation (hyperaemia to haemorrhage), indicating that the observed micronuclei and nuclear anomalies in this study are a likely consequence of cytotoxicity.

Additionally, formaldehyde-induced DPX have been detected in the nasal mucosa of male F344 rats exposed to 0.3, 0.7, 2, 6, or 10 ppm (0.36, 0.84, 2.4, 7.2 or 12 mg/m^3) gaseous formaldehyde for 10 hours (Casanova et al., 1989), and in male rhesus monkeys exposed to 0.7, 2 and 6 ppm for 6 hours (Casanova et al., 1991). Although the precise nature of these cross-links is unknown the possibility that these DPX may produce DNA replication errors cannot presently be dismissed.

**In germ cells**

Data are also available from studies determining the genotoxicity of formaldehyde in germ cells. None of the following studies reported information on cytotoxicity. In an ip study (Fontignie-Houbrechts, 1981), no chromosome aberrations were seen in spermatocytes from male Q mice 8-15 days after a single injection of 50 mg/kg bw formaldehyde solution. A dominant lethal assay was also conducted in this study, in which male Q mice were mated for 7 weeks following a single ip injection of 50 mg/kg bw formaldehyde solution. Compared to controls, a statistically significant increase in post- and pre-implantation loss was seen at week 1 and pre-implantation loss at week 3. However no significant effects was seen on the number of pregnant females or live embryos per dam. Therefore, this study is not considered to have demonstrated a genotoxic effect. Additionally, no significant increase in potential dominant lethal findings were seen after single ip injection of up to 40 mg/kg bw formaldehyde solution (reported to be the ip LD50) to male ICR/Ha mice which were mated for 3 or 8 weeks (Epstein et al., 1972), and following ip injection of 20 mg/kg bw formaldehyde solution (reported to be the ip LD50) to male CD-1 mice which were mated for 8 weeks (Epstein et al., 1968).

In contrast, daily ip injection of rats with 0.125, 0.25 or 0.5 mg/kg bw/day formaldehyde solution (1/4 to 1/16 of the determined ip LD50) for 5 days resulted in a dose related statistically significant increase in epididymal sperm head abnormalities (≥ 106%) and decrease in epididymal sperm count (≥ 41%) at 0.125 mg/kg and above compared to controls (Odeigah, 1997). This study also included a dominant lethal assay in which male rats received daily ip injections of 0, 0.125, 0.25 or 0.6 mg/kg bw/day for 5 days prior to mating for 3 weeks. A significant and dose related decrease was seen in the number of pregnant females mated 1-7 and 8-14 days after treatment of males with ≥ 0.125 mg/kg bw/day (6-19/24 pregnancies compared to 29/30 in the control group), together with a significant dose related increase in the number of dead implants per dam in females mated 1-7 days after treatment of males with ≥ 0.125 mg/kg bw/day (≥ 1.23 compared to 0.43 in controls) and was associated with a corresponding decrease in the number of live foetuses per dam (≤ 5.95 compared to 7.43 in controls).
10.6 Carcinogenicity

The carcinogenic potential of formaldehyde has been investigated in a number of animal studies, predominantly by the inhalation route of exposure.

10.6.1 Inhalation

In the only study conducted in both sexes, groups of F344 rats (approximately 120 per sex per concentration) were exposed to 0, 2.0, 5.6 or 14.3 ppm (0, 2.4, 6.7 or 17.2 mg/m$^3$) formaldehyde 6 hours/day, 5 days week for up to 24 months (Kerns et al., 1983). All animals were subject to a complete and thorough gross and microscopic examination. A significant increased incidence in nasal squamous cell carcinomas was observed in both sexes at 14.3 ppm in the presence of irritation to the nasal tract. The overall incidence in this tumour type at 0, 2.0, 5.6 and 14.3 ppm was 0/118, 0/118, 1/119 and 51/117 in males, and 0/118, 0/118, 1/116 and 52/119 in females, respectively. There were no significant tumour findings in any other tissue. In a further study, groups of male F344 rats (90-150 per concentration) were exposed to 0, 0.7, 2, 6, 10 or 15 ppm (0, 0.84, 2.4, 7.2, 12 or 18 mg/m$^3$) formaldehyde 6 hours/day, 5 days/week for up to 24 months (Monticello et al., 1996). This study is considered the most extensive bioassay conducted to date as proliferative responses were determined at the anterior lateral meatus, posterior lateral meatus, anterior mid-septum, posterior mid-septum, anterior dorsal septum, medial maxilloturbinate and maxillary sinus sites within the nasal tract after 3, 6, 12 and 18 months exposure, as well as at the end of the study. The overall incidence of nasal squamous cell carcinoma in animals was 0/90, 0/90, 0/90, 1/90, 20/90 and 69/147 exposed to 0, 0.7, 2, 6, 10 and 15 ppm, respectively. These tumours were mainly located in the anterior lateral meatus, the posterior lateral meatus and the mid-septum. Nasal polyloid adenomas, located in or adjacent to the lateral meatus, were also observed at 10 ppm (5/90 rats) and 15 ppm (14/147 rats) only. Both tumour types were observed in the presence of irritation to the nasal tract.

Additional bioassays are available in male F344 rats [Tobe et al., 1985 (cited in IPCS, 2002); Kamata et al., 1997]. Exposure-responses in these studies were similar to those seen in the studies by Monticello et al. (1996) and Kerns et al. (1983), that is, an increased incidence in nasal tumours at concentrations > 5.6 ppm (> 6.7 mg/m$^3$) formaldehyde in the presence of irritation (i.e. tumours observed at approximately 14 ppm [16.8 mg/m$^3$] in the studies by Tobe et al., 1985 and Kamata et al., 1997).

Data are available in other strains of rat. In a study in male Sprague-Dawley rats employing a single exposure concentration to formaldehyde (Sellakumar et al., 1985), a significant increase in the incidence of nasal squamous cell carcinoma was observed in animals exposed to 14 ppm (16.6 mg/m$^3$) formaldehyde 6 hours/day, 5 days/week for approximately 24 months compared to controls (0/99 and 38/100, respectively). These tumours were mainly located at the naso-maxillary turbinates and nasal septum and observed in the presence of irritation to the nasal tract. There were no significant tumour findings in any other tissue. In a study in male Wistar rats (26-28/concentration) no significant increase in nasal tumours was observed in animals exposed to 0, 0.1, 1, or 9.8 ppm (0, 0.12, 1.2 or 11.8 mg/m$^3$) formaldehyde 6hours/day, 5days/week for 28 months (Woutersen et al., 1989).
Additional studies are available in male Wistar and female Sprague-Dawley rats (Appelman et al., 1988; Feron et al., 1988; Holmstrom et al., 1989). No significant increase in tumour formation was seen in these studies. However, the small group sizes and/or short duration of exposure to formaldehyde used in these studies limits the significance that can be attached to the data.

Data are also available in other species. In B6C3F1 mice (120 per sex per concentration) exposed to 0, 2.0, 5.6 or 14.3 ppm (0, 2.4, 6.7 or 17.2 mg/m\(^3\)) formaldehyde 6 hours/day, 5 days/week for up to 24 months, squamous cell carcinomas of the nasal tract were seen in two males at the top exposure concentration in the presence of irritation to the nasal tract. No squamous cell carcinomas of the nasal tract were observed in females (Kerns et al., 1983). A study is available in C3H mice that did not observe an increased incidence in pulmonary tumours (Horton et al., 1963). However, the short duration of exposure to formaldehyde (35 weeks), lack of histological examination of the nasal tract and concerns over the health status of the animals, limits the significance that can be attached to the data. In male golden Syrian hamsters (50 per concentration), no tumours were seen in the nasal or respiratory tract, the only tissues examined, of animals exposed to 10 ppm (12 mg/m\(^3\)) formaldehyde, 6 hours/day, 5 days/week for life, or 30 ppm (36 mg/m\(^3\)) 6 hours/day, once a week for life (Dalbey, 1982).

### 10.6.2 Oral

Data are available from drinking water studies in the rat. In the study summarised below by Soffritti et al. (1989) the dose administered were reported in mg/L only. Therefore, the default values in Table 10.2 have been applied to convert mg/L to mg/kg bw. These values are taken from Gold et al. (1984).

**Table 10.2: Default values for dose calculations**

<table>
<thead>
<tr>
<th>Species</th>
<th>Sex</th>
<th>Body weight (kg)</th>
<th>Food intake (g/day)</th>
<th>Water intake (ml/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rat</td>
<td>M</td>
<td>0.5</td>
<td>20</td>
<td>25</td>
</tr>
<tr>
<td>(lifetime studies)</td>
<td>F</td>
<td>0.35</td>
<td>17.5</td>
<td>20</td>
</tr>
<tr>
<td>Rat</td>
<td>M</td>
<td>0.2</td>
<td>20</td>
<td>25</td>
</tr>
<tr>
<td>(other studies)</td>
<td>F</td>
<td>0.175</td>
<td>17.5</td>
<td>20</td>
</tr>
</tbody>
</table>

In the most comprehensive study available (Til et al., 1989), male and female Wistar rats (70 per sex per dose) were administered formaldehyde solution in drinking water for up to 24 months at dose levels that equated to approximately 0, 1.2, 15 or 82 mg/kg bw/day in males and 0, 1.8, 21 or 109 mg/kg bw/day in females. Selected organs of animals in the low and mid dose groups were examined at necropsy (including the stomach), while a complete and thorough gross and microscopic examination was conducted on control and top dose group animals. There were no significant tumour findings in any tissue. Similarly, no significant tumour findings were seen in selected organs (including the stomach) from male and female Wistar rats (20 per sex per dose) administered formaldehyde solution in drinking water for up to 24 months at dose levels that equated to approximately 0, 10, 50 or 300 mg/kg bw/day (Tobe et al., 1989).

In contrast, Soffritti et al. (1989) reported a marked increased incidence in tumours in Sprague-Dawley rats (50 per sex per group) administered 1500 mg/L

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**Formaldehyde**

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(the top dose level) for life. These tumours were leukaemias (all ‘haemolymphoreticular neoplasias’) in males and females (22% and 14%, respectively, compared to 4% and 3% in controls), along with adenomas of the stomach (4%), intestinal adenocarcinomas (2%) and leiomyosarcomas (4%) in males, and intestinal leiomyomas in females (6%). No gastrointestinal tumours were seen in control animals. Using the default values given in Table 10.2, the daily intake of aqueous formaldehyde at the top dose was estimated to have been 75 and 100 mg/kg bw/day in males and females, respectively. However, the pooling of tumour types reported as leukaemias and lymphomas, together with the final report of this study by Soffritti et al. (2002) that reports an increased incidence of these tumours compared to the original summary (with no explanation provided by the authors), means no reliable conclusions can be drawn from the data for these tumours. The later report by Soffritti et al. (2002) provides information on tumour incidences in additional tissues to those reported earlier. Although an increase in testicular interstitial cell adenomas was seen in males, it was not dose related or statistically significant at the top dose. Similarly, although a statistically significant increase was seen for all mammary tumours in females at the top dose (24% compared to 11% in controls), the increase was not dose related, while no dose related or statistically significant increase was seen for specific histologic tumours of the mammary gland.

In an initiation/promotion study in male Wistar rats (Takahashi et al., 1986), papillomas of the forestomach were reported in the presence of irritation in 8/10 animals administered approximately 0.5% formaldehyde solution in drinking water for 32 weeks. No forestomach tumours were seen in control animals.

10.6.3 Dermal

No standard studies are available. Data are available from mouse initiation/promotion studies. No skin tumours were seen in mice (16-20 per sex per dose) topically administered 1% or 10% formaldehyde solution only 3 times/week for 26 weeks (Krivanek et al., 1983,) or 10% formaldehyde solution only 2 times/week for 60 weeks (Iversen, 1986). However, the small group sizes and short duration of exposure to formaldehyde used in these studies prevent any reliable conclusions on the carcinogenic potential of formaldehyde by the dermal route.

10.7 Reproductive toxicity

In the only reproductive study available, a 1-generation study in minks (Li et al., 1999), groups of 12 females were fed 0, 550 or 1100 ppm formaldehyde solution in the diet from 1 month prior to mating (with untreated males) until weaning of kits. However, dose levels of formaldehyde in the feed were determined to be 17, 291 and 662 ppm. No toxicity was observed in parental females. No effect was observed on fertility index or litter size. A statistically significant decrease in kit survival was reported at birth at the top dose (87% compared to 96% in controls). Kit survival was unaffected 3 and 6 weeks post partum. The decrease in kit survival at birth was observed in the absence of a significant increase in mean number dead kits/dam or decrease in live kits/dam. These mean values are considered more reliable markers of adverse effects on fertility. Consequently, it is concluded that no adverse effects on fertility were observed in this study.
However, the absence of parental toxicity means there are concerns that formaldehyde was not robustly tested in this study.

Data are also available from a study by Ward et al. (1984) that investigated the reproductive effect of formaldehyde in both mice and humans. In this study, administration of 100 mg/kg bw/day formaldehyde solution (the only dose level tested) to mice via gavage for 5 consecutive days had no effect on epididymal sperm morphology. Furthermore, in a rat 2-year repeated oral study, no histological changes were observed in the testes or ovaries up to and including the top dose, 82 mg/kg bw/day (Til et al., 1989). Similarly, in repeated inhalation studies of 18 months duration and longer, no histological changes were observed in reproductive organs at the maximum exposure concentration: 14.3 ppm (17.2 mg/m³) in rats and mice (Kerns et al., 1983). Although changes were seen in testicular element concentrations (zinc and copper) at 10.2 ppm (12.2 mg/m³) and 20.3 ppm (24.4 mg/m³) gaseous formaldehyde (see details in section 10.4.1), they were considered to be a secondary non-specific consequence of severe general toxicity; reductions in body weight gain of 38% to 87% (Ozen et al., 2002).

In contrast, effects on male reproductive organs were observed in rodent intraperitoneal (ip) studies. In rats, ip administration of formaldehyde solution for 30 consecutive days resulted in a statistically significant decrease in testicular weight at ≥ 5 mg/kg bw/day (magnitude not reported), a statistically significant decrease in epididymal sperm count (44%), mobility (4%) and viability (17%) at 10 mg/kg bw/day, and histological changes in Leydig cells at ≥ 10 mg/kg bw/day (Chowdhury et al., 1992; Majumder & Kumar, 1995). In further studies, ip administration of formaldehyde for 5 consecutive days resulted in a statistically significant increase in epididymal sperm head abnormalities (≥ 106%) in rats at ≥ 0.125 mg/kg bw/day (Odeighah, 1997), and in mice a statistically significant decrease in sperm mobility (5%) and viability (53%) at ≥ 4 mg/kg bw/day, and sperm count (54%) at ≥ 10 mg/kg bw/day (Yi et al., 2000). However, the relevance of these studies is questionable, as ip administration is not a relevant route of human exposure.

10.8 Developmental toxicity

Data are available from studies via inhalation, oral and dermal routes of exposure.

In an inhalation study (Saïllenfalt et al., 1989), groups of 25 mated female Sprague-Dawley rats were exposed (whole-body) up to 0, 5.2, 9.9, 20 or 39 ppm (0, 6.2, 11.9, 24.0 or 46.8 mg/m³) gaseous formaldehyde for 6 hours/day from day 6 to 20 of gestation. At 39 ppm only, a statistically significant decrease in dam body weight gain (51%) and male (21%) and female (19%) foetal body weight was observed compared to controls. A slight (5%) but statistically significant decrease in male foetal body weight was also seen at 20 ppm. No other treatment-related effects were observed on development. The slight decrease in foetal body weight in males only at 20 ppm is not considered sufficient magnitude to be biologically significant. While the statistically significant decrease in foetal body weight gain at 39 ppm was seen in the presence of a substantial decrease in dam body weight gain, and is therefore considered to be a secondary non-specific consequence of severe maternal toxicity.
In a further inhalation study in Sprague-Dawley rats (Martin, 1990), groups of 25 mated females were exposed (whole-body) up to 10 ppm formaldehyde for 6 hours/day from day 6 to 15 of gestation. At 10 ppm only, a statistically significant reduction in maternal body weight gain was observed (magnitude not reported). No treatment-related effects were seen on development. Thus, formaldehyde did not exhibit developmental toxicity in this study up to a concentration producing maternal toxicity.

In a dietary study (Hurni & Ohder, 1973), groups of 9-10 pregnant Beagle dogs were administered formaldehyde solutions in the diet at dose levels corresponding to approximately 0, 3.1 and 9.4 mg/kg bw/day from day 4 to 56 of gestation. No developmental or maternal toxicity was observed with formaldehyde at either dose level, and therefore, there are concerns that dose levels were not maximised in this study.

A briefly reported dermal study is available in pregnant hamsters (Overman, 1985). Groups of 5-6 pregnant females received a single topical application of 0.5ml of a 37% formaldehyde solution for 2 hours on day 8, 9, 10 or 11 of gestation. A control group of 4 pregnant females received water. An observed increase in resorptions in all formaldehyde treated groups (from 3.2% to 8.1% compared to 0% in controls) was attributed to the severe stress reported in these animals during treatment with formaldehyde. No other maternal or developmental effects were seen. However, the lack of information on the amount of formaldehyde absorbed together with the small group sizes limits the significance that can be attached to the data.
11. Human Health Effects

This chapter is a summary of the health effects of formaldehyde. It is mainly based on the Concise International Chemical Assessment Document (IPCS, 2002), the Toxicological Profile (ATSDR, 1999) and the SIDS Initial Assessment Report (OECD, 2002). Articles published post 1998 are summarised in this chapter.

11.1 Acute toxicity

There are no reports in the literature of human deaths following acute dermal or inhalation exposure to formaldehyde. Human deaths following ingestion of formaldehyde have been reported (Kline, 1925; Levison, 1904). However, the data are from very old case reports (1899-1919) whose reliability cannot be determined. Information is available from more recent cases, which report ulceration and damage along the aero-digestive tract following ingestion of formaldehyde (Allen et al., 1970; Kochhar et al., 1986). Though these cases did not result in death, significant toxicity was observed, requiring drastic medical procedures to be undertaken.

In the case reported by Kochhar et al. (1986), a 26 year old woman who accidentally ingested 45 ml (42.5 grams) 37% formaldehyde solution (equivalent to approximately 700 mg/kg assuming the woman weighed 60 kg) vomited streaks of blood immediately following ingestion. Examination 4 days later showed severe to moderate ulceration of the oesophagus and stomach that resulted in a feeding jejunostomy being performed. In the case reported by Allen et al. (1970) a tracheostomy was conducted on a 14 year old boy following ingestion of approximately 120 ml formaldehyde solution (concentration not reported, nor whether ingestion was accidental or deliberate). Six days later a laparotomy revealed multiple areas of gastric necrosis and, hence, a total gastrectomy and a feeding jejunostomy were performed.

11.2 Irritation/Corrosivity

11.2.1 Skin irritation

The skin irritation potential of formaldehyde solution has been evaluated in a number of international reviews (IPCS, 1989; IARC, 1995; IPCS, 2002; OECD, 2002) and all report formaldehyde solution to be a skin irritant in humans. However, this conclusion is based on anecdotal evidence, with a review of the effects of formaldehyde in solutions on human skin by Malbach (1983) sometimes cited. This review reported that though formaldehyde solution is said to have irritant potential based on human experience, little quantitative data exists. This review also makes the point that since formaldehyde solution is known to cause skin sensitisation, reported irritant effects may be sensitisation effects. Skin rashes were reported by embalmers in the NICNAS survey.
Acute controlled exposure studies of volunteers exposed to airborne formaldehyde at concentrations up to 3 ppm have not found increased reporting of skin irritation symptoms (ATSDR 1999).

11.2.2 Sensory irritation

Sensory irritation is the result of the chemical stimulating the trigeminal nerve endings in the cornea and nasal mucosa, which evokes a stinging or burning sensation in the eyes and upper respiratory tract (nose and throat). This is a receptor mediated mode of action and occurs at relatively low concentrations. Sensory irritation is different to eye and skin irritation/corrosivity used for hazard classification (Section 12.2) and also different from the irritation leading to cytotoxicity, hyperplasia and nasal tumours (Section 10.4.1). These latter examples are a result of physical damage to the cells, whereas sensory irritation is a nerve response.

Formaldehyde exposure has long been associated with irritation to the eyes and upper respiratory tract. Repeated complaints, such as sore eyes and throat by embalmers were reported in the NICNAS survey.

In more recent years, chamber studies have investigated sensory irritation following short-term exposures to known low levels of gaseous formaldehyde.

In chamber studies in healthy and asthmatic volunteers, mild to moderate eye irritation was self-reported following exposure to formaldehyde levels ranging from 0.25 to 3 ppm (0.3 to 3.6 mg/m³) for up to 5 hours, though exposures were generally \( \leq 3 \) hours. Overall, the data from these studies indicate that eye irritation is a more sensitive parameter than nose and throat irritation which was generally self-reported at concentrations \( > 1 \) ppm (Weber-Tscopp et al., 1977; Andersen & Molhave, 1983; Bender et al., 1983; Day et al., 1984; Schachter et al., 1986; 1987; Sauder et al., 1986; 1987; Green et al., 1987; 1989; Kulle et al., 1987; Kulle, 1993; Witek et al., 1987). A summary of these studies can be found in Table 11.1.

It should be noted that a study by Pazdrak et al. (1993) is not included in Table 11.1 because of major methodological shortcomings (e.g. exposures could not be verified as information was not provided regarding the techniques used to generate the aerosol or the methods used to measure formaldehyde). A study by Krakowiak et al. (1998) also has methodological shortcomings and is also not included.

Sensory irritation due to exposure to formaldehyde has rapid onset (Sauder et al. 1987, Yang et al. 2001) and the intensity of effect does not appear to significantly increase with longer exposures (Sauder et al. 1987). This is in accord with the theoretical considerations of sensory irritation where the intensity of response is dependent on the concentration of the substance and not the duration of exposure.

A study is available where exposure to formaldehyde was through modified eye goggles (Yang et al., 2001). Eight volunteers were exposed to 0, 1.65, 2.99 or 4.31 ppm formaldehyde for 5 minutes and eye irritation was self-reported. Individual scores were not reported. Although the higher formaldehyde concentrations resulted in greater eye irritation scores, compared to control exposures irritancy scores were only statistically significant at 1.65 and 4.31 ppm, and only 1.5, 2.5 and 3.0 minutes after the onset of exposure. A study is available
where exposure was via a facemask (Reed & Frigas, 1984). Thirteen subjects who had reported respiratory symptoms to previous exposures of formaldehyde were exposed for 20 minutes to concentrations up to 3 ppm (3.6 mg/m³) formaldehyde. No significant effect was seen on pulmonary function, while self-reports of eye, nose and throat irritation occurred as frequently with clean air as with formaldehyde. A summary of these studies is included in Table 11.1.

With the exception of Weber-Tschopp et al. (1977), Bender et al. (1983), Pazdrak et al. (1993) and Yang et al. (2001), the studies in Table 11.1 also determined the effect of formaldehyde exposure on pulmonary functions. No statistically significant exposure-related effect was seen on forced vital capacity (FVC), forced expiratory volume in 1 second (FEV₁₀), peak expiratory flow rate (PEFR), or the maximal flow at 50% of the vital capacity (MEF50%) in healthy and asthmatic subjects exposed up to 2.0 ppm (2.4 mg/m³) for up to 3 hours.

In contrast, small but statistically significant decreases were seen in FEV₁₀ (2%) and FEFR (7%) in 9 healthy volunteers after 30 minutes exposure to 3 ppm (3.6 mg/m³) formaldehyde but not after 1 or 3 hour exposure periods (Sauder et al., 1986). In a further study by this project team, using the same exposure level and duration, no effects were observed in asthmatics (Sauder et al., 1987). In a study by Green et al. (1987), exposure to 3 ppm formaldehyde for approximately 1-2 hours resulted in small but statistically significant decreases (2% to 3%) in FEV₁₀ and FVC in 22 healthy volunteers. Conversely, no significant deficits in pulmonary function were seen in 16 asthmatic subjects similarly exposed. In a further study by Green et al. (1989), although there was no effect on FVC, a small (6%) decrease in forced expiratory flow rate between 25% and 75% FVC (FEFR₂₅-₇₅) was seen in 24 healthy volunteers exposed to 3 ppm formaldehyde for approximately 2 hours.

Overall, the weight of evidence indicates there is no effect on pulmonary function at concentrations up to 3 ppm, the highest exposure level tested.

A study is available investigating mucous flow rate in the nasal cavity of 16 volunteers exposed to 0.25, 0.4, 0.8 or 1.7 ppm (0.3, 0.48, 0.96 or 2.0 mg/m³) formaldehyde for 4-5 hours (Andersen & Molhave, 1983). Compared to control values, the mucous flow rate was reduced at 0.25 ppm and above. However, the response did not increase at concentrations above 0.4 ppm. The relevance of this finding to human health is unclear.

Data are available from community (Ritchie & Lehnen, 1987; Broder et al., 1988) and workplace studies (Alexandersson & Hedenstierna, 1988; 1989; Holmstrom & Wilhelmsson, 1988; Horvath et al., 1988; Holness & Nethercott, 1989; Uba et al., 1989). However, for determining the irritant potency of formaldehyde, the data from these uncontrolled environments are not considered as reliable as data from controlled chamber studies, due primarily to the unknown contribution of other substances. The workplace and community studies are summarised in Section 11.4.

An extensive review of chamber, community and workplace studies to formaldehyde was recently conducted (Bender, 2002). Overall, this review concluded that it is not possible to identify a specific threshold for irritation, due primarily to the self-reporting of irritation that has no diagnostic accuracy. This is demonstrated by reports of irritation with placebo (zero) exposures in chamber (see Table 11.1) and workplace studies (Holness & Nethercott, 1989). However,
Bender (2002) went on to state that using chamber studies, which provide the highest quality data, some individuals (5% to 20%) begin to sense irritation from 0.5 to 1 ppm (0.6 to 1.2 mg/m$^3$), though the reported response rate is often similar in controls (i.e. a response rate of 20% to 30% is not unusual). At levels of 1 ppm (1.2 mg/m$^3$) and greater, one can attribute responses to formaldehyde with greater certainty. Furthermore, although asthmatics are thought to be more sensitive to irritants, studies by Green et al. (1987), Sauder et al. (1986; 1987) and Witek et al. (1987) have demonstrated that at concentrations of 2 - 3 ppm (2.4 - 3.6 mg/m$^3$) for up to 3 hours, asthmatics were no more sensitive to formaldehyde than non-asthmatics.

Therefore, although formaldehyde is a known eye and upper respiratory tract irritant in humans, the limitations of the available data and subjective nature of sensory irritation do not allow identification of a definitive no-observed-effect level (NOEL). The data from chamber studies demonstrate that the sensory irritation responses at levels of $\varepsilon$ 1 ppm (1.2 mg/m$^3$) can definitely be attributed to formaldehyde. Some individuals begin to sense irritation from 0.5 ppm (0.6 mg/m$^3$), although the response rate is often similar to that reported in controls. Although there is limited evidence that some individuals report sensory irritation at concentrations as low as 0.25 ppm (0.3 mg/m$^3$) the data are very unreliable. Therefore, the LOEL is considered to be 0.5 ppm.

The odour threshold of gaseous formaldehyde varies widely ranging from 0.05 to 1.0 ppm. However, for most people the odour threshold is in the 0.5 to 1.0 ppm range (OECD, 2002).

11.3 Sensitisation

11.3.1 Skin

There are many published case reports and clinical studies that clearly indicate aqueous formaldehyde to be a human skin sensitiser (Lindskov, 1982; Andersen & Molhave, 1983; Cronin, 1991; Ebner & Kraft, 1991; Liden et al., 1993; Trattner et al., 1998). Indeed, formaldehyde solution has long been known as a cause of contact allergy and is included in all standard series for patch testing. Data from several recent patch tests studies are presented below, and support the conclusion that formaldehyde is a skin sensitiser.

Over the last 10 years, 1691 workers with suspected contact dermatitis were referred to the Occupational Dermatology Research and Education Centre (ODREC) in Melbourne and were routinely patch tested using a standard series of 30 common allergens including formalin and formaldehyde releasing preservatives. In addition, formaldehyde resins were included in the test when clinically relevant. The results are summarised in Table 11.2.
### Table 11.1: Irritative effect of gaseous formaldehyde in humans

<table>
<thead>
<tr>
<th>Duration</th>
<th>Physical activity</th>
<th>Number of volunteers</th>
<th>Results</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>90 min</td>
<td>None</td>
<td>15 asthmatics (7 male and 8 females, all non-smokers)</td>
<td><strong>Pulmonary irritation:</strong> No significant change to FEV$<em>{1.0}$ following exposure to 0.007, 0.1 or 0.7 ppm formaldehyde. <strong>Comment:</strong> no significant correlation between exposure levels and change in FEV$</em>{1.0}$ in the group as a whole or volunteers with the highest histamine reactivity. Effects of sensory irritation were not reported.</td>
<td>Harving et al., 1990</td>
</tr>
<tr>
<td>5 hr</td>
<td>None</td>
<td>16 (11 males and 5 females) of which 5 were smokers</td>
<td><strong>Eye irritation and/or dry nose/throat</strong>&lt;br&gt;0.25 ppm 19 %&lt;br&gt;0.4 ppm 31 %&lt;br&gt;0.8 ppm 94 %&lt;br&gt;1.7 ppm 94 %&lt;br&gt;<strong>Pulmonary irritation:</strong> No significant change in FVC, FEV$<em>{1.0}$ and FEFR$</em>{25-75}$ following exposure to 0.25, 0.4, 0.8 or 1.7 ppm formaldehyde. <strong>Comment:</strong> Individuals were asked to rate their level of discomfort. At all exposure levels, the highest individual rating was ‘discomfort’, which was the middle rating. The average rating for all exposures was ‘slight discomfort’. Following the first 2 hours exposure, 0.25 ppm caused more ‘discomfort’ that 0.4 ppm. The results are not published in a peer reviewed journal.</td>
<td>Andersen &amp; Molhave, 1983</td>
</tr>
<tr>
<td>6 min</td>
<td>None</td>
<td>28 at 0 ppm, 12 at 0.35 ppm, 26 at 0.56 ppm, 7 at 0.70 ppm, 5 at 0.90 ppm and 27 at 1.00 ppm</td>
<td><strong>Eye irritation and/or dry nose/throat</strong>&lt;br&gt;0 ppm Not applicable&lt;br&gt;0.35 ppm 42 %&lt;br&gt;0.56 ppm 54 %&lt;br&gt;0.70 ppm 57 %&lt;br&gt;0.90 ppm 60 %&lt;br&gt;1.00 ppm 74 %&lt;br&gt;<strong>Comment:</strong> Eye irritation measured as percentage of subjects whose response time to formaldehyde was less than response time to clean air. Individuals were known to respond to formaldehyde (previously reporting eye irritation) and served as own controls.</td>
<td>Bender et al., 1983</td>
</tr>
</tbody>
</table>
Table 11.1: Irritative effect of gaseous formaldehyde in humans (continued)

<table>
<thead>
<tr>
<th>Duration</th>
<th>Physical activity</th>
<th>Number of volunteers</th>
<th>Results</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 hr</td>
<td>During exposure to 2 ppm intermittent moderate exercise for 8 min every half hour</td>
<td>19 (10 males and 9 females) non-smokers exposed to each concentration except 0.5 ppm (10 volunteers) and 3 ppm (9 volunteers)</td>
<td>Eye irritation 0 ppm: 5 %, 0.5 ppm: 5 %, 1.0 ppm: 26 %, 2.0 ppm: 53 %<em><strong>, 3.0 ppm: 100%</strong></em> Odour perception: &lt; 0.5 ppm: 5 %, 0.5 ppm: 40%*<strong>, 1.0 ppm: 26 %, 2.0 ppm: 58 %</strong>, 3.0 ppm: 78%** Nose/throat irritation:</td>
<td>Kulle, 1993; Kulle et al., 1987</td>
</tr>
<tr>
<td>90 min</td>
<td>None</td>
<td>18 (9 had previous complaints of effects to UFFI)</td>
<td><strong>Eye and throat irritation:</strong> Following exposure to 1 ppm formaldehyde 83 % and 28 % of volunteers reported eye and throat irritation, respectively. <strong>Pulmonary irritation:</strong> No statistically significant change on FVC, FEV₁₀ and FEFR₂₅ – ₇₅ following exposure to 1 ppm formaldehyde. <strong>Comment:</strong> complaints of eye and throat irritation were common in both groups (i.e. those previously complaining of effects to UFFI and those who had not) exposed to formaldehyde.</td>
<td>Day et al., 1984</td>
</tr>
<tr>
<td>1.5 min</td>
<td>None</td>
<td>48</td>
<td>Volunteers exposed to concentrations ranging from 0.3 – 4.0 ppm formaldehyde. The authors report that the irritation threshold was situated between 1 and 2 ppm. No further data available.</td>
<td>Weber-Tschopp et al., 1977</td>
</tr>
<tr>
<td>5 min</td>
<td>None</td>
<td>8 (4 males and 4 females) of which 1 male and 1 female were smokers</td>
<td><strong>Eye irritation:</strong> Mild to moderate irritation ratings seen following exposure to 1.65, 2.99 and 4.31 ppm. Severity was greatest 1.0 – 1.5 minutes after the onset of exposure and then declined. At 5 minutes, eye irritation ratings to 1.65 ppm and clean air (0 ppm) were comparable. <strong>Comment:</strong> Eye irritation reported to clean air with a slight increase in intensity seen with exposure duration.</td>
<td>Yang et al., 2001</td>
</tr>
</tbody>
</table>
Table 11.1: Irritative effect of gaseous formaldehyde in humans (continued)

<table>
<thead>
<tr>
<th>Duration</th>
<th>Physical activity</th>
<th>Number of volunteers</th>
<th>Results</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Slight to severe:</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Eye irritation</td>
<td>Odour perception</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(0 ppm (R))</td>
<td>(0 ppm (E))</td>
</tr>
<tr>
<td>40 min</td>
<td>R = rest</td>
<td>15 non-smokers</td>
<td>0 %</td>
<td>47 %</td>
</tr>
<tr>
<td></td>
<td>E = 10 min</td>
<td></td>
<td>0 ppm (R)</td>
<td>0 %</td>
</tr>
<tr>
<td></td>
<td>moderate exercise</td>
<td></td>
<td>0 ppm (E)</td>
<td>7 %</td>
</tr>
<tr>
<td></td>
<td>(conducted on a</td>
<td></td>
<td>2.0 ppm (R)</td>
<td>53 %</td>
</tr>
<tr>
<td></td>
<td>different day)</td>
<td></td>
<td>2.0 ppm (E)</td>
<td>53 %</td>
</tr>
</tbody>
</table>

**Pulmonary irritation:** Pulmonary function measured 5, 15, 20 and 40 minutes after the onset of exposure. Compared to baseline values, no statistically significant decreases in FVC, FEV<sub>1.0</sub>, MEF50% and MEF40% were seen with exposure to 2 ppm formaldehyde during both resting and exercise.

**Comment:** interpretation of the results by Paustenbach et al., (1997): eye irritation more sensitive parameter than nose and throat irritation.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th>Slight to severe:</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Eye irritation</td>
<td>Odour perception</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(0 ppm (R))</td>
<td>(0 ppm (E))</td>
</tr>
<tr>
<td>40 min</td>
<td>R = rest</td>
<td>15 laboratory workers exposed long-term to formaldehyde</td>
<td>0 ppm (R)</td>
<td>0 %</td>
</tr>
<tr>
<td></td>
<td>E = 10 min</td>
<td></td>
<td>0 ppm (E)</td>
<td>0 %</td>
</tr>
<tr>
<td></td>
<td>moderate exercise</td>
<td></td>
<td>2.0 ppm (R)</td>
<td>47 %</td>
</tr>
<tr>
<td></td>
<td>(conducted on a</td>
<td></td>
<td>2.0 ppm (E)</td>
<td>40 %</td>
</tr>
<tr>
<td></td>
<td>different day)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Pulmonary irritation:** pulmonary function measured 5, 15, 20 and 40 minutes after the onset of exposure. Compared to baseline values, no statistically significant decreases in FVC, FEV<sub>1.0</sub>, PEFR, MEF50% and MEF40% were seen with exposure to 2 ppm formaldehyde during both resting and exercise.

**Comment:** authors concluded that persons exposed long-term to formaldehyde had similar upper respiratory symptom frequency and severity as persons not previously exposed (see Schachter et al., 1986).
Table 11.1: Irritative effect of gaseous formaldehyde in humans (continued)

<table>
<thead>
<tr>
<th>Duration</th>
<th>Physical activity</th>
<th>Number of volunteers</th>
<th>Results</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>40 min</td>
<td>R = rest, E = 10 min moderate exercise</td>
<td>15 asthmatics</td>
<td>Eye irritation: 0 ppm (R) = 7 %, 0 ppm (E) = 14 %, 2.0 ppm (R) = 73 %, 2.0 ppm (E) = 36 %</td>
<td>Eye irritation: 0 ppm (R) = 7 %, 0 ppm (E) = 14 %, 2.0 ppm (R) = 73 %, 2.0 ppm (E) = 36 %</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Odour perception: 0 ppm (R) = 33 %, 0 ppm (E) = 57 %, 2.0 ppm (R) = 100 %, 2.0 ppm (E) = 100 %</td>
<td>Odour perception: 0 ppm (R) = 33 %, 0 ppm (E) = 57 %, 2.0 ppm (R) = 100 %, 2.0 ppm (E) = 100 %</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Nose irritation: 0 ppm (R) = 20 %, 0 ppm (E) = 14 %, 2.0 ppm (R) = 47 %, 2.0 ppm (E) = 36 %</td>
<td>Nose irritation: 0 ppm (R) = 20 %, 0 ppm (E) = 14 %, 2.0 ppm (R) = 47 %, 2.0 ppm (E) = 36 %</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Throat irritation: 0 ppm (R) = 27 %, 0 ppm (E) = 21 %, 2.0 ppm (R) = 33 %, 2.0 ppm (E) = 43 %</td>
<td>Throat irritation: 0 ppm (R) = 27 %, 0 ppm (E) = 21 %, 2.0 ppm (R) = 33 %, 2.0 ppm (E) = 43 %</td>
</tr>
<tr>
<td>1 hr</td>
<td>Healthy persons: intermittent strenuous activity Asthmatics: intermittent moderate exercise</td>
<td>22 healthy persons (H)</td>
<td>Symptoms scored moderate to severe: Eye irritation: 0 ppm = 0 %, 3.0 ppm (H) = 27 %<strong>, 3.0 ppm (A) = 19 %</strong> Odour Perception: 0 ppm = 0 %, 3.0 ppm (H) = 23 %<strong>, 3.0 ppm (A) = 31 %</strong> Nose/throat irritation: 0 ppm = 0 %, 3.0 ppm (H) = 32 %<strong>, 3.0 ppm (A) = 31 %</strong></td>
<td>Witek et al., 1987</td>
</tr>
<tr>
<td></td>
<td></td>
<td>16 asthmatics (A)</td>
<td></td>
<td>Green et al., 1987</td>
</tr>
</tbody>
</table>

**Pulmonary irritation:** Although some reductions were seen to FEV₁₀₀ and MEF50% over the exposure duration they occurred randomly with exposure to clean air and 2 ppm formaldehyde. No significant reduction was seen in FVC.

**Comment:** authors considered that the observed reductions in pulmonary function probably represented airway lability in asthmatics.

**Pulmonary irritation:** Pulmonary function measured prior to exposure and 17, 25, 47 and 55 minutes after the onset of exposure. In healthy volunteers, and compared to control exposures, a statistically significant decrease of 2 %* on FCV was seen after 47 minutes to 3ppm and of 3 %* on FVC, FEV₁₀₀ and FEV₃₀ after 55 minutes exposure. No statistically significant reduction was seen at other assessment times or on FEFR₂₅–₇₅ in healthy volunteers, or on FVC, FEV₁₀₀, FEV₃₀ and FEFR₂₅–₇₅ in asthmatics.

**Comment:** asthmatics were not more sensitive to the irritant effects of formaldehyde than non-asthmatics.
### Table 11.1: Irritative effect of gaseous formaldehyde in humans (continued)

<table>
<thead>
<tr>
<th>Duration</th>
<th>Physical activity</th>
<th>Number of volunteers</th>
<th>Results</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 hrs</td>
<td>Intermittent</td>
<td>24 healthy persons (non-smokers)</td>
<td><strong>Eye, nose and throat irritation:</strong> Compared with exposures to clean air, a statistically significant effect was seen at all time points on eye, nose and throat irritation with exposure to 3 ppm formaldehyde.</td>
<td>Green et al., 1989</td>
</tr>
<tr>
<td></td>
<td>physical exercise</td>
<td></td>
<td><strong>Pulmonary function:</strong> Pulmonary function measured prior to exposure and 20, 50, 80 and 110 minutes after the onset of exposure. Compared to control exposures, a statistically significant decrease (&lt; 10 %) in FEF&lt;sub&gt;25–75&lt;/sub&gt; was only reported with 50 and 80 minutes exposure to 3 ppm formaldehyde. No statistically significant reductions were seen on FVC, FEV&lt;sub&gt;1.0&lt;/sub&gt; or FEV&lt;sub&gt;3.0&lt;/sub&gt;.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td><strong>Comment:</strong> a significant formaldehyde effect on odour was also reported (no further details available).</td>
<td></td>
</tr>
<tr>
<td>3 hr</td>
<td>Intermittent</td>
<td>9 healthy persons (non-smokers)</td>
<td><strong>Individual scores for severity ranged from none to moderate:</strong></td>
<td>Sauder et al., 1986</td>
</tr>
<tr>
<td></td>
<td>physical exercise</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td><strong>Eye irritation</strong></td>
<td><strong>Odour Perception</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0 ppm</td>
<td>0.00</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3.0 ppm</td>
<td>0.78**</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td><strong>Pulmonary irritation:</strong> Pulmonary function measured prior to exposure and 15, 30, 60, 120 and 180 minutes after the onset of exposure. Compared to control exposures, a statistically significant decrease of 2 %* on FEV&lt;sub&gt;1.0&lt;/sub&gt; and 7 %** on FEF&lt;sub&gt;25–75&lt;/sub&gt; was seen only with 30 minutes exposure to 3.0 ppm formaldehyde. No statistically significant reduction was seen on FVC.</td>
<td>Sauder et al., 1987</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td><strong>Comment:</strong> individual responses to formaldehyde exposure ranged from −5% to +1% for FEV&lt;sub&gt;1.0&lt;/sub&gt; and −14% to +2% for FEF&lt;sub&gt;25–75&lt;/sub&gt;.</td>
<td></td>
</tr>
<tr>
<td>3 hr</td>
<td>Intermittent</td>
<td>9 asthmatics (non-smokers)</td>
<td><strong>Individual scores for severity ranged from none to severe:</strong></td>
<td>Sauder et al., 1986</td>
</tr>
<tr>
<td></td>
<td>physical exercise</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0 ppm</td>
<td>0.00</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3.0 ppm</td>
<td>1.33**</td>
</tr>
</tbody>
</table>
### Table 11.1: Irritative effect of gaseous formaldehyde in humans (continued)

<table>
<thead>
<tr>
<th>Duration</th>
<th>Physical activity</th>
<th>Number of volunteers</th>
<th>Results</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>20 min</td>
<td>None</td>
<td>13 persons (2 males and 11 females) with symptoms of asthma.</td>
<td><strong>Eye, nose and throat irritation</strong>: Self-reports of eye, nose and throat irritation occurred as frequently with clean air [symptoms were not reported for the different exposure levels (0.1, 1.0 and 3.0 ppm)]. <strong>Pulmonary function</strong>: Pulmonary function measured prior to exposure, immediately after and up to 24 hours after the onset of exposure. Compared with exposures to clean air, no significant decrease reported in FEV₁.₀ or FEV₂₅ - ₇₅ with exposure concentrations up to 3.0 ppm formaldehyde. <strong>Comment</strong>: Of the 13 subjects, 3 and 5 subjects were not challenged as they had unequivocal or convincing histories of asthma, respectively, 2 subjects were not challenged with methacholine because of time restraints, and 1 of remaining 3 gave a positive challenge to methacholine.</td>
<td>Reed &amp; Frigas, 1984</td>
</tr>
</tbody>
</table>

* Significantly different from control (p < 0.05)
** Significantly different from controls (p < 0.01)
*** Significantly different from control (p ≤ 0.005)
**** Significantly different from controls (p < 0.02)

# Complained of various non-respiratory adverse effects from the urea formaldehyde foam insulation (UFFI) in their homes.
FEFR₂₅ - ₇₅, Forced expiratory flowrate between 25% and 75% FVC
FEV₁₀, Forced expiratory volume in one second
FVC, Forced vital capacity
MEF50%, Maximum expiratory flow at 50% of vital capacity
PEFR, Peak expiratory flow rate
Ppm, Parts per million
Over a 2-year period in a Danish dermatology clinic, of 40 patients who gave a positive patch test to their own cosmetic products, 5 (12.5%) gave a positive result to formaldehyde (Held et al., 1999). In a Finish dermatology clinic, 82 of 1414 patients (5.8%) patch tested over a 6-year period with a modified European standard series gave a positive result to 1% formaldehyde solution (Kanerva et al., 1999). As part of a study on occupational skin diseases, 223 nurses were patch tested with a supplemented European standard series and prick tests conducted for common allergens (Kiec-Swierczynska, 2000). Prick tests indicated 80 (36%) nurses were atopic. A positive patch test to 1.0% formaldehyde solution was observed in 46 nurses (20.6%).

A case report is available of a 30-year old man who developed occupational allergic dermatitis working in a clothing warehouse (Cockayne et al., 2001). Formaldehyde resins, which were used in the textile industry, were suspected. Positive patch tests were reported with aqueous formaldehyde and formaldehyde resin.

Table 11.2: Case report of skin sensitisation by ODREC*

<table>
<thead>
<tr>
<th>Type of Formaldehyde Product Tested</th>
<th>No. of Positive Tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>Formalin</td>
<td>51</td>
</tr>
<tr>
<td>Formaldehyde releasing preservatives</td>
<td></td>
</tr>
<tr>
<td>DMDM Hydantoin</td>
<td>11</td>
</tr>
<tr>
<td>Imidazolidinyl urea (Germall 115)</td>
<td>23</td>
</tr>
<tr>
<td>Diazolidinyl urea (Germall II)</td>
<td>27</td>
</tr>
<tr>
<td>Dowicil 200 (Quarternium 15)</td>
<td>35</td>
</tr>
<tr>
<td>Formaldehyde resins</td>
<td></td>
</tr>
<tr>
<td>Melamine formaldehyde</td>
<td>5</td>
</tr>
<tr>
<td>Phenol formaldehyde resin (Novolac)</td>
<td>2</td>
</tr>
<tr>
<td>Phenol formaldehyde resin</td>
<td>6</td>
</tr>
<tr>
<td>Urea formaldehyde</td>
<td>3</td>
</tr>
<tr>
<td>4-tert butyl phenol formaldehyde resin</td>
<td>18</td>
</tr>
</tbody>
</table>

*All workers were tested for formalin, but not all were tested for formaldehyde resins. The names in bracket are trade names.

A number of human studies were conducted to induce (Marzulli & Maibach, 1974) and elicit skin sensitisation in sensitised individuals [Marzulli & Maibach, 1973 cited in the IPCS review (1989); Jordan et al., 1979; Hilton et al., 1998]. The CICAD (IPCS, 2002) concluded that the concentration of formaldehyde likely to elicit contact dermatitis reactions in hypersensitive individuals may be as
low as 30 mg/L (0.003%). ATSDR (1999) concluded that allergic skin responses in sensitised individuals exposure to concentrations below 0.25% to 0.05% formaldehyle in solution are rare.

There are no human data to suggest that exposure to formaldehyde gas causes skin sensitisation.

11.3.2 Respiratory

Bronchial Challenge tests

Data are available from studies that conducted bronchial challenge tests with gaseous formaldehyde on workers with asthmatic symptoms, to determine whether the observed asthma was attributable to this chemical. Single and/or double blind bronchial challenge tests conducted in 13 workers exposed to formaldehyde for up to 9 years (Frigas et al., 1984), and a single worker who had not been exposed to formaldehyde for 3 years (Grammer et al., 1993), were negative. In the Frigas et al. (1984) study, no reaction to bronchial challenge with formaldehyde was seen in a worker who had hyperresponsive airways (i.e. positive bronchial challenge to methacholine).

Positive bronchial challenges to formaldehyde have been observed in workers with asthmatic symptoms. Over a 6-year period, 12 of 230 patients referred to a clinic and had reportedly been exposed to formaldehyde gave positive bronchial challenge tests to formaldehyde (Nordman et al., 1985). Only one of these 12 tests was conducted in a blind manner. Furthermore, 9 of the 12 responders had hyperresponsive airways as shown by positive bronchial challenge tests to histamine or methacholine.

A positive bronchial challenge to formaldehyde was observed in a recent study in a single worker who had hyperresponsive airways (positive bronchial challenge to methacholine) and was exposed to several chemical agents whose exact components were unknown but did include formaldehyde (Kim et al., 2001). Similarly, though 7 of 15 workers (47%) gave positive responses to formaldehyde in open bronchial challenge tests (Burge et al., 1985), bronchial hyperresponsiveness was observed in 2 responders and 1 non-responsive subject. Additionally, co-exposure to other chemicals including isocyanates and hardwood dust had occurred in 12 workers, of which 3 had given a positive challenge to formaldehyde.

A study was conducted with three nurses, a technician and a visitor to a dialysis unit who were all regularly exposed to formaldehyde and had developed asthmatic symptoms (Hendrick & Lane, 1975, 1977). Positive bronchial challenges to formaldehyde were seen in 2 of the nurses, one of whom had pre-existing asthma. In a follow up study on these two nurses 2 years later, a positive bronchial challenge to formaldehyde was only observed in the nurse with pre-existing asthma (Henderick et al., 1982).

Open bronchial challenge tests to formaldehyde were conducted in 7 staff from an endoscopy unit and x-ray department who had asthmatic symptoms associated with glutaraldehyde exposure (Gannon et al., 1995). Positive responses were seen in 3 workers, which included the only 2 individuals with co-exposure to formaldehyde. This result suggests possible cross-reactivity between formaldehyde and glutaraldehyde.
Data are also available for healthy workers and volunteers. Negative bronchial challenge tests were observed in 15 healthy workers exposed to formaldehyde for between 1 to 21 years (Schachter et al., 1985). Bronchial challenges with formaldehyde in healthy volunteers were also negative (Sauder et al., 1986).

Additionally, negative bronchial challenges were seen in 9 people who complained of adverse health effects from the urea formaldehyde foam insulation used in their homes (Day et al., 1984), and in asthmatic subjects with hyperresponsive airways (Sheppard, 1984; Harving et al., 1990) and those without hyperresponsive airways (Witek et al., 1987).

**Clinical diagnosis data**

Studies focusing on the clinical diagnosis of asthma in patients, where no bronchial challenge test was performed to identify the agent responsible, are also available.

In studies determining the effect on lung function following workplace exposure to gaseous formaldehyde, no change in lung function was seen in a pathologist who suffered chest tightness (Kwong et al., 1983). Comparison of formaldehyde-exposed workers (with or without symptoms) with those not exposed revealed no changes in lung function in one study (Nunn et al., 1990), and a slight decrease over shift in another (Alexandersson et al., 1982). Decreased lung function was seen in a further study in (mostly) symptomatic workers compared to unexposed controls, though no changes in parameters were seen over a working day, week or weekend (Schoenberg & Mitchell, 1975).

**Epidemiology studies**

In a Swedish population-based case-control study of 20 000 subjects, 15 813 (aged 21 - 51 years) responded to a mailed questionnaire on occupational exposure, asthma, respiratory symptoms, smoking and atopy (Toren et al., 1999). A total of 362 subjects with physician diagnosed asthma or self-reported asthma-like symptoms were compared against a total of 2044 controls. Occupational exposure to gaseous formaldehyde (information on exposure levels not obtained) was not associated with an increased risk of asthma.

An Australian case-control study investigated the increased risk of asthma in children from exposure to gaseous formaldehyde in 80 households (Garrett et al., 1999). A total of 148 children aged 7 - 14 were investigated, of which 53 (36%) were diagnosed as asthmatic by a doctor. Information was obtained from parental interviews on parental allergy, parental asthma and presence of pets. Household formaldehyde levels were determined by passive sampling; mean of 12.6 ppb (15.1 $\times 10^{-6}$ m$^3$), with a maximum of 111 ppb (133 $\times 10^{-6}$ m$^3$). After adjustment for confounding factors, such as parental asthma, no association was seen between asthma and formaldehyde exposure. However, there was a weak, but not statistically significant, trend to more children with respiratory symptoms in higher formaldehyde exposure groups. In a further Australian case-control study (Rumchev et al., 2002), household formaldehyde levels were determined by passive sampling in the homes of 88 children aged 6 months to 3 years who were diagnosed at hospital with asthma, and compared with 104 community controls. Cases had a statistically significant higher mean formaldehyde exposure compared to controls, 32 ppb (38 $\times 10^{-6}$ m$^3$) and 20 ppb (24 $\times 10^{-6}$ m$^3$), respectively.
After adjustment for confounding factors, such as indoor air pollutants, relative humidity, indoor temperature, atopy, family history of asthma, age, sex socio-economic status, pets and environmental tobacco smoke, it was reported that children exposed to formaldehyde levels of 60 \( \mu \text{g/m}^3 \) have a 39% increase in odds of having asthma compared to children exposed to < 10 \( \mu \text{g/m}^3 \) (OR estimated to be approximately 1.4 95% CI 1.1-1.7 from data presented in a graph). However, considering the marginally increased risk observed, together with the number of potential sources of bias, such as selection bias and validity of diagnosis in the young, this study is not considered to provide sufficiently robust evidence of an association between formaldehyde exposure and increased risk of asthma in children.

**Immunology data**

Specific immunoglobulin E (IgE) antibodies to formaldehyde-human serum albumin conjugates have occasionally been detected in workers (Patterson et al., 1986; Kramps et al., 1989; Grammer et al., 1993; Wantke et al., 2000) and children exposed to formaldehyde from a school building (Wantke et al., 1996), though without any correlation with respiratory symptoms. Other studies have failed to detect the antibody (Nordman et al., 1985; Patterson et al., 1986; Thrasher et al., 1987; Kramps et al., 1989; Grammer et al., 1990; Kim et al., 1999; Baba et al., 2000). Similarly, specific IgG antibodies to the same conjugate have only occasionally been observed in exposed people (Grammer et al., 1990, 1993; Kim et al., 1999).

11.4 Non-neoplastic effects

11.4.1 Respiratory-related effects

The effect of gaseous formaldehyde on respiratory symptoms, pulmonary function and morphology of the nasal tract has been investigated in populations exposed in occupational and community environments.

**Occupational exposure**

Conflicting results have been observed in studies investigating the effect of occupational exposure to formaldehyde on pulmonary functions. In a number of studies of chemical, furniture and plywood workers, pre-shift reduction of up to 12% in lung function parameters (e.g., forced vital capacity, forced expiratory volume, forced expiratory flow rate) were reported for mean formaldehyde concentrations that were \( \leq 0.42 \) ppm (\( \leq 0.5 \) mg/m\(^3\)) (Alexandersson & Hedenstierna, 1988; 1989; Herbert et al., 1994; Holmstrom & Wilhelmsson, 1988) and, in one study at 1.13 ppm (1.3 mg/m\(^3\)) (Malaka & Kodama, 1990). Changes were generally small and transient over a work shift, with a cumulative effect over several years that was reversible after relatively short periods without exposure (e.g. 4 weeks); effects were more obvious in smokers than non-smokers (Alexandersson & Hedenstierna, 1989). In the only study where it was examined, a dose-response relationship between formaldehyde exposure and decreased lung function was observed in a group of 21 workers in wood product manufacturing exposed to mean formaldehyde concentrations of 0.35 – 0.42 ppm (0.42 – 0.50 mg/m\(^3\)) (Alexandersson & Hedenstierna, 1989). In contrast, no conclusive evidence of diminished lung function was observed in studies of larger numbers.
of workers (89 - 125) in resin manufacturing (Nunn et al., 1990), funeral service industries (Holness & Nethercott, 1989) and wood product manufacturing (Horvath et al., 1988), who were exposed to higher mean formaldehyde concentrations (up to > 2 ppm [> 2.4 mg/m³]).

These studies also examined symptoms of respiratory irritancy in workers. A higher prevalence of symptoms, such as nose, throat and eye irritation, cough and/or ‘wheeze’ was seen in workers exposed to formaldehyde compared to controls in the studies by Alexandersson & Hedenstierna (1988, 1989); Herbert et al. (1994); Holmstrom & Wilhelmsson (1988); Holness & Nethercott (1989); Malaka & Kodama (1990); Uba et al. (1989); and Wilhelmsson & Holmstrom (1992). However, these studies generally assessed a small numbers of workers (38 – 103) and it was not possible to meaningfully examine exposure response. A study by Horvath et al. (1988) did conduct such an analysis. In this study, a dose-response relationship was seen between formaldehyde concentration and prevalence of symptoms. Workers in this study (totaling 109) were exposed to 0.17 - 2.93 ppm (0.20 – 3.5 mg/m³) formaldehyde. In contrast, in a study by Nunn et al. (1990) there was no evidence to suggest that respiratory symptoms (such as wheeze) were more common in 125 workers exposed to concentrations up to and greater than 2.0 ppm (> 2.4 mg/m³) formaldehyde compared to controls.

Data are also available from studies that have investigated the histological changes within the nasal epithelium of workers occupationally exposed to gaseous formaldehyde.

In a case-control study of 15 workers in a plywood factory exposed to 0.08 – 0.6 ppm (0.1 - 0.7 mg/m³) formaldehyde through use of urea-formaldehyde glue, a statistically significant increase in the incidence of squamous metaplasia was seen in workers exposed to formaldehyde (Ballarin et al., 1992). However, there was also co-exposure to respirable wood dust whose contribution to these findings cannot be excluded. The most comprehensive study, and the only one with individual estimates of exposure based on area and personal sampling, investigated histological effects in 70 workers at a formaldehyde manufacturing plant and 36 controls (Holmstrom et al., 1989). A statistically significant increase in the mean histological score for morphological changes was seen in formaldehyde-exposed workers compared to controls; mean exposure 0.25 ppm (0.3 mg/m³) formaldehyde, with frequent short peaks of exposures above 0.8 ppm (0.96 mg/m³). This study also examined histopathological changes in the nasal epithelium in workers exposed to both 0.17 – 0.25 ppm (0.20 - 0.3 mg/m³) formaldehyde and wood dust, and found no significant changes when compared to controls. A further study of 75 workers exposed to 0.08 – 0.9 ppm (0.1 - 1.1 mg/m³) formaldehyde (with peaks of 4.2 ppm [5.0 mg/m³] or 0.5 – 0.9 ppm [0.6 - 1.1 mg/m³]) and wood dust observed statistically significant increases in mean histopathological scores for both exposure groups compared to controls (Edling et al., 1988). There was no significant variation between the two exposure groups themselves. The mean histopathological score was also approximately the same regardless of duration of exposure, although this may be attributable to the small numbers of the sub-groups (i.e. 23 - 28).

In contrast, a cross-sectional study of 80 workers in paper processing plants exposed to 0.02 - 2 ppm (0.024 - 2.4 mg/m³) gaseous formaldehyde through use of phenol-formaldehyde resins reported no association between “abnormal” cytology and formaldehyde exposure after controlling for age (Berke, 1987). In a
case-control study, no significant difference was seen in the incidence of histopathological findings in 37 workers at a formaldehyde manufacturing plant exposed to 0.5 - 2 ppm (0.6 - 2.4 mg/m³) formaldehyde, though the degree of metaplastic alteration was more pronounced among formaldehyde exposed workers (Boysen et al., 1990).

**Community exposure**

In a survey of 1726 occupants of homes containing urea-formaldehyde foam insulation (UFFI) and 720 residents in control homes with median formaldehyde levels of 38 ppb (maximum 227 ppb) and 31 ppb (maximum 172 ppb), respectively, no effects on lung parameters were observed (Broder et al., 1988). In contrast, levels of peak expiratory flow rates (PEFR) decreased linearly in 298 children (6 - 15 years old) exposed to 60 - 140 ppb formaldehyde in the home (Krzyzanowski et al., 1990). The decrease at 60 ppb was equivalent to 22% of the PEFR of non-exposed children while at 30 ppb it was 10%. In the same survey, a small transient decrement in PEFR was seen in adults (≥ 16 years old) only in the morning, and mainly in smokers.

The prevalence of self-reported symptoms, such as eye, nose and throat irritation was determined in these community studies. There were increases in prevalence of symptoms primarily at exposure > 120 ppb (> 0.14 mg/m³) in the study by Broder et al. (1988). However, in this study, health complaints of residents in UFFI homes significantly decreased after remediation (i.e. UFFI removal) although levels of formaldehyde were unchanged. No increase in self-reported symptoms was observed in the study by Krzyzanowski et al. (1990), though, in contrast, the prevalence in physician-reported chronic bronchitis or asthma increased in children (6 - 15 years old) exposed to 60 – 140 ppb formaldehyde, especially in those exposed to environmental tobacco smoke. A further study investigated the reported health complaints (eye irritation, nose/throat irritation, and headaches) in nearly 2000 residents in mobile and conventional homes (Ritchie & Lehan, 1987). A higher prevalence for all symptoms was reported at concentrations > 300 ppb (> 0.36 mg/m³) formaldehyde, with eye irritation the most frequently reported health effect; 89% of residents exposed to this concentration reported eye irritation. The proportion of the study group reporting eye irritation below 100 ppb (0.12 mg/m³) was low, at 1% of residents.

Additionally, in the study investigating community exposure by Broder et al. (1988), a small transient increase in the incidence of nasal epithelial squamous metaplasia was seen in UFFI-subjects intending to have their UFFI removed; 18% compared to 15% in controls.

**11.4.2 Neurological effects**

Evidence of neurological symptoms and impaired performance in neurobehavioural tests were seen in cross-sectional surveys of histology technicians exposed to gaseous formaldehyde in a series of studies by the same investigators (Kilburn et al., 1985b, 1987, 1989; Kilburn & Warshaw, 1992; Kilburn, 1994). However, co-exposure to solvents, such as xylene, toluene and chloroform, which are known to produce neurotoxic effects in humans, prevent any reliable conclusions being drawn from the data on the neurotoxic potential of formaldehyde.
11.5 Genotoxicity

Surveys are available that investigated genetic effects in peripheral lymphocytes, nasal and buccal mucosal cells of workers occupationally exposed to formaldehyde.

In studies assessing peripheral lymphocytes, no increased incidence in either chromosome aberrations, sister chromatid exchanges (SCE) or micronucleated cells (MN) were seen in 15 workers manufacturing or processing formaldehyde (Fleig et al., 1982), 30 medical students (Vasudeva & Anand, 1996), 23 anatomy students (Ying et al., 1997; 1999) and 6 pathology students (Thomson et al., 1984). Additionally, no increased incidence of DNA-protein cross-links was seen in 10 furniture workers (Zhitkovich et al., 1996).

An increased incidence in SCE in peripheral lymphocytes was seen in 90 pathology students (Shaham et al., 2002), 13 workers reported to be regularly exposed to formaldehyde (Shaham et al., 1997), 8 anatomy students (Yager et al., 1986) and 31 workers exposed to phenol-formaldehyde resins (Suskov & Sazonova, 1982). An increased incidence in chromosome aberrations, SCE and MN was seen in 13 anatomy students (He et al., 1998), while an increased incidence in MN, but not SCE, was observed in 29 mortuary students (Suruda et al., 1993). A study of 20 paper workers reported an increased incidence in chromosome aberrations but not SCE (Bauchinger & Schmid, 1985), however, this study has been criticised for the statistical analysis used, and the findings were considered incidental (Engelhardt et al., 1987). An increased incidence in chromosome aberrations was reported in a study in children (Dobias et al., 1988) and a study of workers (Kitaeva et al., 1996). However, only limited details were provided for these studies, which were reported in abstract form only. An increased incidence in DNA-protein-cross link was also seen in 12 workers, reported to be regularly exposed to formaldehyde (Shaham et al., 1997).

In studies investigating the incidence of MN in nasal and buccal cells, an increased incidence was seen in buccal but not nasal cells in studies of 29 and 28 mortuary students (Suruda et al., 1993; Titenko-Holland et al., 1996), while an increase was seen in both cell types in 25 anatomy students (Ying et al., 1997). An increased incidence in MN in nasal cells was also seen in 15 wood workers (Ballarin et al., 1992). An increased incidence in MN in buccal cells was reported in anatomy technicians and anatomy students, however only limited details are available for this Russian study, as only the abstract was reported in English (Kitaeva et al., 1996).

11.6 Carcinogenicity

The finding in the early 1980s of tumours in the nasal tract of rats exposed to formaldehyde in inhalation studies led to concerns for workers occupationally exposed to formaldehyde. Extensive epidemiological studies investigating respiratory tract cancers have since been conducted in workers. These studies, that include cohort mortality studies and case-control studies in industrial workers and professionals, have examined the incidence of cancers in the nasal tract, pharynx or lungs. An overview of three meta-analyses of these numerous epidemiology studies is presented below (Blair et al., 1990a, Partanen, 1993, and Collins et al., 1997). A more comprehensive summary of these studies can be found in Table 9 and 10 in the CICAD (IPCS, 2002) review, which is attached in

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Appendix 3. Additionally, recent case-control and cohort studies (post-1998), investigating the incidence of upper respiratory tract cancers in workers occupationally exposed to formaldehyde (Armstrong et al., 2000; Laforest et al., 2000; Vaughan et al., 2000; Hildesheim et al. 2001; Marsh et al., 2002; Berrino et al., 2003; Coggon et al., 2003; Elci et al., 2003; Hauptman et al., 2003; 2004; Pinkerton et al., 2004), and a meta-analysis of 12 case-control studies investigating the incidence of sinonasal cancers (Luce et al., 2002), are also presented in Section 11.6.1.

Possible associations between occupational exposure to formaldehyde and non-respiratory tract cancers have also been investigated to a lesser extent. In studies investigating increased risks of various non-respiratory cancers, such as melanoma, brain, connective tissue, pancreatic, and colon, increased risks have been occasionally observed but without any consistent pattern (e.g. Stroup et al., 1986; Stayner et al., 1988; Hayes et al., 1990; Holly et al., 1996; Dumas et al., 2000). However, recently data has been published (including updates of major cohort studies of industrial workers) that report a relationship between formaldehyde exposure and lymphohematopoietic cancers (specifically leukaemia). Since this cancer type was not specifically evaluated in the CICAD (IPCS, 2002), a review of all the available data is presented in Section 11.6.2. Additionally, a recently published case-control study and meta-analyses investigating the association between formaldehyde exposure and pancreatic cancer are also presented in Section 11.6.2.

11.6.1 Nasal tract, pharynx and pulmonary tumours Meta-analyses

Blair et al. (1990a) conducted a meta-analysis of 32\(^1\) studies covering occupational exposure to formaldehyde in industrial workers and professionals (embalmers, anatomy technicians and pathologists). The data were re-analysed by Partanen (1993) and included an additional three case-control studies\(^1\). Furthermore, in the meta-analysis by Partanen (1993) a number of changes in the selection of input values were made that were considered more appropriate, and relative risks determined using a different model from that of Blair et al. (1990a). Despite these changes the results of this re-analysis were generally in close agreement with the original meta-estimates by Blair et al. (1990a).

A significantly increased risk was found for nasopharyngeal cancers in workers with the highest category of exposure to formaldehyde in the meta-analyses conducted by both Blair et al. (1990a) and Partanen (1993) (meta-relative risk value (mRR) = 2.1, 95% CI 1.1 - 3.5 and mRR = 2.7, 95% CI 1.4 - 5.6, respectively). The two meta-analyses showed no increased risk between formaldehyde exposure and lung cancer among professionals. The mRR for lung

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\(^1\) Harrington and Oakes, 1984; Harrington and Shannon, 1975; Peterson and Milham, 1980; Jensen and Andersen, 1982; Fayerweather et al., 1983; Friedman and Ury, 1983; Marsh, 1983; Milham, 1983; Walrath and Fraumeni, 1983; Wong, 1983; Achesson et al., 1984a; 1984b; Coggon et al., 1984; Levine et al., 1984; Liebling et al., 1984; Malker and Weiner, 1984; Olsen et al., 1984; Walrath and Fraumeni, 1984; Partanen et al., 1985; Stayner et al., 1985; Walrath et al 1985; Bertazzi et al., 1986; 1989; Blair et al., 1986; 1987; 1989; 1990b; Bond et al., 1986; Gallagher et al., 1986; Hayes et al., 1986a; Logue et al., 1986; Stroup et al., 1986; Vaughan et al., 1986a; 1986b; Roush et al., 1987; Stayner et al., 1988; Gerin et al., 1989; Hayes et al, 1990.

\(^1\) Brinton et al., 1984; Gallagher et al., 1986; Merletti et al., 1991.
cancer for industrial workers was marginally, but significantly, increased for those with low/low-medium exposure to formaldehyde (both mRR = 1.2, 95% CI 1.1 - 1.3), but a significantly increased risk was not observed in both meta-analyses for those exposed to higher/substantial levels of formaldehyde. The observed marginally increased risk in the low dose group in the absence of a dose response does not demonstrate strong evidence of an association between formaldehyde exposure and lung cancer. For nasal cancers, Blair et al. (1990a) found no increased risk for formaldehyde exposure overall, while Partanen (1993) found a borderline significantly increased risk of sinonasal cancers in workers with substantial exposure to formaldehyde (mRR = 1.7, 95% CI 1.0 - 2.8).

In a more recent and comprehensive meta-analysis, Collins et al. (1997) initially considered 47 epidemiology studies. Several of these studies were not included in the analysis, because workers who had formaldehyde exposure were not evaluated separately or the study only reported relative risks, the study population was included in a more recent study, or the methodology and results were insufficiently described. In total the meta-analysis was based on the results from 11 cohort, 3 proportionate mortality and 18 case-control studies, and included new data published since Partanen (1993). Furthermore, the authors of studies were contacted to obtain data not included in their publications. The exposure potential of jobs that were classified as having formaldehyde exposure in the community-based case-control studies was also reviewed, as exposure assessment was much more uncertain in these studies than in cohort studies.

When all studies were included, no increased risk of lung cancer was seen with exposure to formaldehyde (mRR = 1.0, 95% CI 0.9 - 1.0). In cohort studies, a very small borderline, though significant, increased risk was seen for industrial workers (mRR = 1.1, 95% CI 1.0-1.2), while no increased risk was seen for pathologists (mRR = 0.5, 95% CI 0.4 - 0.6) or embalmers (mRR = 1.0, 95% CI 0.9 - 1.1). Similarly, no increased risk was seen in the case-control studies (mRR = 0.8, 95% CI 0.7 - 0.9).

No increased risk of sinonasal cancers was seen with exposure to formaldehyde (mRR = 1.0, 95% CI 1.0 - 1.1). Evaluating by study design revealed no increased risk for cohort studies (mRR = 0.3, 95% CI 0.1 - 0.9) but a significantly increased risk for case-control studies (mRR = 1.8, 95% CI 1.4 - 2.3). This increased risk was attributable to a significantly increased risk for the combined 6 European case-control studies (mRR = 2.9, 95% CI 2.2 – 4.0), whereas no increased risk was seen for the combined 5 US case-control studies (mRR = 1.0 95% CI 0.7 - 1.5). Collins et al. (1997) report that it is difficult to reconcile European findings with other findings unless it is assumed that confounding factors, or bias, were affecting the results.

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1 Harrington and Shannon, 1975*; Jensen and Andersen, 1982*; Fayerweather et al., 1983*; Hernberg et al., 1983a; 1983b; Walrath and Fraumeni, 1983*; Coggon et al., 1984*; Levine et al., 1984*; Walrath and Fraumeni, 1984*; Brinton et al., 1985; Bond et al., 1986*; Bertazzi et al., 1989*; Blair et al., 1986*; Hayes et al., 1986a*; Olsen et al., 1986; Stroup et al., 1986*; Vaughan et al., 1986a*; 1986b*; Roush et al., 1987*; Stayner et al., 1988*; Gerin et al., 1989*; Hayes et al., 1990*; Partanen et al., 1990; Hall et al., 1991; Matanoski, 1991; Chiazze et al., 1993; Gardner et al., 1993; Luce et al., 1993; West et al., 1993; Marsh et al., 1994; Andjelkovich et al., 1995 (*) included in the analysis by Blair et al., 1990a and Partanen, 1993. 

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A significantly increased risk of nasopharyngeal cancers was seen with exposure to formaldehyde (mRR = 1.3, 95% CI 1.2 - 1.5). However, evaluation of nasopharyngeal cancers was hampered in some industrial cohort studies, as expected numbers were not reported when there were no observed deaths. To overcome this, the expected number of deaths was estimated based on the ratio of expected lung cancers to nasopharyngeal cancers in the study by Blair et al. (1986) that reported nasopharyngeal deaths. Expected numbers were also not reported in the cohort studies of embalmers and medical specialists. Using a similar approach, based on the ratio of expected lung cancers to nasopharyngeal cancers in the study by Hayes et al. (1990), a non-significant increased risk was found for nasopharyngeal cancers and exposure to formaldehyde when all industrial cohort studies were combined (mRR = 1.2, 95% CI 0.4 - 2.5). While no increased risk of nasopharyngeal cancers was seen for all cohort studies combined (mRR = 1.0, 95% CI 0.4 – 2.5), a non-significant increased risk of such cancers was seen for all case-control studies combined (mRR = 1.3, 95% CI 0.9 - 2.1).

Collins et al. (1997) concluded that the data did not provide convincing evidence of a casual relationship between formaldehyde exposure and nasopharyngeal cancers. The authors attributed the differences in their results to the two earlier meta-analysis to be mainly due to the inclusion of a number of recently published negative cohort studies and the correction for non-reporting of expected deaths in some cohort studies.

A pooled analysis of 8 case-control studies by t’ Mannetje et al. (1999) are included in a more recent review by Luce et al. (2002) who conducted a pooled analysis of 12 case-control studies\(^1\) conducted in 7 countries. The review examined the associations between sinonasal cancers and occupational formaldehyde exposure. Studies were selected on availability of information on histological type of cancer, age, sex, smoking and occupational history. A total of 930 cases (680 men, 250 women), including 432 squamous cell carcinomas (330 men, 102 women) and 195 adenocarcinomas (169 men, 26 women), diagnosed between 1968 and 1990 were evaluated along with 3136 controls (2349 men, 787 women). The probability of exposure to a number of occupational substances (including formaldehyde) was determined using a job exposure matrix. The study focused on cumulative exposure although results of other exposure variables were presented when they gave additional information. After adjustment for age, a small non-significant increased risk was seen for squamous cell carcinomas in males and females with a high probability of exposure (odds ratio (OR) = 1.2, 95% CI 0.8 – 1.8 and OR = 1.5, 95% CI 0.6 – 3.8, respectively for a > 90% probability of exposure). After adjustment for age and cumulative exposure to wood and leather dust a significantly increased risk was seen between adenocarcinomas and medium (0.25 - 1 ppm) and high (> 1 ppm) intensity of exposure to formaldehyde in men (OR = 2.4, 95% CI 1.3 - 4.5 and OR = 3.0, 95% CI 1.5 – 5.7, respectively). Only age was adjusted for in women, with a significantly increased risk seen between adenocarcinomas and high probability of formaldehyde exposure (OR = 6.2, 95% CI 2.0 - 19.7).

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\(^{1}\) Cecchi et al., 1980, Luce et al., 1993 and Leclerc et al., 1994; Hardell et al., 1982; Brinton et al., 1984 and Brinton et al., 1985; Merler et al., 1986; Hayes et al., 1986a and Hayes et al., 1986b; Vaughan et al., 1986a, Vaughan, 1989 and Vaughan and Davis, 1991; Bolm-Audorff et al., 1990; Comba et al., 1992a; Comba et al., 1992b; Zheng et al., 1992; Magnani et al., 1993; Mack and Preston-Martin unpublished data, presented in Luce et al., 2002.
Luce et al. (2002) also evaluated cases of sinonasal adenocarcinoma where there was no exposure to wood or leather dust. A significantly increased risk was only seen for adenocarcinomas in females with a high probability of exposure (OR = 11.1, 95% CI 3.2 – 38.0, based on 5 cases). No significant increased risk was seen in males for low, medium or high probability of exposure. An analysis was also undertaken in men only of formaldehyde exposure by maximum exposure to wood dust. For no or low exposure to wood dust a non-significant increased risk was seen for adenocarcinomas with high and medium level exposure to formaldehyde (mRR = 2.2, 95% CI 0.8 – 6.3 based on 4 cases).

**Recent case-control studies**

In a study by Berrino et al. (2003), 315 males aged less than 55 years, diagnosed with laryngeal or hypopharyngeal cancer over a 3 – 5 year period in the late seventies to the early eighties in 6 centres in France, Italy, Spain and Switzerland were investigated. Most cases were interviewed, and information on occupational exposures, smoking and alcohol consumption, socio-economic status and diet obtained. Occupational exposures to substances, including formaldehyde, were determined using a job exposure matrix. Cases in each centre were matched by age and sex to a random sample of the general population (819 controls in total). After adjustment for potential confounding factors, such as smoking, alcohol consumption and other occupational exposures (including, wood dust and asbestos), a small increased risk, not statistically significant (OR = 1.3, 95% CI 0.8 – 2.0), was seen for exposure to formaldehyde. Analysis of duration of exposure (any probability) to formaldehyde showed no positive trend (although for 10 – 19 years exposure OR = 2.2, 95% CI 1.2 – 4.2 and OR = 1.3, 95% CI 0.6 – 2.8 for > 20 years exposure). Additionally, for analysis of the anatomical site of tumour origin, it was seen for endolarynx (n = 213) and hypolarynx (n = 100) cancers that though an increased risk was seen for those workers possibly exposed to formaldehyde (OR = 1.4, 95% CI 0.8 – 2.7 and OR = 1.3, 95% CI 0.6 – 2.6), no increased risk was seen for workers who were probably or certainly exposed to formaldehyde.

In a study by Elci et al. (2003), 940 males diagnosed with laryngeal cancer between 1979 and 1984 at a hospital in Istanbul, Turkey, were investigated. Cases were interviewed and information on occupational history, smoking and alcohol consumption obtained. Occupational exposures to substances, including formaldehyde, were determined using a job exposure matrix. Cases were matched with 1519 males who had other cancers thought not to share similar etiologic factors with laryngeal cancer. After adjustment for potential confounding factors, such as age, smoking and alcohol consumption, no increased risk was seen for formaldehyde exposure. For analysis of the anatomical site of tumour origin, a small non-significant increased risk was only seen for cancers originating in the glottic area (OR = 1.2, 95% CI 0.8 – 2.0). No exposure-response relationship was seen for either intensity or probability of exposure to formaldehyde and cancers originating in the glottic area (or for laryngeal cancers originating in the supraglottic or subglottic area).

Hildesheim et al. (2001) investigated occupational exposure to formaldehyde among 375 newly diagnosed cases of nasopharyngeal cancers in two tertiary care hospitals in Taiwan between July 1991 and December 1994. These cases were matched on sex, age and geographical residence to 325 population controls. Data were collected from cases and controls by interviews and questionnaires.
Occupational exposures were reviewed (blindly) by an industrial hygienist. A total of 74 cases with formaldehyde exposure were identified. After adjustment for a number of confounding factors, such as socio-demographic characteristics and cigarette smoking, a small non-statistically significant increased risk was seen for nasopharyngeal cancers and exposure to formaldehyde (OR = 1.4, 95% CI 0.93 – 2.2). Additionally, no statistically significant trend was seen for either duration or cumulative exposure to formaldehyde and nasopharyngeal cancers. Similarly, no dose response was observed for analysis of years since first exposure. Exposure to wood dust, with the exception of age at first exposure ≥ 25 years, resulted in greater increased risks than for exposure to formaldehyde, and the authors concluded that exposure to formaldehyde is less clearly linked to nasopharyngeal cancer than wood dust.

The study by Hildesheim et al. (2001) also tested blood samples from cases and controls for various anti-Epstein-Barr virus (EBV) antibodies which, the authors report, are associated with nasopharyngeal cancers. Among those seropositive to antibodies for EBV (360 cases, 94 controls), a significantly increased risk was seen for exposure to formaldehyde (OR = 2.7, 95% CI 1.2 - 6.2). However, as with the above analysis, no dose response was seen with increasing duration or cumulative exposure to formaldehyde.

In a study by Armstrong et al. (2000), 282 Chinese residents in Malaysia diagnosed with nasopharyngeal carcinomas between January 1987 and June 1992 were investigated. These residents were interviewed about their occupational history, diet, alcohol consumption and tobacco use, and each case matched by age and sex to a Malaysian Chinese control. Following adjustment for potential confounders, no increased risk was found for nasopharyngeal cancers and occupational exposure to formaldehyde. Additionally, no dose response was observed for duration of exposure to formaldehyde and nasopharyngeal carcinomas. However, only 51 of 564 cases reported occupational exposure to formaldehyde, and of these 51 cases only 8 had accumulated exposure ≥ 10 years.

Laforest et al. (2000) investigated occupational exposure to formaldehyde among 201 and 296 newly diagnosed cases of (primary) squamous cell hypopharyngeal and laryngeal cancers in men, respectively, reported in 15 French hospitals between January 1989 and April 1991. Information on demographic characteristics, alcohol and tobacco consumption, and lifetime occupational history were obtained through interviews. Occupational exposures were determined using a job exposure matrix. Controls were patients with (primary) cancers at different body sites, in the same or nearby hospitals during the same period and matched by age. After adjustment for potential confounding factors, such as smoking, alcohol consumption and other occupational exposures (including asbestos and man made mineral fibres), a statistically significant trend was seen for hypopharyngeal cancers and the probability of exposure to formaldehyde (P_{trend} <0.005, OR = 3.8, 95% CI 1.5 - 9.5 for the highest probability of exposure). No significant trend was noted for these cancers, however, in respect to duration or cumulative exposure to formaldehyde. When cases with a low probability of exposure to formaldehyde were excluded increased risks were observed for exposure to formaldehyde, with a statistically significant trend observed for duration of exposure (P <0.04) and for cumulative level of exposure (p <0.14). Neither the ORs nor any trend suggested an association between formaldehyde exposure and laryngeal cancer.
Vaughan et al. (2000) investigated occupational exposure to formaldehyde among 196 newly diagnosed cases of nasopharyngeal cancers reported in five US cancer registries between April 1987 and June 1993. These epithelial cancers were classified into 3 histological groups: 54 cases of undifferentiated and non-keratinising, 118 cases of differentiated squamous cell and 24 cases of unspecified epithelial. A total of 244 community controls were randomly selected and matched by age, gender and cancer registry. Data were collected for cases and controls by telephone interviews. Information on a number of confounding factors, such as history of occupational and chemical exposure, demographic background, medical history, family history of cancer, smoking and alcohol consumption, were collected. Estimates of potential exposure to formaldehyde were carried out on a job-by-job basis by experienced industrial hygienists who were blinded to the status of the subjects. After adjustment for potential confounding factors, no increased risk was seen between potential exposure to formaldehyde and undifferentiated and non-keratinising carcinomas. Excluding these histological cancer types, a statistically significant trend was seen between nasopharyngeal cancers and both exposure duration ($P_{\text{trend}} = 0.014$, OR = 2.7, 95% CI 1.2 - 6.0 for the top exposure duration of > 18 years) and cumulative exposure ($P_{\text{trend}} = 0.033$, OR = 3.0, 95% CI 1.3 - 6.6 for the greatest cumulative exposure of > 1.10 ppm years), for 25 and 24 cases, respectively, that were considered to have had a possible, probable or definitive exposure to formaldehyde. However, when cases with a low probability of exposure to formaldehyde were omitted the significance of the trend decreased for both duration ($P_{\text{trend}} = 0.069$) and cumulative exposure ($P_{\text{trend}} = 0.13$). While for definitive exposure to formaldehyde, although highly significant trends were reported for duration and cumulative exposure ($P_{\text{trend}} < 0.001$), this is based on only 10 available cases. These ORs for formaldehyde were essentially unaffected by adding exposure to wood dust to the models.

**Recent cohort studies**

**The NCI study (Hauptmann et al., 2004)**

The National Cancer Institute cohort of industrial workers in the USA was recently extended by 15 years and a mortality study of solid cancers undertaken (Hauptmann et al., 2004). Details of the study design and follow up can be found in Hauptmann et al., (2003) (see Section 11.6.2). Briefly, the cohort consisted of 25 619 workers and standardised mortality ratios (SMRs) were derived using the person-years method and compared with the expected numbers of deaths for the national population. Additionally, relative risks (RR), stratified by cumulative exposure, average exposure intensity, highest peak exposure, and duration of exposure, compared to workers in the low exposure category were calculated. Potential confounding was evaluated for duration of exposure to 11 other substances and for duration of work as a chemist or laboratory technician.

Mortality from all causes, all cancers, and all solid malignant neoplasms was significantly less than expected, regardless of exposure status. Compared to the national population a significantly increased risk was seen for nasopharyngeal cancers (SMR = 2.1, 95% CI 1.1 – 4.2). Additionally, the relative risk based on an internal comparison group for nasopharyngeal cancers increased with average exposure intensity, cumulative exposure, highest peak exposure, and duration of exposure to formaldehyde ($P_{\text{trend}} = 0.066, 0.025, 0.001$ and 0.147, respectively).
Among the 10 deaths for nasopharyngeal cancer, 2 were not exposed to formaldehyde and never exposed to particulates, whereas 7 were exposed to formaldehyde and particulates. This prevented an analysis of formaldehyde exposure separating those workers exposed, and not exposed, to particulates. A slight non-significant increased risk was seen for cancers of the nose and nasal cavity (SMR = 1.2, 95% CI 0.4 – 3.7). No increased risk was seen for the larynx or lung.

An original mortality study by Marsh et al. (1996), of the plant that reported the greatest excess risk of nasopharyngeal cancers in the US National Cancer Institute cohort reported above was recently extended by 14 years (Marsh et al., 2002). In this update of the plastic producing plant, the cohort consisted of 7328 men employed from 1 January 1945 to 31 December 1998 analysed for malignant cancers of the upper and lower respiratory tract. For this 1998 update, work histories and exposures were not updated beyond that of the previous assessment (up to 1995). Exposure estimates were determined from available sampling data, job descriptions and personal communications. The median average intensity of exposure to formaldehyde was 0.138 ppm, and the majority of workers had worked less than 1 year at the plant. SMRs were derived using the person-years method for several exposure measures and compared with the expected numbers of deaths for the national population and the local two counties area, adjusted for race, sex, age, calendar time, year of hire, duration of employment and time since first employment. Mortality from all cancers was close to the national and local rate. A statistically significant increased risk was seen for death from cancers of the buccal cavity and pharynx when compared with national (SMR = 1.8, 95% CI 1.2 – 2.6) and local rates (SMR = 1.53, 95% CI 1.03 - 2.15), and for pharyngeal cancer (total of 22 deaths) when compared with the national (SMR=2.6, 95% CI 1.7 – 4.0) and local rates (SMR = 2.2, 95% CI 1.4 – 3.4). An analysis of these pharyngeal cancers showed a statistically significant increased risk for the nasopharynx (SMR = 4.9, 95% CI 2.0 – 10.2 compared to national rates, and SMR = 5.0, 95% CI 2.0 – 10.3 compared to local rates), though this was based on only 7 such deaths.

Local rate based SMRs for pharyngeal and nasopharyngeal cancers were then determined according to selected work history and formaldehyde exposure measures. A statistically significant increased risk of pharyngeal and nasopharyngeal cancers was seen in workers employed during the 1947 – 1956 period (SMR = 3.2, 95% CI 1.9 – 5.1 and SMR = 8.1, 95% CI 3.0 – 17.7, respectively), but not the 1941 – 1946 or 1957+ period. Similarly, for time since first employment a statistically significant increased risk was seen for nasopharyngeal cancers and 20 – 29 years (SMR = 8.7, 95% CI 1.8 – 25.5) but not for greatest time since first employment (≥30 years). For pharyngeal cancers a statistically significant increased risk was seen for the greatest time since first exposure (SMR = 2.8, 95% CI 1.4 – 4.9). A statistically significant increased risk was seen for both pharyngeal and nasopharyngeal cancers for exposure durations of > 0 - < 1 year (SMR = 2.4, 95% CI 1.2 – 4.2 and SMR = 5.8, 95% CI 1.6 – 14.9, respectively) and > 10 years (SMR = 3.7, 95% CI 1.2 – 8.5 and SMR = 12.5, 95% CI 1.5 – 45.0, respectively) but not for 1 – 9 years. Furthermore, analysis of the median average intensity of exposure revealed a statistically significant increased risk for exposures of 0.03 – 0.159 ppm formaldehyde for pharyngeal (SMR = 3.8, 95% CI 1.5 – 7.9) and nasopharyngeal cancers (SMR = 15.3, 95% CI 4.2 – 39.1) but not for > 0 - <0.03 ppm and ≥0.16 ppm.
formaldehyde for either cancer. For cumulative exposure a statistically significant increased risk was seen for 0.004 – 0.219 (SMR = 5.9, 95% CI 1.2 – 17.2) and ≥0.22 ppm-years (SMR = 7.5, 95% CI 1.6 – 21.9) for nasopharyngeal cancers only.

Analysis of exposure to > 0.2 or > 0.7 ppm formaldehyde and duration of exposure was also undertaken. Although a statistically significant increased risk was seen for pharyngeal and nasopharyngeal cancers and duration of exposures of ≥10 years for > 0.2 ppm, no statistically significant increased risk was seen for the greatest duration of exposure with > 0.7 ppm formaldehyde, while a statistically significant increased risk was seen for unexposed workers and pharyngeal cancers (SMR = 2.1, 95% CI 1.2 – 3.5).

In this study (Marsh et al., 2002), a nested case-control study was conducted on the 22 reported pharyngeal cancer deaths. Each case was matched on race, sex, age and year of birth to four controls from the cohort. An attempt was also made to obtain information on smoking history and exposures outside of work through telephone calls or a knowledgeable informant (usually a surviving family member). When analysis was adjusted for smoking and year of hire no statistically significant increased risk of pharyngeal cancers was seen for duration of exposure, cumulative exposure, median average intensity of exposure and the time since first employment. Indeed, long-term workers (≥1 year) showed a reduced or nearly equal risk for pharyngeal cancers compared to short-term workers. As for the cohort study, workers hired during the 1947 – 1956 period were at greater risk. The authors concluded that the pattern of findings suggest that the observed nasopharyngeal cancers are not associated with formaldehyde exposure, and may reflect the influence of non-occupational risk factors or occupational risk factors associated with employment outside the plant.

The complete NCI cohort data were recently reanalysed by Marsh and Youk (2005). SMRs were derived for the US national and regional rates and internal cohort-based RR for four formaldehyde exposure metrics (highest peak, average intensity, cumulative and duration) using both the Hauptmann et al. (2003) categories and an alternative categorization based on tertiles of all nasopharyngeal deaths among exposed subjects. SMRs and RRs were determined for each of the 10 study plants and by two plant groups (Plant 1 vs Plants 2 – 10). As reported by Marsh et al. (2002) the majority (6 of 10) of the nasopharyngeal cancers were observed in plant 1 of the 10 plants forming the NCI cohort. Since Marsh et al. (2002) previously reported on nasopharyngeal cancers in plant 1 and the pattern observed for such is similar in this later evaluation, only a brief overview of the analysis by Marsh and Youk (2005) is presented below, which focuses on the findings in plants 2 – 10.

In contrast to the findings in plant 1, a deficit in nasopharyngeal deaths was seen among formaldehyde-exposed workers in plants 2 – 10 combined (regional rate based SMR = 0.65, 95% CI 0.08 – 2.33) and all non-baseline highest peak exposure categories were less than 1 with no evidence of an exposure-response relationship observed. Furthermore, none of the corresponding exposure-response relationships was statistically significant for plants 2 – 10 combined. The authors also found that reanalysis of the nasopharyngeal findings seen by Hauptmann et al. (2004) for the highest exposure category, was driven entirely by the excess risk in plant 1 at highest peak exposure. Overall, the authors concluded that the
nasopharyngeal findings in the NCI cohort were not associated with formaldehyde exposure.

**The NIOSH study (Pinkerton et al., 2004)**

The follow up of an existing cohort of garment workers exposed to formaldehyde (Stayner et al., 1988) was recently extended by 16 years in a retrospective cohort mortality study by Pinkerton et al. of the National Institute of Occupational Safety and Health (Pinkerton et al., 2004). The cohort consisted of 11,030 workers employed after 1955 at 3 garment facilities in the USA and followed through to December 1998. Subjects had been identified from employment records and their vital status was determined. Personal and static air monitoring data were available from 1981 in one plant and 1984 in the others, and showed mean 8 hour time-weighted average levels of formaldehyde exposure ranging from 0.09 to 0.2 ppm. The authors considered it likely that formaldehyde levels were substantially higher in earlier years. SMRs were derived using the person-years-at-risk method and compared with the expected numbers of deaths for both the national population and local population. The SMRs were stratified by duration of exposure, time since first exposure and year of first exposure.

Results were only presented using national rates though it is stated that results with local rates were similar. Mortality from all causes and from all cancers was significantly less than expected, and mortality for pharyngeal, laryngeal and trachea, bronchus and lung cancers were also less than expected. No cancers of the nasopharynx or nose were observed. In addition to analysis of underlying cause of death, this study also analysed all causes on the death certificate using multiple cause mortality methods. No cancers of the nasal cavities or nasopharynx were identified in the MCOD (multiple cause of death) analysis.

**The MRC study (Coggon et al., 2003)**

The follow up on an existing cohort of British chemical workers exposed to formaldehyde (Gardener et al., 1993) was recently extended by 11 years by Coggon et al. of the Medical Research Council’s Environmental Epidemiology Unit at the University of Southampton (Coggon et al., 2003). The cohort consisted of 14,014 men employed after 1937 at six British chemical factories and followed through to December 2000. Subjects had been identified from employment records, and their jobs had been classified for potential exposure to formaldehyde using a job-exposure matrix, as no measurements to formaldehyde had been taken before 1970. Subjects were placed into one of 5 determined exposure categories ranging from background levels to > 2 ppm formaldehyde. Subjects’ vital status were determined and SMRs derived using person-years method and compared with the expected numbers of deaths for the national population. It was observed that mortality among the cohort for all cancers was slightly, though significantly, higher (SMR = 1.10, 95% CI 1.04 – 1.16) and the increase was greater in men with high exposure (> 2ppm) to formaldehyde (SMR = 1.3, 95% CI 1.2 – 1.4). The increase in all cancers arose principally from an increase in cancers of the stomach and lung. SMRs were determined for these cancers for each formaldehyde exposure category. After adjustment for local variations in mortality, a statistically significant increase was only seen for lung cancer in men with high formaldehyde exposure (SMR = 1.3, 95% CI 1.1 – 1.4). The risk was highest in men exposed before 1965 when occupational hygiene was less developed and the highest exposures to formaldehyde would be expected to
have occurred (SMR = 1.3, 95% CI 1.1 – 1.5). However, a statistically non-significant inverse trend was seen for the number of years worked in high exposure jobs ($P_{\text{trend}} = 0.13$) and showed no trend to increase with time since first employed in such a job ($P_{\text{trend}} = 0.93$). According to the authors, the observation that mortality was highest in those who had worked in jobs with high levels of exposure for less than 1 year suggests confounding by non-occupational factors, such as smoking. In this study mortality from nasopharyngeal and sino-nasal cancers in the cohort were less than expected.

Summary

Many epidemiology studies have investigated formaldehyde exposure and cancer of the respiratory tract. The strongest evidence of an association has been observed for nasopharyngeal cancers. The most recent meta-analysis (Collins et al., 1997) concluded that although there was an increased, non-significant risk of nasopharyngeal cancers, overall, the data did not provide sufficient evidence to establish a causal relationship between nasopharyngeal cancers and formaldehyde exposure. Studies published since the meta-analysis provide mixed results for both case-control studies and cohort studies. Three large industrial cohort studies with a long follow-up have been recently published (Hauptman et al., 2004; Pinkerton et al., 2004; Coggon et al., 2003). The study by Hauptman et al. (2004) found that compared to the national population, there was a significantly increased risk of nasopharyngeal cancer. In addition, the relative risk increased with average exposure intensity, cumulative exposure, highest peak exposure and duration of exposure to formaldehyde. However, no such cancers were seen in the study by Pinkerton et al. (2004), while no increased risk was seen by Coggon et al. (2003). Similarly, mixed results have been observed in recent case-control studies of formaldehyde exposure and nasopharyngeal cancer.

It is noted that, as with all epidemiology studies, the epidemiological investigations for formaldehyde have study limitations, such as the absence of direct exposure measurements and the potential of confounding factors, such as co-exposure to other chemicals and/or wood dust. However, the numerous findings of increased risk of nasopharyngeal cancers cannot be entirely attributed to such potential limitations in study design. Therefore, although it cannot be definitely concluded that occupational formaldehyde exposure results in the development of nasopharyngeal cancer, there is some evidence to suggest a causal association between formaldehyde exposure and nasopharyngeal cancer. Follow-up of the National Cancer Institute cohort continues and the findings should assist in further elucidating the strength of the association between formaldehyde and nasopharyngeal cancer.

There are several case-control studies that indicate an increased risk for sinonasal cancer and formaldehyde exposure, but this has not been observed in cohort studies. The most recent meta-analysis (Collins et al., 1997) concluded that the data did not support an association between formaldehyde and sinonasal cancer. There is limited and inconsistent evidence with respect to laryngeal and lung cancers. Overall, the available data do not support an association between sinonasal, laryngeal and lung cancers and formaldehyde exposure.
11.6.2 Non-respiratory tract cancers

Lymphohematopoietic cancers Meta-analysis

Collins and Lineker (2004) conducted a meta-analysis of 18 epidemiological studies\(^1\) (12 cohort mortality studies, 4 proportionate mortality and 2 case-control studies) published between 1975 – 2004, that reported leukaemia and occupational exposure to formaldehyde. Criteria were applied in the selection of studies and, consequently, not all studies reporting leukaemia in formaldehyde-exposed workers published between the dates stated were included in this analysis. For all 18 studies analysed a very slight increased risk for leukaemia was observed (mRR = 1.1, 95% CI 1.0 – 1.2) in the absence of heterogeneity across studies (p = 0.07). When analysed by occupation, increased risks were seen for embalmers (mRR = 1.6, 95% CI 1.2 – 6.0) and pathologists/anatomists (mRR = 1.4, 95% CI 1.0 – 1.9) with consistency seen across studies (p = 0.97 and p = 0.96, respectively). No increased risk was seen for industrial workers, whom the authors report may have had higher average daily exposures and peak exposures than embalmers, pathologists and anatomists. The authors concluded that this meta-analysis does not provide reliable evidence of an association between formaldehyde exposure and leukaemia, due to the absence of consistent findings across study types and inconsistent findings of small increased leukaemia rates across job types (that suggest the possibility of confounding factors).

In a previous meta-analysis conducted by Blair et al. (1990a) of 32 case-control and cohort studies\(^2\) a statistically significant increase in mortality from leukaemia was reported in professionals: embalmers, anatomy technicians and pathologists (mRR = 1.6, confidence intervals not reported). A slight and non-statistically increased risk was seen among industrial workers (mRR = 1.1, confidence intervals not reported). No increased risk was observed for Hodgkin’s lymphoma among professional or industrial workers.

Case-control studies

A population-based case-control study was conducted in Iowa and Minnesota (United States) to evaluate associations between occupational exposures (including formaldehyde) and leukaemia in 513 cases identified from the cancer registry of Iowa between March 1981 and October 1983, and from Minnesota hospitals between October 1980 and September 1982 (Blair et al., 2001). Cases (confirmed by pathology diagnosis) were matched to 1087 controls, for age, vital status and geographical residence. Data were collected through interviews, with surrogates where necessary. In addition to occupational history, information was also collected on residential history, drinking water sources, smoking, alcohol use, medical history, family history of cancer, education and other demographic

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\(^1\) Harrington and Shannon, 1975; Linos et al., 1980; Walrath and Fraumeni, 1983; Harrington and Oakes, 1984; Levine et al., 1984; Walrath and Fraumeni, 1984; Stroup et al., 1986; Edling et al., 1987; Ott et al., 1989; Hayes et al., 1990; Hall et al., 1991; Matanoski et al., 1991; Dell and Teta, 1995; Andjelkovich et al., 1995; Hansen and Olsen, 1995; Coggon et al., 2003; Hauptmann et al., 2003; Pinkerton et al., 2003.

\(^2\) A listing of the studies included in this meta-analysis can be found in the foot note in Section 11.6.1.
variables. Exposures were determined using a job exposure matrix and probability and intensity of exposure determined. ORs were adjusted for use of pesticides, postsecondary education, use of hair dyes, first degree relative with a haematolymphopoietic cancer and smoking, and determined by histologic type of leukaemia: acute myeloid; acute lymphocytic; chronic myeloid; chronic lymphocytic; and myelodysplasia. For formaldehyde exposure there were no cases of acute lymphocytic leukaemia, while no increased risks were seen for acute myeloid leukaemia and myelodysplasia. Small increased risks, not significant, were only seen for chronic myeloid leukaemia (OR = 1.3, 95% CI 0.6 – 3.1) and chronic lymphocytic leukaemia (OR = 1.2, 95% CI 0.7 – 1.8) to low/medium exposure of formaldehyde. Results for high exposure are not presented here as they are of limited value being based on only one case for each cancer type.

Nisse et al. (2001) investigated the association between occupational (including formaldehyde) and environmental factors, and myelodysplastic syndromes diagnosed among 204 patients from September 1991 to February 1996 in Lille, France. These cases were matched on sex, age and geographical residence to 204 population controls. Data were collected by interviews and questionnaires. The OR for formaldehyde exposure was not reported, suggesting that there was no increased risk and/or the number of cases with exposure to formaldehyde was so few to allow a meaningful analysis of the data.

Tatham et al. (1997) investigated the relationship between occupational exposures (including formaldehyde) and three subgroups of non-Hodgkin’s lymphoma (small cell diffuse, follicular and large cell diffuse) in 1048 men diagnosed with such cancers between December 1984 and November 1988. Cases (confirmed by pathology diagnosis) were identified from cancer registries in Atlanta, Connecticut, Iowa, Kansas, Miami, San Francisco, Detroit and Seattle (United States) and matched to 1659 controls for age and geographical residence. Data were collected for cases and controls by telephone interviews on background characteristics, medical, work and military history, and lifestyle. Consequently, exposure was self-reported. ORs were adjusted for the following potential confounding factors: age at diagnosis/case selection, education, ethnicity, year entered study, Jewish religion, having never married, AIDS risk behaviours, use of seizure medication, service in Vietnam (i.e. potential exposure to Agent Orange), and smoking. A small non-significant increased risk was seen for all cases of non-Hodgkin’s lymphoma (OR = 1.2, 95% CI 0.9 – 1.5). Similar results were seen for small and large cell diffuse lymphoma, while no increased risk was seen for follicular lymphoma.

West et al. (1995) investigated the association between ‘newly’ diagnosed cases of myelodysplastic syndromes in 400 patients from South Wales, Wessex and West Yorkshire (UK) and exposures through occupation, environment and hobby. Controls (number not reported) were selected from outpatient clinics and inpatient wards of medicine, ear nose and throat, orthopaedics and geriatrics, and matched to cases for age, geographical residence, hospital and year of diagnosis. Data on lifetime exposures through occupation, environment or hobby were collected by questionnaire, structured and semi-structured interview. ORs were determined for duration of exposure and for formaldehyde and were 1.2, 2.3 and 2.0 for ≥10 hours lifetime exposure of low intensity (14 cases), ≥50 hours lifetime exposure of medium or high intensity (7 cases) and ≥2500 hours lifetime exposure.
exposure of medium or high intensity (4 cases), respectively. Confidence intervals were not reported, though it is stated that these ORs were not statistically significant.

Partanen et al. (1993) investigated occupational exposure among 7307 male production workers employed in the wood industry in Finland between 1945 and 1963 and traced through the Finnish cancer registry. From this cohort 4 cases of Hodgkin’s disease, 8 cases of non-Hodgkin’s lymphoma and 12 cases of leukaemia diagnosed between 1957 and 1982 were matched by age and vital status to 152 controls from the same cohort free of cancer in 1983. Exposures were determined using a job exposure matrix. Cases were interviewed or questionnaires sent to their next of kin. A non-statistical increased risk was seen for leukaemias and lymphomas combined and exposure to formaldehyde (OR = 2.5, 95% CI 0.8 – 7.6). Only 3 of the 7 cases were not co-exposed to wood dust and, consequently, a meaningful analysis of exposure to formaldehyde alone could not be undertaken. Adjusting the analysis for exposure to wood dust (or solvents) did not substantially alter the results. For analysis of cancer type, increased risks were seen for leukaemia (OR = 1.4, 95% CI 0.3 – 7.9) and non-Hodgkin’s lymphoma (OR = 4.2, 95% CI 0.7 – 26.6), however, this analysis was based on a small number of cancers (2 and 4, respectively), which limited the statistical power of these analyses.

A population-based case-control study of leukaemia (n = 578) and non-Hodgkin’s lymphoma (n = 622) in white males in Iowa and Minnesota (United States) was briefly reported in the ‘letters section’ of a published journal (Linos et al., 1990). A non-significant increased risk was seen for total non-Hodgkin’s lymphoma (OR = 3.2, 95% CI 0.8 – 13.4) and total leukaemia (OR = 2.1, 95% CI 0.4 – 10.0) among embalmers and funeral directors following adjustment for age and state. A significantly increased risk was seen specifically for follicular non-Hodgkin’s lymphoma (OR = 6.7, 95% CI 1.2 – 37.1) and acute myeloid leukaemia (OR = 6.7, 95% CI 1.2 – 36.2) in these professions. Limited methodological details were presented and the estimates were based on only 3 exposed cases for each cancer type, so statistical power was limited.

A case-control study was conducted in Montreal Canada to investigate possible associations between occupational exposures (including formaldehyde) and cases of cancer diagnosed from September 1979 to December 1985 (Gerin et al., 1989). A total of 53 cases of Hodgkin’s lymphoma and 206 cases of non-Hodgkin’s lymphoma were compared with 2599 controls diagnosed with cancers of other organs and 533 population controls from the Montreal area. Data were obtained through interviews or questionnaires and used to determine potential occupational exposures. ORs were adjusted for the following potential confounding factors: age, ethnicity, socio-economic status, smoking, ‘dirtiness’ of the job (to distinguish white collar work histories from blue-collar ones), and other potential occupational and non-occupational confounders. No increased risk was seen for non-Hodgkin’s lymphomas and exposure to formaldehyde for less than, and over, 10 years exposure at estimated medium or high levels of exposure. Similarly, no increased risk was seen between formaldehyde exposure and Hodgkin’s lymphoma. Analysis of exposure subgroups was not conducted for this cancer, as there were only 8 exposed cases.

The case-control group described above by Gerin et al. (1989) was also evaluated by Fritschi and Siemiatycki (1996) for possible associations between
occupational exposures (including formaldehyde) and cases of Hodgkin’s lymphoma, non-Hodgkin’s lymphoma (for which there was a small increase in cases with $n = 54$ and $n = 215$, respectively) and myeloma. As for the previous analysis, this study provides no evidence of an association between formaldehyde exposure and non-Hodgkin’s lymphoma. Results for Hodgkin’s lymphoma and myeloma were not presented due to either a lack of prior evidence of an association or fewer than 4 exposed cases.

**Cohort studies**

A number of cohort studies are also available. Several of these cohorts have recently been updated and only the most recent updates are presented below.

The follow up of an existing cohort of garment workers exposed to formaldehyde (Stayner et al., 1988) was recently extended by 16 years in a retrospective cohort mortality study (Pinkerton et al., 2004). Details of the study design can be found in Section 11.6.1. Briefly, the cohort consisted of 11 030 workers employed after 1955 at 3 garment facilities in the USA and followed through to December 1998. Subject’s vital status was determined and SMRs derived and compared with the expected numbers of deaths for both the national population and local population. The SMRs were stratified by duration of exposure, time since first exposure and year of first exposure.

Results were only provided using national rates, though it is reported that results with local rates were similar. Mortality from all causes and from all cancers was significantly lower than expected, and mortality for all lymphatic and haematopoietic cancers was slightly lower than expected. Additional analysis for more detailed subgroups (i.e. mortality since 1960) for leukaemia showed a very small non-significant increased risk (SMR = 1.1, 95% CI 0.7 – 1.6) that was due to a non-significant increased risk for myeloid leukaemia (SMR = 1.4, 95% CI 0.8 – 2.4). After results were stratified by duration of exposure and time since first exposure an increased risk was seen for myeloid leukaemia (SMR = 2.4, 95% CI 1.0 to 5.0) among workers with both 10 or more years of exposure and 20 years or more since first exposure. In addition to analysis of underlying cause of death, this study also analysed all causes on the death certificate using multiple cause mortality methods (MCOD). After results were stratified by duration of exposure and time since first exposure, a significantly increased excess was seen for leukaemia deaths, specifically myeloid leukaemia (SMR = 2.55, 95% CI 1.10 – 5.03, for workers with both 10 or more years of exposure and 20 years since first exposure).

The follow up on an existing cohort of British chemical workers exposed to formaldehyde (Gardener et al., 1993) was recently extended by 11 years (Coggon et al., 2003). Details of the study design and follow up can be found in Section 11.6.1. Briefly the cohort consisted of 14 014 men employed after 1937 at six British chemical factories and followed through to December 2000. Subjects’ vital status were determined and SMRs derived and compared with the expected numbers of deaths for the national population. It was observed that the mortality among the cohort for all cancers was very slightly, though significantly, higher (SMR=1.10, 95% CI 1.04 – 1.16). Mortality from leukaemia and other lymphatic and haematopoietic cancers was generally lower than expected for the full cohort and in men with high exposures to formaldehyde.

*Formaldehyde*
The National Cancer Institute cohort of industrial workers in the USA was recently updated, 15 years from the original study by Blair et al. (1986), to evaluate the association between formaldehyde exposure and lymphohaematopoietic cancers (Hauptmann et al., 2003). The cohort consisted of 25 619 workers employed before January 1966 at 10 industrial plants and followed through to December 1994. Exposure to formaldehyde was estimated from work histories collected through to 1980 based on a job-exposure matrix and some monitoring data. No information on formaldehyde exposure was collected after 1980. SMRs were derived using the person-years method and the expected numbers of deaths were derived from the national population. Relative risks (RR), stratified by cumulative exposure, average exposure intensity, highest peak exposure, and duration of exposure, and compared to workers in the low exposure category, were also determined. The low exposure categories were 0.1-1.9 ppm for peak exposure, 0.1-0.4 ppm for average exposure intensity, 0.1-0.4 ppm-year for cumulative exposure and 0.1-4.9 years for duration of exposure. It was assumed that the exposure rate for all jobs, and over time, was constant. Peak exposure was estimated from knowledge of the job tasks and a comparison with 8-hour time-weighted averages. Potential confounding was evaluated for duration of exposure to 11 other substances (including benzene) and for duration of work as a chemist or laboratory technician.

Mortality from all causes, all cancers, and all solid malignant neoplasms was significantly less than expected, regardless of exposure status. Similar results were found for lymphatic and haematopoietic cancers in general and for specific cancer types including non-Hodgkin’s lymphoma, multiple myeloma and leukaemia. For Hodgkin’s disease, there was a slight increase, not statistically significant (SMR 1.3, 95%CI 0.8 to 2.0), amongst exposed workers. However, a statistically significant increased risk was seen for lymphohaematopoietic cancers with peak exposure of 2-3.9 ppm (RR = 1.7, 95% CI 1.1 – 2.6) and ≥ 4.0 ppm (RR = 1.9 95% CI 1.3 – 2.8), and for an average exposure intensity of 0.5 – 0.9 ppm (RR = 1.6, 95% CI 1.1 – 2.4) and ≥ 1.0 ppm (RR = 1.5, 95% CI 1.01 – 2.2). A statistically significant exposure response relationship was seen between peak exposure to formaldehyde and all lymphohaematopoietic cancers (P_{trend} = 0.002). This was primarily due to an exposure response relationship for myeloid leukaemia (P_{trend} = 0.009, with a RR = 3.5, 95% CI 1.3 - 9.4 for the highest peak exposure category of ≥ 4 ppm). For average exposure intensity and myeloid leukaemia a statistically significant increased risk was seen for the highest exposure category of > 1 ppm (RR = 2.5, 95% CI 1.03 - 6.0), although the exposure response relationship was only of borderline significance (P_{trend} = 0.088). For both duration and cumulative exposure only slightly increased risks, not statistically significant, were seen for lymphohematopoietic cancers and myeloid leukaemia specifically. The exposure response relationship for these endpoints was not statistically significant. For Hodgkin’s lymphoma, a statistically significant increased risk was seen in workers with average exposure intensity of 0.5-0.9 ppm (RR 4.7, 95% CI 1.6 - 13.8) but not ≥ 1 ppm. Additionally, a statistically significant exposure response relationship was seen for both peak and cumulative exposure and Hodgkin’s disease (P_{trend} = 0.042 and P_{trend} = 0.045, respectively). Generally, slight non-significant increased risks were seen for multiple myeloma and lymphatic leukaemia for all the analyses undertaken.
In summary, Hauptman et al. (2003) found a significant trend and association for myeloid leukaemia with both peak and average exposure intensity to formaldehyde, a weak association with duration of exposure, and no association with cumulative exposure.

The NCI cohort was recently reanalysed by Marsh and Youk (2004). SMRs were derived for the US national and regional rates and internal cohort-based RR for formaldehyde exposure metrics (highest peak, average intensity, cumulative and duration) using both the Hauptmann et al. (2003) categories and an alternative categorization based on tertiles of deaths from all leukaemia among exposed subjects. Additionally, for highest peak exposure, RRs were determined by the duration of time worked in the highest peak category and the time since highest exposure, while for average intensity of exposure RRs were determined by the duration of exposure and the time since first exposure. Similar to Hauptmann et al. (2003), no association was seen for cumulative and duration of formaldehyde exposure. However, the comparison using external groups revealed that the elevated leukaemia and myeloid leukaemia RRs and associated trends reported by Hauptmann et al. (2003) for highest peak exposure and average exposure intensity occurred because null (or slight) to moderate mortality excesses were compared with statistically significant baseline category deficits in death. Furthermore, the alternative analysis of duration of time worked in the highest peak exposure category did not indicate an association or higher increased risk among those workers who had experienced high peaks for a longer time. Similarly, no consistent evidence was seen that leukaemia or myeloid leukaemia risks increased for average exposure intensity and duration of exposure in a given average exposure intensity category, time from the first exposure, highest peak exposure, and for combined average exposure intensity and first exposure.

Marsh et al. (1996) studied 1 of the 10 industrial plants included in the National Cancer Institute cohort. However, since this study is included in the Hauptmann et al. (2003) studies and the results for ‘all lymphopoietic tissues’ are briefly reported, a detailed summary of this study is not provided.

A recent analysis of the above 3 recent cohorts (Pinkerton et al., 2004, Coggon et al., 2003, and Hauptman et al., 2003) was undertaken to evaluate the evidence for causality (Cole and Axten, 2004), based on epidemiologic criteria modified and updated by Cole (1997) from the criteria advanced in 1965 by Hill (Hill, 1965).

Cole and Axten (2004) point out that the recent analyses of leukaemia findings in the NCI cohort by Hauptman (2003) that address dose-response relationships are not based on SMRs and the attendant comparison with general population rates, but internal comparisons expressed as RRs. Cole and Axten (2004) state that it is unlikely that there is any excess of myeloid leukaemias among NCI exposed workers, as the SMR for all leukaemia is < 1.00 based on 65 deaths of which 43% are myeloid leukaemias, while in the US, among white males 20 years of age and over, the corresponding percentage based on deaths in 1979 - 1981 is 46%. Using the NCI observed number of 43% for myeloid leukaemias and the same approach, Cole and Axten (2004) estimated that, from the deaths for all leukaemia, the maximum likely SMR for myeloid leukaemias among the high exposure group in the study by Coggon et al. (2003) would be < 1.00.

Cole and Axten (2004) applied four criteria for determining causation. They report that the first criteria ‘replicability’ was not met, as the study by Coggon et
al. (2003), which probably involved the highest exposure, is negative. Also the study reported by Pinkerton et al. (2004) ‘is less positive’ than the NCI cohort, which was not highly consistent within itself. The second criteria ‘strength’ of association was not met, as the SMR as a whole for the collective body of data is < 1.00 for leukaemia. Even if the Coggan et al. (2003) study is ignored, the SMR for myeloid leukaemia for the other two studies combined was estimated to be < 1.00 by the authors (data not presented). The third criteria ‘coherence’ was not met as the available data indicates that inhaled formaldehyde is rapidly metabolised, does not reach the bone marrow and is, therefore, unlikely to induce leukaemia. The fourth criteria ‘response to manipulation’ was not met for the NCI cohort, as the long-term trend in reduction of formaldehyde exposure in the plants has not been followed by a reduction in the previously observed risk of leukaemia or myeloid leukaemia (i.e. only the recent report and not earlier ones suggest a myeloid leukaemia excess). Therefore, the formaldehyde-leukaemia hypothesis failed each of the four criteria of general causation applied by the authors, who concluded that the increased incidence of leukaemia reported in these three large cohort studies was not plausible.

Mortality was investigated in workers who were exposed to wood and enrolled in the American Cancer Society’s Cancer Prevention Study-II in 1982 (Stellman et al., 1998). The cohort was followed up for 6 years and consisted of 363 823 men. Information on exposure to formaldehyde was obtained through self-reporting. Incidence density ratios were used to determine RR which were adjusted for age and smoking. The comparison group was men exposed to formaldehyde but not employed in a wood-related job and who reported no exposure to wood dust. An increased risk was seen for woodworkers exposed to formaldehyde for all lymphatic and haematopoietic cancers (RR = 3.4, 95% CI 1.1 – 10.7) and specifically leukaemia (RR 5.8, 95% CI 1.4 – 23.3). In contrast, in men not employed in a wood-related job but exposed to formaldehyde, a non-significant increased risk was seen for all lymphatic and haematopoietic cancers (RR = 1.2, 95% CI 0.8 – 1.8), with no increased risk seen specifically for leukaemia or non-Hodgkin’s lymphoma.

A standardised proportionate cancer incidence study was undertaken of workers in Denmark born between 1897 and 1964 whose cancer was diagnosed between 1970 and 1984 (Hansen & Olsen, 1995). The cohort consisted of 91 182 men identified from the Danish cancer registry and for whom work histories were obtained using the Supplementary Pension Fund. The Danish Product Register was used to determine potential formaldehyde exposure. Standardised proportionate incidence ratios were determined for specific cancers and adjusted for age and calendar time. For non-Hodgkin’s lymphoma, Hodgkin’s lymphoma and leukaemia the observed number of cases was either close to, or less than, expected.

A mortality study of workers exposed to formaldehyde at an iron foundry in the US was undertaken (Andjelkovich et al., 1995). The cohort consisted of 3929 men employed during the period from January 1960 through to May 1987. SMRs were derived using the person-years-at-risk method and the mortality of this group was compared with the US population and 2032 workers at the foundry with no exposure to formaldehyde during the same time period. After reviewing work histories exposures were determined to be 0, 0.05, 0.55 or 1.5 ppm formaldehyde. Mortality from all cancers was close to the national rate for both
the exposed and unexposed population. For the exposed population, mortality from each of lymphosarcoma and reticulosarcoma, Hodgkin’s lymphoma and leukaemia was less than expected.

A mortality study of workers at a formaldehyde resin plant in Italy was undertaken (Bertazzi et al., 1986; 1989). The cohort consisted of 1332 men employed at the plant for at least 30 days between 1959 and 1980 and followed up for a further 6 years (up to 1986) in the second study. The only exposure data available for formaldehyde were airborne measurements taken between 1974 and 1979. Mean levels were 0.2 to 3.8 mg/m³ formaldehyde with maximum values up to 9.8 mg/m³ reported. Work histories were reconstructed for past employees. SMRs were derived using person-years-at-risk method, and the mortality of this group compared with the local and national population, and adjusted for gender, age and calendar time. Mortality for all cancers was slightly higher compared to local rates and significantly higher compared to the national rate (SMR = 1.5, 95% CI 1.1 – 2.1). A non-significant increased risk was seen for haematologic cancers (SMR = 1.7, confidence intervals not reported) when compared with the national rate, which was reported to become ‘very modest’ when compared with the local rate. Additionally, it was reported that analysis by latency and duration of employment failed to suggest an association.

A nested case-control study of non-Hodgkin’s lymphoma (52 cases), multiple myeloma (20 cases), nonlymphocytic leukaemia (39 cases) and lymphatic leukaemia (18 cases) was conducted within a cohort of 29 139 men from two chemical manufacturing facilities and a research and development centre (Ott et al., 1989). Cases that had died between 1940 and 1978 were each matched with five controls from the total employee cohort employed in the same decade with the same survival period. Exposure to 21 chemicals (including formaldehyde) was determined based on workplace area and activities. ORs for formaldehyde were 2.0, 1.0, 2.6 and 2.6 for non-Hodgkin’s lymphoma, multiple myeloma, nonlymphocytic leukaemia and lymphocytic leukaemia, respectively (based on only 1 – 2 cancers of each type). Confidence intervals were not reported. It was reported that the age adjusted analysis did not significantly change the ORs (data not presented).

Cancer mortality and incidence were investigated among workers exposed to formaldehyde at a Swedish plant manufacturing abrasive materials (Edling et al., 1987). The cohort consisted of 911 workers employed between 1955 and 1983. Exposure to formaldehyde was reported to be 0.1 – 1.0 mg/m³ (no further details provided). Expected numbers were calculated using the person-years-at-risk method for the national population and stratified for age, calendar year and gender. Mortality from all cancers was close to the expected rate. A non-significant increased risk was observed for non-Hodgkin’s lymphoma (SMR = 2.0, 95% CI 0.2 – 7.2) and multiple myeloma (SMR = 4.0, 95% CI 0.5 – 14.4). This analysis was based on the presence of only 2 cancers of each type in the exposed group. No other lymphohaematopoietic cancers were observed.

Information is also available from a number of cohort studies in professionals, such as embalmers, funeral directors and pathologists. While it would be anticipated that occupational exposure would include formaldehyde among such

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1 Only the abstract was available in English
professionals, no information on occupational exposure was reported in these studies and, hence, the etiologic agent could not be identified.

A study of the mortality of pathologists and medical laboratory technicians in the UK by Harrington and Shannon (1975) was followed up by Harrington and Oakes (1984), and new entrants added to the cohort. A further, and most recent, follow up of this cohort was by Hall et al. (1991) who also included additional entrants to the cohort. In this most recent study, vital status was determined in a cohort of 4512 members of the Royal College of Pathologists followed from December 1973 to December 1986. Only 3068 male pathologists and 803 female pathologists were analysed and it is not transparent from the article why the 740 unaccounted individuals were not included in the analysis. SMRs were derived and compared with rates in the general population of England, Wales or Scotland adjusted for gender, age and calendar time. Mortality from all cancers was significantly below the expected rate for males in England and Wales (SMR = 0.4, 95% CI 0.3 – 0.6) but was close to that expected for females in England and Wales. Increased risks, not statistically significant, were seen for lymphatic and haematopoietic cancers, and specifically leukaemia, in male (SMR = 1.4, 95% CI 0.7 – 2.7 and SMR = 1.3, 95% CI 0.3 – 3.7, respectively) and females (SMR = 1.8, 95% CI 0.4 – 9.8 and SMR = 4.3, 95% CI 0.1 – 24.2) in England and Wales. No information on lymphatic and haematopoietic cancers or leukaemia was reported for male pathologists in Scotland.

The causes of mortality of 3649 white and 397 non-white male US embalmers and funeral directors, who had died between 1975 and 1985 were examined (Hayes et al., 1990). Subjects had been identified through licensing boards and state funeral directors’ associations from 32 states and the District of Columbia, the National Funeral Directors Association and nine state offices of vital statistics. The proportionate mortality ratio (PMR) and the proportionate cancer mortality ratio (PCMR) were determined and compared with the national population adjusted for sex, race, age and calendar year. For PMRs the mortality for all cancers was significantly greater than expected for whites and non-whites. A statistically significant excess was seen for embalmers and funeral directors for lymphatic and haematopoietic cancers (PMR = 1.3, 95% CI 1.1 – 1.6 for whites, and PMR = 2.4, 95% CI 1.4 – 4.0 for non-whites). The PCMR for these cancers was also significantly elevated (PCMR = 1.3, 95% CI 1.1 – 1.6). When analysis of cell-type-specific mortality was undertaken a borderline statistically significant excess was seen in white males only for myeloid leukaemia (PMR = 1.6, 95% CI 1.0 – 2.4) and other unspecified leukaemia (PMR = 2.1, 95% CI 1.2 – 3.3). Additionally, when lymphatic and haematopoietic cancers were examined by occupation, a statistically significant excess was seen for funeral directors (PMR = 1.6, 95% CI 1.2 – 1.9) but not embalmers.

A mortality study of male pathologists listed in the US Radiation Registry of Physicians and the American College of Pathologists was conducted (Logue et al., 1986). The cohort consisted of 5585 members enrolled from January 1962 to December 1977 and followed to December 1977. Age adjusted mortality rates were compared with a cohort of 7942 male radiologists. Additionally, SMRs were determined using the person-years method and compared with deaths in white males for the national population in 1970. SMRs were adjusted for age and calendar time for many causes of death. The age-adjusted mortality for all cancers was slightly lower in pathologists compared to radiologists, as was mortality for
each of lymphatic and haematopoietic cancers, and leukaemia. The SMRs for lymphatic and haematopoietic cancers and leukaemia in pathologists were 0.48 and 1.06, respectively. Confidence intervals were not reported, but neither of these values was statistically significant.

A mortality study of members of the American Association of Anatomists was conducted (Stroup et al., 1986). The cohort consisted of 2317 men who joined the association between 1888 and 1969. Vital status was determined between 1925 and 1979. SMRs were derived for the US white male population for the period 1925 to 1979 and for the male members of the American Psychiatric Association (APA) who joined between 1900 and 1969 as reference groups. SMRs, also adjusted for age and time-specific mortality rates, were compared with the national population. Mortality from all cancers was significantly less than expected (SMR = 0.6, 95% CI 0.5 – 0.8). An increased risk, not statistically significant, was seen for leukaemia (SMR = 1.5, 95% CI 0.7 – 2.7) in anatomists compared to the US white male population. Cell-type-specific mortality rates for US white males were available beginning 1969, and for the period 1969 to 1979. An increased risk was seen for chronic myeloid leukaemia (SMR = 8.8, 95% CI 1.8 – 25.5) though this increase was based on only 3 cases. In contrast, when members of the APA were used as the reference group no increased risk was seen for leukaemia, though this analysis was only up to 1969 and did not undertake cell-type-specific mortality for leukaemia.

A study of the mortality of Ontario (Canada) undertakers was conducted (Levine et al., 1984). The cohort consisted of 1477 men licensed during 1928 through to 1957 and followed up until the end of 1977. Because mortality rates were not available before 1950, person years and deaths in the cohort were not analysed prior to this date. Therefore, SMRs adjusted for age and calendar year were derived and compared with men in Ontario between 1950 and 1977. Mortality from all cancers was slightly lower than expected. SMRs were not consistently reported for the various cancers. For lymphatic and haematopoietic cancers, 8 were observed compared to 4 expected, and specifically for leukaemia 4 were observed compared to 2.5 expected. However, these observed increases were not statistically significant.

A cohort study of the mortality of embalmers licensed in California (US) consisted of 1007 white males licensed between 1916 and 1978 and who died between 1925 and 1980 (Walrath & Fraumeni, 1984). PMRs and PCMRs were determined and compared with the national population adjusting for age, race and calendar year. The PMR for mortality from all cancers was significantly greater than expected (PMR 1.2). The PMR for cancers of the lymphatic and haematopoietic system was 1.2 and specifically for leukaemia 1.75, which was a statistically significant excess. Among embalmers licensed for 20 years or more the PMR for leukaemia was also statistically significant (PMR 2.2). Additionally, for leukaemia, 6 of the 12 observed cases were myeloid (4 expected). Confidence intervals were not reported in this study. The number of observed lymphosarcoma and reticulosarcoma cancer deaths was not elevated.

A study of the mortality of embalmers licensed in New York State (US) was conducted (Walrath & Fraumeni, 1983). The cohort consisted of 1132 white males and 79 non-white males licensed between 1902 and 1980 and who died between 1925 and 1980. PMRs and PCMRs were determined and compared with the national population adjusting for age, race and calendar year. The PMR for
mortality from all cancers was slightly greater than expected for white males and significantly elevated in non-white males (PMR 1.4). The PMR for lymphatic and haematopoietic cancers, lymphoma and reticulosarcoma, other lymphatic cancers and leukaemia was 1.2, 1.1 (PCMR 0.8), 1.2 and 1.4 (PCMR 1.2), respectively, for white males. Confidence intervals were not reported but none of these values was statistically significant. For leukaemia, of the 12 observed cases 6 were myeloid (4.1 expected). For non-white males it was reported that mortality from cancers of the lymphatic haematopoietic system was significantly increased (data not provided, but stated to be on the observation of only 3 such cases). There was no significant difference in PMRs for white males when analysed by time from first licence and by age at first licence.

Summary

Several epidemiology studies have shown a small increased risk for lymphohaematopoietic cancers, particularly myeloid leukaemia, in workers who may have been exposed to formaldehyde at work. This has been observed principally in studies of professional workers. In these studies, no information on occupational exposures was available and it cannot be excluded that the observed increases were due to occupational exposures other than formaldehyde. Until recently, these findings have not been supported by studies of industrial workers. However, 2 of 3 recent updates of cohort studies of industrial workers provide some evidence for increased risk. An association was seen in an analysis of the largest cohort of US industrial workers by Hauptmann et al. (2003) between peak exposure to formaldehyde and leukaemia, with a stronger association for myeloid leukaemia. However, a reanalysis of the data by Marsh and Youk (2004), using additional analysis, provided little evidence to support the suggestion of a causal association. An increased risk for leukaemia was also seen in a large cohort of US garment workers (Pinkerton et al., 2004), while no such increased risk was observed in a large cohort of UK industrial workers (Coggon et al., 2003). Overall, it is considered that the epidemiology data are insufficient to establish a causal association between occupational exposure to formaldehyde and leukaemia. This conclusion is supported by a recent evaluation of the substantial biological evidence on the disposition and toxicity of inhaled formaldehyde in experimental animals and humans, particularly as it pertains to effects on the blood and bone marrow (Heck and Casanova, 2004). The authors of this review, which did not include an evaluation of the available epidemiology evidence, concluded that a leukemogenic effect of inhaled formaldehyde is not biologically plausible. Heck and Casanova (2004) give several reasons for drawing this conclusion, including rapid metabolism at the site of deposition, no measurable effects on bone marrow tissues in several species following inhalation exposure, and failure of formaldehyde to induce leukaemia in several long-term bioassays.

Pancreatic cancer

Collins et al. (2001) conducted a meta-analysis of 14 epidemiological studies (8 cohort mortality studies\(^1\), 4 proportionate mortality\(^2\) and 2 case-control studies\(^3\), published between 1983 – 1999, that reported pancreatic cancers and

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\(^1\) Levine et al., 1984; Blair et al., 1986; Stroup et al., 1986; Stayner et al., 1988; Matanoski, 1991; Hall et al., 1991; Gardener et al., 1993; Andjelkovich et al., 1995


\(^3\) Gerin et al., 1989; Kernan et al., 1999
occupational exposure to formaldehyde. Direct exposure measurements were provided in some studies, for others information on job titles was used to determine exposure levels. Overall, a very slight increased risk was seen for pancreatic cancer and formaldehyde exposure (mRR = 1.1, 95% CI 1.0 - 1.3) with no substantial heterogeneity seen across studies (p = 0.12). When studies were stratified by occupation the greatest risk was seen in embalmers (mRR = 1.3, 95% CI 1.0 - 1.6) and pathologists and anatomists (mRR = 1.3, 95% CI 1.0 - 1.7) with a greater heterogeneity seen (p = 0.90 and p = 0.30, respectively), indicating a greater consistency among studies when stratified by job type. No increased risk was seen for industrial workers (mRR = 0.9, 95% CI 0.8 - 1.1), who the authors reported were likely to have had higher average exposure and higher peak exposures to formaldehyde. Additionally, in the only two studies that evaluated pancreatic cancer risk with exposure levels (Blair et al., 1986; Kernan et al., 1999 – both in industrial workers), no linear trend was seen for pancreatic cancer and increasing exposure to formaldehyde. Thus, it cannot be excluded that exposures other than formaldehyde may have attributed to the very small increased risk observed among embalmers, and pathologists and anatomists, while the exclusion of studies with no reported cases of pancreatic cancer among formaldehyde workers may have biased the review towards a positive result.

Ojajarvi et al. (2000) conducted a meta-analysis of 92 epidemiological studies published between 1969 and 1998 that reported cases of pancreatic cancer and occupational exposure(s) and/or job categories. These 92 studies, which were not clearly identified, presented data for 161 different exposed populations, with exposure assessed in 57 populations through job titles, in 25 through expert assessments, in 15 through job exposure matrices, and in 60 through other, mixed, or unexplained methods. Industrial hygiene measurements were available for only 4 populations. Data were organised and analysed by populations rather than studies. A total of 5 populations were identified that had received exposure to formaldehyde. It is not reported how exposure was assessed in these five populations. No increased mRR was seen for formaldehyde exposure and pancreatic cancers overall. Similarly, stratification of studies by sex and diagnostic quality (i.e. whether histological diagnosis was conducted) or study type did not result in an increased mRR.

A population-based case-control study based on death certificates from 24 US states was conducted to determine if occupations/industries or work-related exposures to solvents (including formaldehyde) were associated with pancreatic cancer deaths (Kernan et al., 1999). A total of 63 097 deaths from pancreatic cancer were identified between 1984 - 1993, and matched by state, race, gender and age to 252 386 controls who died from causes other than cancer in the same time period (excluding deaths due to pancreatic diseases). Data on occupation and industry were obtained from death certificates, and exposure determined using a job-exposure matrix. After adjustment for potential confounding factors, such as age, race, gender, marital status, metropolitan and residential status, a significantly increased risk was observed between low and medium levels of formaldehyde exposure and pancreatic cancers in white males (OR = 1.2, 95% CI 1.1 – 1.4 and OR = 1.2, 95% CI 1.1 – 1.3, respectively) and low, medium and high levels of formaldehyde exposure in white females in the absence of a dose response (OR = 1.3, 95% CI 1.1 – 1.5, OR = 1.4, 95% CI 1.2 – 1.7 and OR = 1.3, 95% CI 1.0 – 1.7). Similarly for probability of exposure, a significantly increased risk was only seen between low and medium probabilities of formaldehyde.
exposure and pancreatic cancers in white males. For white females, a significant, dose-related, increased risk was seen for low, medium and high probabilities of formaldehyde exposure (OR = 1.3, 95% CI 1.1 - 1.6, OR = 1.4, 95% CI 1.2 - 1.7 and OR = 1.5, 95% CI 1.3 - 1.9, respectively). No significantly increased risks of pancreatic cancer were seen in black males and black females between formaldehyde exposure intensity and probability of exposure. When sex and racial type were pooled together and analysed according to probability of exposure a significantly increased risk was seen for low (OR = 1.2, 95% CI 1.1 - 1.3), medium (OR = 1.2, 95% CI 1.1 - 1.3) and high (OR = 1.4, 95% CI 1.2 - 1.6) probabilities. In contrast, when cases were analysed according to intensity of exposure, a significant increase was only seen for low (OR = 1.2, 95% CI 1.1 – 1.3) and medium exposure levels (OR = 1.2, 95% CI 1.1 –1.3). Although a dose-response pattern was not apparent for intensity of exposure, the dose-response relationship for probability of exposure was usually consistent across each level of exposure intensity, though this is attributed to incidences observed in white females and not white males, black males or black females.

Overall, these studies do not support an association between formaldehyde exposure and pancreatic cancers.

11.7 Reproductive toxicity

Only limited information is available for this endpoint in humans. A Finnish retrospective study examined fertility among female woodworkers exposed to gaseous formaldehyde between 1985 and 1995 (Taskinen et al., 1999). Data on pregnancy history, time to pregnancy, occupational exposure and previous gynaecological diseases were obtained by self-reported questionnaires. From a total of 1094 women who had delivered at least one child since working in the wood industry 602 (55%) responded to a mailed questionnaire. This total contained 235 women who were exposed to formaldehyde. For women exposed to formaldehyde, workplace exposure measurements were obtained. If such information was not available a judgement was made to obtain exposure information from a “comparable” workplace. Women were assigned into low (119 cases), medium (77 cases) and high (39 cases) dose groups, for which mean exposure levels were determined to be 0.07, 0.14 and 0.33 ppm formaldehyde, respectively. Time to pregnancy data were used to determine the fecundability density ratio (FDR) of women exposed to formaldehyde compared to those who were not exposed. Following adjustments for potential confounders, such as employment, maternal smoking and alcohol consumption, irregular menstrual cycles and number of children, the FDR was significantly decreased in the high dose group only (0.64, 95% CI 0.43-0.92). FDR values in the medium and low dose groups were 0.96 (95% CI 0.72-1.26) and 1.09 (95% CI 0.86-1.37), respectively. Exposure to other workplace chemicals, such as organic solvents and phenols, was not associated with decreased FDR.

However, limitations are present in the design of this study, such as the use of judgement or self-reports of workplace exposure to gaseous formaldehyde. This could have introduced recall bias into the study. When workplace exposure data were obtained, it is unclear what type of monitoring data were used (e.g. personal or area exposure data). Failure to clinically diagnose an effect on fertility in women who reported increased time to pregnancy is also a study limitation. Furthermore, as the degree of fertility is related to both partners, fathers should
have been interviewed to determine any confounding factors, and if required, examination of paternal exposure conducted. Overall, the limitations in study design prevent any reliable conclusions to be drawn from the data on the potential reproductive toxicity of formaldehyde.

In a Russian cross-sectional study of female workers exposed to gaseous formaldehyde through use of urea-formaldehyde resins by Shumilina (1975) (reported in Russian, summary from IPCS, 1989), though an increased incidence of menstrual disorders and problems with pregnancy were reported, there was no difference in fertility between the exposed and control groups. However, the limited details reported together with the presence of possible confounding factors that were not evaluated mean that no reliable conclusions can be drawn from this study.

A cross-sectional study investigated sperm count and morphology in 11 autopsy workers exposed to formaldehyde for between one month and “several” years (Ward et al., 1984). Time-weighted exposures of 0.61-1.32 ppm gaseous formaldehyde (weekly exposure range 3-40 ppm/hour) were obtained from personal and area monitoring. Exposed workers were matched for age and customary use of alcohol, tobacco and marijuana to controls. No effects on sperm count or morphology were observed in formaldehyde-exposed workers. However, the small study size limits the significance that can be attached to this result.

11.8 Developmental toxicity

A number of epidemiology studies are available investigating the effects of occupational exposure to a number of chemicals, including formaldehyde, on spontaneous abortions. These surveys have reported conflicting results on the relative risk (RR) of spontaneous abortion among women occupationally exposed to formaldehyde.

In a cross-sectional study of female workers in university laboratories in Sweden, the RR was calculated to be 2.6 (95% CI 0.9-7.4) among 10 women exposed to formaldehyde (Axelsson et al., 1984). In an American case-control study, the RR was calculated to be 2.1 (95% CI 1.0-4.3) in 51 cosmetologists (e.g. hairdressers and beauticians) exposed to formaldehyde after adjustment for potential confounders (John et al., 1994). In a Finish case-control study of female workers in laboratories the RR was calculated to be 3.5 (95% CI 1.1-11.2) in 11 women exposed to formaldehyde (Taskinen et al., 1994). A Finnish cohort study evaluated spontaneous abortions in 52 female wood workers and calculated the RR to be 3.2 (95% CI 1.2-8.3), 1.8 (95% CI 0.8-4.0) and 2.4 (95% CI 1.2-4.8) in the high, medium and low formaldehyde exposure groups, respectively, after adjustment for potential confounders (Taskinen et al., 1999).

In contrast, no increased RR of spontaneous abortion and occupational exposure to formaldehyde was seen in a Finish cohort study of 50 hospital sterilising staff (Hemminki et al., 1982), a Finish case-control study of 30 nurses (Hemminki et al., 1985), a French cohort study of 139 nurses (Stucker et al., 1990), and a Finish population-based case-control study of 1808 women (Lindbohm et al., 1991) who all reported exposure to formaldehyde. Additionally, no increased RR was seen between occupational exposure to formaldehyde and malformations in those studies that assessed this outcome (Hemminki et al., 1985; Taskinen et al., 1994).
A comprehensive review of all the available data, including the meta-analysis data evaluating the relationship between spontaneous abortions and occupational exposure to formaldehyde, was conducted by Collins et al. (2001). For studies that showed an increased RR, some important limitations in study design were highlighted, such as the use of self-reported data or judgement on the level of exposure with no attempt to validate the exposure estimates with measurements. Furthermore, only the studies by John et al. (1994) and Hemminki et al. (1982) made adjustments to RR estimates for important confounding factors, such as age, heavy lifting or prolonged standing, though none of the studies examined other exposures that may have contributed to the risk of spontaneous abortions.

For the meta-analysis, when occupation was considered, an increased mRR for spontaneous abortions was only observed among laboratory workers. However, this only occurred in those studies that relied on self-reports of exposure, suggesting a potential recall bias. Additionally, no increased mRR was seen in studies that used evaluation of work tasks to determine exposure. Furthermore, evidence of publication bias was found, as increased mRRs were limited to small studies. When these biases were taken into account no association was seen between spontaneous abortions and exposure to formaldehyde (mRR= 0.7 [95% CI 0.5-1.0]).

A Lithuanian population-based case-control study investigating low birth weight is available (Grazuleviciene et al., 1998). Data were obtained from self-reported questionnaires and geographic air pollution data. No statistically significant association between low birth weight and formaldehyde exposure was seen after adjustment for confounding factors, such as education, smoking status, maternal hazardous work, parity and infectious diseases. Axelsson et al., (1984) and Taskinen et al. (1994) also found no association between low birth weight and formaldehyde exposure. Low birth weight of offspring, anaemia and toxæmia were more frequent in the formaldehyde-exposed group than controls in a study by Shumilina (1975). The limited details reported, together with the presence of possible confounding factors that were not evaluated, means that no reliable conclusions can be drawn from this study (reported in Russian, summary from IPCS, 1989).
12. Hazard Classification

This section discusses the classification of the health effects of formaldehyde according to the NOHSC Approved Criteria for Classifying Hazardous Substances (the Approved Criteria) (NOHSC, 2004). The Approved Criteria are cited in the NOHSC National Model Regulations for the Control of Workplace Hazardous Substances (NOHSC, 1994c) and provide the mandatory criteria for determining whether a workplace chemical is hazardous or not.

Where adequate human data were unavailable and/or inappropriate, the classification for health hazards has been based on experimental studies (animal and in vitro tests). In extrapolating results from experimental studies to humans, consideration was given to relevant issues, such as quality of data, weight of evidence, metabolic and mode of action/mechanistic profiles, inter- and intra-species variability and relevance of exposure levels.

Classification of formaldehyde in accordance with the OECD Globally Harmonized System of Classification and Labelling of Chemicals (GHS) (UNSCEGHS, 2005) can be found in Appendix 4.

Formaldehyde is currently listed in the OASCC’s Hazardous Substances Information System (DEWR, 2004) with classification of R23/24/25 (toxic by inhalation, in contact with skin, and if swallowed), R34 (causes burns), R43 (may cause sensitisation by skin contact) and R40 (limited evidence of a carcinogenic effect, Category 3 carcinogen).

12.1 Acute toxicity

Although there are old reports of human deaths following ingestion of formaldehyde solution, no reliable quantitative data are available on the doses consumed. Recent cases reported ulceration and damage along the aero-digestive tract, with a feeding jejunostomy performed following ingestion of approximately 700 mg/kg bw of formaldehyde solution, and a tracheostomy and gastrectomy performed following ingestion of an unquantifiable dose. In animal studies, oral LD50 values of 800 and 260 mg/kg bw are available in the rat and guinea-pig, respectively. A dermal LD50 of 270 mg/kg bw in the rabbit, and 4-hour inhalation LC50 values of 480 and 414 ppm (0.578 and 0.497 mg/L) in the rat and mouse, respectively, are also available.

The LC50 value in rats, the preferred species, equates to ‘toxic’ by inhalation while the value in mice is almost at the cut-off value for toxic/very toxic. Thus, it is proposed that the classification as ‘toxic’ be retained. The oral LD50 values support classification as ‘harmful’. However, although no deaths occurred in recent cases of ingestion in humans they are considered to represent a potentially lethal dose given the significant toxicity observed, and drastic medical procedures undertaken. Consequently, it is considered appropriate to regard formaldehyde as ‘toxic’ by the oral route and retain its current classification as such. The dermal LD50 value in rabbits supports classification as ‘toxic’.

Classification: Based on the human and animal data, formaldehyde meets the Approved Criteria for classification as ‘Toxic by inhalation’ (risk phrase R23),
‘Toxic in contact with skin’ (risk phrase R24) and ‘Toxic if swallowed’ (risk phrase R25).

12.2 Irritation

Skin reactions have been reported in humans, however, because formaldehyde solution is a known skin sensitiser it is difficult to determine whether observed reactions are due to irritation or sensitisation.

In animals, although formaldehyde solution is reported to be a primary skin and eye irritant, this is based on old anecdotal evidence rather than robust animal studies. Data are available from a recent rabbit low-volume eye test (LVET) where 10 x1 of 37% formaldehyde solution produced irritation of the cornea, conjunctiva and iris three hours post-instillation. Additionally, ‘necrosis/loss’ of corneal kerocytes was reported in eyes from animals sacrificed one day post-instillation, and corneal injury was determined to extend at times to 93.2% of corneal thickness. In a repeated dermal study in mice, skin irritation was reported following application of ≥0.5% formaldehyde solution, 5 days/week for 3 weeks.

A single 6-hour exposure to 15 ppm (18 mg/m³) gaseous formaldehyde produced histological changes to the nasal tract of rats indicative of a direct irritant effect. Data are also available in Alarrie assays in mice. Although the reliability of this assay has been questioned (i.e. non-reproducibility of results and species variation in RD50 values) the data supports the histological findings that gaseous formaldehyde causes irritation to the respiratory tract.

Thus, there are sufficient data to show formaldehyde is a skin, eye and respiratory irritant. The observations of severe irritation in the rabbit LVET and comprehensive injury to the cornea with 10 x1 of 37% formaldehyde solution, along with skin irritation at concentrations ≥0.5% in a mouse repeat dermal study, raise concerns that corrosivity could be observed if animal studies were conducted to OECD Test Guidelines, i.e., at higher concentrations in skin studies and with 0.1 ml in eye studies. Additionally, corrosive injuries to the oesophagus and stomach were observed in humans following ingestion of formaldehyde solution. Consequently, it is considered appropriate to regard formaldehyde solution as corrosive.

Classification: Based on the human and animal data, including observations in cases of human ingestion, formaldehyde meets the Approved Criteria for classification as ’causes burns’ (risk phrase R34).

12.3 Sensitisation

Formaldehyde solution is a known skin sensitiser and is included in standard series for patch testing. In addition to skin sensitisation being clearly observed in numerous clinical trials and case reports in humans, positive results have been observed in a large number of animal studies in guinea-pigs and mice.

When determining whether a chemical is a respiratory sensitiser immunological mechanisms do not have to be demonstrated, and for human evidence it is necessary to take into account the size of the population and the extent of exposure. Although large numbers of people are exposed to gaseous formaldehyde, there are very few reported cases of well-conducted bronchial
challenge tests in humans giving a positive response to formaldehyde. Conversely, several studies have reported negative bronchial challenge tests. However, limited evidence indicates that formaldehyde may elicit a respiratory response in some very sensitive individuals with bronchial hyperactivity, probably through irritation of the airways. Additionally, studies determining the effect on lung function following workplace exposure to formaldehyde in air, along with epidemiology studies, do not indicate formaldehyde to be a respiratory sensitiser. There is generally little correlation between the presence of formaldehyde-specific antibodies and respiratory symptoms in humans. Similarly, in animals, the results of immunoglobulin-E tests and cytokine profiles do not provide evidence that formaldehyde can induce respiratory sensitisation, though there is limited evidence available indicating that it may enhance allergic responses to other respiratory sensitisers. Thus, the available human and animal data indicates formaldehyde in air is unlikely to induce respiratory sensitisation.

**Classification:** Based on the human and animal data formaldehyde meets the Approved Criteria for classification as ‘May cause sensitisation by skin contact’ (risk phrase R43) but not for sensitisation by inhalation.

### 12.4 Repeat dose toxicity

Effects on pulmonary function, histological changes within the nasal epithelium, and neurobehaviour were investigated in populations exposed to gaseous formaldehyde in occupational and/or community environments. Though transient decreases in lung function across a work shift have been observed in some studies, overall, the data do not provide conclusive evidence that formaldehyde exposure induces major changes in pulmonary function. Conflicting results for histological changes within the nasal epithelium have been observed for workers occupationally exposed to formaldehyde. Although histological changes were observed in the most extensive and well conducted study (Holmstrom et al., 1989), the weight of causality is weak, due primarily to the limited number of investigations of relatively small populations that do not permit adequate investigations of exposure response. Additionally, it is not reported whether these studies examined other exposures that may have contributed to the observed histopathological changes. This is also true for the observance of histopathological changes in a community study. Consequently, the histopathological findings cannot be attributed to formaldehyde exposure. Likewise, there is presently no convincing evidence that indicates formaldehyde is neurotoxic.

In animals, no evidence of systemic toxicity was seen in rat inhalation and oral studies up to approximately 2 years duration, or in the only dermal study available, a 2- to 3-week rat study. Toxicity in response to irritation was restricted to the site of contact: skin irritation in the dermal study, histological changes in the nasal tract in inhalation studies, and stomach in oral studies.

**Classification:** Based on the available human and animal data formaldehyde does not meet the Approved Criteria for classification as causing serious damage to health by prolonged exposure through inhalation, ingestion or dermal contact.
12.5 Genotoxicity

Overall, epidemiology data from occupational studies investigating cytogenetic effects in nasal and buccal cells are suggestive of formaldehyde having a weak localised genotoxic activity, while the evidence for a systemic activity, including peripheral lymphocytes, is equivocal. Small group sizes and the often limited details reported, limit the significance that can be attached to the observed effects. The main concern is that there was co-exposure to other chemicals in these studies (e.g. phenol in embalming fluid and resins, and wood dust in paper production) whose contribution to the observed effects cannot be precluded. Consequently, no reliable conclusions can be drawn from human data on the genotoxic potential of formaldehyde.

In vitro, formaldehyde was clearly genotoxic in bacterial and mammalian cells: Ames test (+/- S9); gene mutation (-S9); chromosome aberration (+/-S9); SCE (+/-S9); and produced DNA single strand breaks and DNA protein cross-links (-S9). In vivo, several ip and inhalation studies are available in rodents investigating the genotoxicity of formaldehyde in somatic cells. Negative results were seen in bone marrow cytogenetic and micronuclei studies conducted to validated test methodology. A statistically significant increase in chromosomal aberrations (chromatid or chromosome breaks) in the bone marrow was reported in a single study that used a prolonged exposure period (4 months) and for which only limited details are available. Similarly, a positive result was seen in only one of several studies investigating tissues other than the bone marrow; a marginal, but statistically significant, increase in chromosomal aberrations (chromatid or chromosome breaks) in pulmonary macrophages. In the only oral study, which used a non-validated test method, a statistically significant increase in the proportion of cells with micronuclei and nuclear anomalies was seen in cells from the stomach, duodenum, ileum and colon of rats. However, the observed effects clearly correlated with severe local irritation (hyperaemia and haemorrhage), and are thus considered a likely consequence of cytotoxicity. Formaldehyde exposure did induce DPX in the nasal tract of rats and monkeys. In ip studies in germ cells in vivo, effects on sperm morphology and dominant lethal findings were seen in a single study that employed a 5-day exposure period. Although negative studies for germ cells used only a single administration, much higher dose levels were employed.

Thus, the limited positive results in somatic cells in vivo are from cytogenetic studies that employed non-validated test methodology and, as such, neither study is considered to provide conclusive evidence of genotoxicity as uncertainty exists in interpreting the reliability of the data. In contrast, negative findings were observed in several studies conducted to validated test methodology. Similarly, the positive result in a single study in germ cells is not considered to provide conclusive evidence that formaldehyde is a germ cell genotoxicant, as negative results were seen in other studies at higher dose levels. The only other finding was the formation of DPX in the nasal tract following inhalation.

Formaldehyde is genotoxic in vitro, and it appears that the chemical is weakly genotoxic at the site of contact in vivo. The relevance of the finding that formaldehyde is capable of producing DPX formation is unclear.

**Classification:** Based on the human and animal data formaldehyde dose not meet the Approved Criteria for classification as a mutagenic substance.
12.6 Carcinogenicity

There are a large number of epidemiology studies available (case-control and cohort) in industrial workers and professionals, investigating the incidence of cancers in the nasal tract, pharynx or lungs. Conflicting results have been observed in these studies. To consolidate the findings, meta-analysis of the data was conducted by Blair et al. (1990), Partanen (1993) and Collins et al. (1997). No association was seen in any meta-analysis for gaseous formaldehyde exposure and lung cancer. In contrast to earlier meta-analyses, the most comprehensive evaluation of the data by Collins et al. (1997) found no association (all studies combined) between sinonasal cancers and exposure to formaldehyde. An association was observed for nasopharyngeal cancers in this meta-analysis, however, this was considered to be due to non-reporting of expected numbers in some industrial cohort studies. Following an adjustment for non-reporting of expected numbers, a non-significant increased risk was observed for nasopharyngeal cancers. Mixed results (i.e. occasional associations) have been observed for nasopharyngeal cancers in recent (post-1997) case-control and cohort studies. Consequently, it is considered that although the human data do not provide strong evidence of a causal association, it is acknowledged that there is some human evidence that occupational exposure to gaseous formaldehyde may result in the development of nasopharyngeal cancer.

Increased risks of various non-respiratory cancers have occasionally been seen in some studies, with the most evidence being for leukaemia, particularly myeloid leukaemia. A recent update of a major cohort study of industrial workers reported an association for myeloid leukaemia and peak exposures to formaldehyde in air (Hauptmann, 2003). However, a reanalysis of the data, using additional analyses, provided little evidence to support the suggestion of a casual association (Marsh & Youk, 2004). In recent updates of two other major cohort studies of industrial workers, an increased risk of leukaemia was seen in US garment workers (Pinkerton, 2004), while no such increased risk was seen in UK industrial workers (Coggon, 2003). Furthermore, conflicting results were seen in earlier epidemiology studies investigating leukaemia in industrial workers (i.e. a slight increased risk or no risk). Increased risks for leukaemia have been observed in several studies of professional workers (e.g. embalmers), however, data on exposure to formaldehyde is not available for these studies. Overall, the data is considered insufficient to clearly establish an association between formaldehyde exposure and leukaemia. This conclusion is consistent with the present toxicokinetic profile and animal carcinogenicity data for formaldehyde.

In inhalation carcinogenicity animal studies, a significantly increased incidence in nasal squamous cell carcinomas was observed in rats at concentrations $> 6$ ppm formaldehyde. Nasal polyploid adenomas were also observed in a single study at 15 ppm formaldehyde, however, the non-reproducibility of these findings at similar concentrations (14-14.3 ppm) in other studies indicates that they are not treatment related. In contrast, an absence or no significant increased incidence in nasal tumours was observed in mice and hamsters at equivalent or greater exposure concentrations that produced such tumours in rats. In oral carcinogenicity studies, no significant tumour findings were seen in the most comprehensive study available up to the top dose of 82 and 109 mg/kg bw/day in male and female rats, respectively (Til, 1989). Although an increase in ‘haemolymphoreticular tumours’ was seen in male and female rats at the top dose
of 75 and 100 mg/kg bw/day, respectively, in another study, this study was criticised for its 'pooling' of tumour types whose incidence has been inconsistently reported. Similarly, although an increase in papillomas of the forestomach was seen in an initiation/promotion study where rats were administered 0.5% in drinking water for 32 weeks, the study has been questioned over its histological diagnosis of benign tumours. In contrast, no leukaemias or stomach tumours were seen in the most comprehensive study to date, which employed comparable or higher dose levels of formaldehyde solution. No skin tumours were seen in mouse initiation/promotion studies, the only dermal data available. Therefore, the available data in animals do not support formaldehyde being carcinogenic by the dermal or oral routes.

The International Programme on Chemical Safety (IPCS) developed a conceptual framework in 2001 based on the general principles involved in considering the chemical induction of a specific tumour in animals (Sonich-Mullin et al., 2001). The data for nasopharyngeal cancers and leukaemia and formaldehyde exposure have been evaluated using this framework (Appendix 5). The postulated mode of action for nasopharyngeal cancers is that inhalation of formaldehyde causes inhibition of mucociliary clearance, followed by nasal epithelial cell regenerative proliferation resulting from cytotoxicity and DPX that leads to mutation, and consequent tumour formation. By considering the available data in the IPCS framework, it was concluded that the postulated mode of action for formaldehyde-induced tumours in the nose is likely to be relevant to humans, at least qualitatively. In contrast, a mechanism by which formaldehyde may induce leukaemia has not been identified and the framework highlights the low degree of confidence that may be ascribed to the hypothesis that formaldehyde induces leukaemia.

Overall, it is considered that the available epidemiology data are not sufficient to establish a casual relationship between formaldehyde exposure and cancer. For nasopharyngeal cancers there are several epidemiological studies that show an increased risk, whereas other studies do not. There is also clear evidence from inhalation studies of nasal squamous cell carcinomas in the rat, though not the mouse and hamster. The postulated mode of action for these tumours is considered likely to be relevant to humans. Therefore, based on the available nasopharyngeal cancer data, formaldehyde should be regarded as if it may be carcinogenic to humans following inhalation exposure. There are also concerns for an increased risk for formaldehyde-induced myeloid leukaemia, however, the available data are not considered sufficient to establish an association and there is currently no postulated mode of action to support such an effect.

IARC concluded that formaldehyde is carcinogenic to humans (Group 1), on the basis of sufficient evidence in humans and sufficient evidence in experimental animals. IARC’s conclusion is as follows:

*Nasopharyngeal cancer mortality was statistically significantly increased in a cohort study of United States (US) industrial workers exposed to formaldehyde, and was also increased in two other US and Danish cohort studies. Five of seven case-control studies also found elevated risk for formaldehyde exposure. The Working Group considered it was “improbable that all of the positive findings…could be explained by bias or by unrecognised confounding effects” and concluded that there is sufficient evidence in humans that formaldehyde causes nasopharyngeal
cancer. Leukaemia mortality, primarily myeloid-type, was increased in six of seven cohorts of embalmers, funeral-parlour workers, pathologists, and anatomists. These findings had previously been discounted because an increased incidence of leukaemia had not been seen in industrial workers. Recent updates, however, report a greater incidence of leukaemia in two cohorts of US industrial workers and US garment workers, but not in a third cohort of United Kingdom chemical workers. The Working Group concluded that there is “strong but not sufficient evidence for a causal association between leukaemia and occupational exposure to formaldehyde”. Several case-control studies have associated exposure to formaldehyde with sinonasal adenocarcinoma and squamous-cell carcinoma. However, no excess of sinonasal cancer was reported in the updated cohort studies. The Working Group concluded that there is limited evidence in humans that formaldehyde causes sinonasal cancer.

In rats, several inhalation studies have shown that formaldehyde induces squamous-cell carcinoma of the nasal cavity. Four drinking-water studies gave mixed results. Formaldehyde also shows cocarcinogenic effects when inhaled, ingested, or applied to the skin of rodents.

Formaldehyde is genotoxic in in-vitro models, animals and humans. Increased numbers of DNA–protein crosslinks have been found in peripheral blood lymphocytes of exposed workers, in the upper respiratory tract of monkeys, and in the rat nasal mucosa. Cell proliferation increases substantially at formaldehyde concentrations higher than six parts per million in rats, amplifying the genotoxic effects. The Working Group concluded that, “both genotoxicity and cytotoxicity have important roles in the carcinogenesis of formaldehyde in nasal tissues”. By contrast, the Working Group could not identify a mechanism for leukaemia induction, and this tempered their interpretation of the epidemiological evidence.’ (IARC, 2004b).

The available data do not support formaldehyde being carcinogenic by the dermal or oral routes.

**Classification:** Based on the above, formaldehyde meets the Approved Criteria for classification as a Category 2 carcinogen with risk phrase R49 ‘May cause cancer by inhalation’. This is a different category with the IARC classification which is Category 1, (known human carcinogen), principally due to differences in the carcinogen classification criteria and also consideration of the weight of evidence.

12.7 Reproductive effects

Only a few epidemiology studies are available. A retrospective investigation of fertility reported a significant increase in the time to pregnancy (i.e. decrease in the fecundability density ratio) in female workers exposed to formaldehyde. However, limitations in study design prevent any reliable conclusions being drawn from the data. Similarly, in cross-sectional studies, although no difference was seen in female fertility or male sperm count and morphology between formaldehyde exposed workers and controls, study limitations restrict the significance that can be attached to the data.
In the only fertility study available in animals, formaldehyde did not produce an adverse effect on fertility in minks, though there are concerns that formaldehyde was not robustly tested in this oral study. No effect on epididymal sperm morphology was seen in an oral mouse study at the only dose tested, and no effects on the testes have been reported in rodents in a chronic repeat oral study and chronic inhalation studies. In contrast, although effects have been seen on epididymal sperm following intraperitoneal administration this is not a relevant route of human exposure.

**Classification:** Based on the human and animal data formaldehyde does not meet the Approved Criteria for classification as a reprotoxicant.

### 12.8 Developmental toxicity

There is no human evidence to indicate occupational exposure to formaldehyde is associated with low birth weight or malformations. For studies investigating spontaneous abortions, the inconsistent findings observed in epidemiological studies and limitations in study design, including the potential for recall and publication bias, mean the findings cannot be attributed to occupational exposure to formaldehyde.

In animal studies, the only effect observed following inhalation was a reduction in foetal body weight that was a secondary non-specific consequence of severe maternal toxicity. No effects on development were seen in an oral study though dose levels were not maximised. No robust dermal study is available that allows the developmental toxicity of formaldehyde to be reliably determined.

**Classification:** Based on the human and animal data formaldehyde does not meet the Approved Criteria for classification as a developmental toxicant.
13. Environmental Exposure

13.1 Ambient air concentrations

In this section, the predicted environmental concentration (PEC) of formaldehyde is calculated for various environmental compartments using modelling techniques. The modelling results are presented as annual averages and maximum 24-hour averages. Annual averages are relevant for long-term (chronic) exposure, whereas 24-hour averages are more representative of acute exposure. An averaging time of 24 hours is also specified for formaldehyde in the Air Toxics National Environmental Protection Measure (NEPC, 2004) with the monitoring investigation level set at 40 ppb (see Section 18.1.1 for details). First, a PEC value for each of the point and diffuse sources of release is calculated, and then these values are combined to determine a final PEC. Where available, published monitoring studies are also summarised and used to verify the PEC values.

The formaldehyde release estimates are primarily from the NPI emission database (NPI database at www.npi.ea.gov.au). Most of the NPI emissions data are themselves estimations, determined by a range of techniques, including mass balance calculations, use of emissions factors, and sampling and direct measurement. As such, the PEC predictions should be interpreted cautiously owing to uncertainties in the initial release estimates.

A number of different approaches have been adopted to calculate PECs, depending on the type of source. The modelling was carried out by Commonwealth Scientific and Industrial Research Organisation (CSIRO) Atmospheric Research Division and details of the modelling techniques and results are provided in Appendix 6.

13.1.1 Point source emissions from industry

Emissions of formaldehyde resulting from industrial activities are difficult to assess owing to the high diversity in use patterns and the high number of both small and large companies using formaldehyde or manufacturing products containing formaldehyde. While the NPI estimates are a reasonably good indicator of the major contributors, the data are incomplete. Data from the Australian Bureau of Statistic (ABS) suggest that from 5000 to 10 000 companies should be reporting emissions (although not all of these companies necessarily emit formaldehyde), but only about 3000 facilities reported emissions in the 2001–2002 reporting year and 3400 for the 2002-2003 reporting year.

Figure 13.1 provides a breakdown by industry category of point source emissions from the 34 industries and 196 facilities reporting formaldehyde emissions to the NPI in the financial year 2001-2002 and 38 industries and 257 facilities for the 2002-2003 financial year. These emissions are combined and appear as industry emissions in Figure 8-1. Some of the original NPI industry categories have been changed or combined for this report.

The major industrial contributors of atmospheric point source emissions of formaldehyde are the mining, wood and paper industries, and electricity supply. In the following summaries of point source data, the average emissions are used.
to represent emissions and potential exposure concentrations owing to the wide variability in releases from each industrial facility including some facilities reporting no emissions. The minimum and maximum emissions are also reported.

The detailed emission data for a number of major industries are tabulated in Appendix 7.

The details of modelling for PEC values, such as source configuration and modelling techniques are presented in Appendix 6, Section A2. Only results are reported here. The release estimates used in the modelling are primarily from emissions data listed in the NPI database for the 2001-2002 reporting year. The 2002-2003 NPI data reported in this section became available after the modelling was conducted, therefore, were not used in the PEC estimations. However, it is expected to be directly proportional to those estimated for 2001-2002.

All PEC values are calculated using the conversion factor $1 \text{ ppb} = 1.20 \mu g/m^3$, which is appropriate for ambient conditions of 25 °C.

**Figure 13.1:** Formaldehyde emissions (NPI database) for each industry category for (a) 2001-2002 and (b) 2002-2003. The figure in brackets indicates the number of facilities reporting in each category.
Mining operations

The average and maximum formaldehyde emission rates derived from the NPI database from the various types of mining operations are given in Table A7-1 in Appendix 7.

Metal ore mining activities (iron, gold, silver-lead, or nickel) contributed the highest emissions, although some facilities in this category reported no emissions of formaldehyde. The average emission rate for mining activities was 12 203 kg/year with a maximum of 401 112 kg/year for a nickel mining activity in Western Australia in the 2001-2002 reporting year. For the 2002-2003 reporting year the average emission rate was 7254 kg/year with a maximum of 363 769 kg/year for an iron mining activity in Western Australia.

Emissions of formaldehyde from mining operations are expected to occur mainly via vehicle exhaust from mining equipment and transport, cleaning and site maintenance activities, power generation using fossil fuels, combustion in boilers, and blasting.

The calculated annual average PEC at 100 m from the edge of the activity was 1.8 ppb and the maximum 24-hour average was 8.1 ppb based on the average source emissions for the 2001-2002 reporting year. These results are approximately inversely proportional to the diameter of the area source (for a given emission rate). (see Appendix 6, A2.1 for details)

Given that the main sources of emissions from mining operations are distributed surface sources, the area of emissions is likely to be approximately proportional to the emissions rate, so that PECs from the largest emitter are expected to be similar to those from the average emitter.

Wood and paper product manufacturers

Release estimates from the NPI database for the years 2001-2002 and 2002-2003 indicate the wood and paper manufacturing industry contributed the second highest proportion of point source emissions of formaldehyde from industrial facilities. The average emission rates were 8195 and 7061 kg/year for the 2001-2002 and 2002-2003 reporting years, respectively, with a maximum of 51 844 kg/year (2002-2003 data) for an individual wood products activity (Table A7-2, Appendix 7). This is not surprising considering that one of the primary uses of formaldehyde is in the production of urea formaldehyde and phenol formaldehyde resins, which are used mainly as adhesives in the manufacture of particleboard, fibreboard, and plywood.

Emissions of formaldehyde from the wood and paper industries are expected to occur mainly through fugitive and point source emissions of vapours from process and storage areas, and with some emissions of formaldehyde from combustion activities. The processes emitting vapours will differ with the type of industry, but may include gluing and veneering, steam heating, wood preservation treatment, and drying activities. Combustion sources include wood and paper drying, incinerating, and boiler operations.

The calculated annual average PEC 100 m from a facility with average emission rates was 4.8 ppb and the maximum 24-hour average was 36 ppb. The highest estimated PECs from the largest emitter were 16 ppb (annual average) and 119 ppb (maximum 24-hour average) (see Appendix 6, A2.2 for details).
sensitivity analysis showed that the PECs are much more sensitive to the configuration of the source of the fugitive emissions than the stack emissions. All of the wood and paper product industries in the NPI database are located outside major urban areas.

To refine the estimates, further modelling of formaldehyde emissions from the highest emitter for wood and paper manufacturing industries was undertaken by EML Air Pty Ltd. EML included the typical facility layout, including configuration of the sources of formaldehyde emissions as inputs into the model. The revised estimates for the highest emitter of wood and paper facilities were 2 ppb (annual average PEC) and 37 ppb (maximum 24-hour average PEC) (see Appendix 17). CSIRO reviewed the EML Air Pty Ltd estimates and confirmed that the model had been correctly applied (see Appendix 18).

Limited boundary data for ground formaldehyde levels around wood manufacturing plants were provided by AWPA and PAA. In total, 37 samples were collected around 5 plants between 1999 and 2005. No details on test methods were provided. About half the number of samples (18 out of 37) showed concentrations of formaldehyde < 10 ppb. Two samples of 66 ppb were measured around a plant that emits formaldehyde at ε 20 000 kg/year. There is no indication whether the plant is one of the largest formaldehyde emitters.

**Electricity supply**

Most electricity generated in Australia is produced in steam cycle plants, with over 90% of plants using fossil fuel combustion to drive the steam turbines coupled to the electricity generators. Coal and natural gas are the main fossil fuel sources (ESAA, 1997). Thus, emissions of formaldehyde from the electrical supply industry result primarily from coincidental production during fuel combustion. Discharges are mainly into the air via stacks.

In the NPI reporting years 2001-2002 and 2002-2003, the electrical supply industry reported total emissions of 177 303 and 163 918 kg/year of formaldehyde, respectively, with averages of 4792 and 3998 kg per facility. However, only a small proportion of the electrical supply companies in Australia actually reported emissions to the NPI. In 2001-2002, the majority (33 of 37) of companies reporting emissions were small isolated facilities operating throughout QLD and using diesel internal combustion to generate power. The range of emissions from these facilities varied between 0.92-70 kg/year. The remaining four facilities (3 in NSW, 1 in QLD) reported significantly higher emissions, between 29 012 and 85 614 kg/year, with two of these facilities generating power from coal seam methane. Emissions from combustion of coal-bed gas are likely to be high due to formation of formaldehyde by oxidation of methane.

The calculated PECs are 0.11 ppb (annual average) and 1.12 ppb (maximum 24-hour average). For the largest emitter using different source configuration (see Appendix 6, A2.3 for details), similar PECs of 0.10 ppb (annual average) and 0.98 ppb (maximum 24-hour average) were produced. These PEC estimates are conservative because buoyant plume rise was ignored by setting the efflux temperature to 25°C.
Materials manufacture

Release estimates from NPI for the years 2001-2002 and 2002-2003 indicate that emissions vary widely with the type of material being manufactured (Table A7-3, Appendix 7). The average emission rates were 3664 and 2293 kg/year, respectively.

Basic non-ferrous metal manufacturing contributed the highest emissions with the bulk of emissions from this category being discharged from alumina production facilities (maximum emission rate 35 000 kg/year, in 2001-2002). Emissions from alumina production facilities occur primarily through combustion of fossil fuels in furnaces and boilers during bauxite processing, vent emissions from bulk storage of hydrocarbons, and vapour emissions during certain stages of processing.

The estimated PECs from modelling are 2.1 ppb (annual average) and 16 ppb (maximum 24-hour average). For the largest emitter (an aluminium refinery), PECs of 0.78 ppb (annual average) and 8.2 ppb (maximum 24-hour average) were calculated (see Appendix 6, A2.4 for details).

Petroleum refining, oil and gas extraction

Release estimates from NPI for the years 2001-2002 and 2002-2003 indicate the petroleum refining, and oil and gas extraction industries contributed a total of 21 700 (1085 tonnes x 2%) and 51 000 kg (1022 tonnes x 5%) of point source emissions of formaldehyde (from 6 and 10 reporting facilities), respectively. Emissions ranged between about 5 and 8883 kg per year (average 3162 kg/year) in 2001-2002 and between 14 and 36 150 kg per year (average 5488 kg/year) in 2002-2003, with petroleum refining contributing the highest emissions.

Emissions of formaldehyde from petroleum refining are expected to occur mainly through combustion activities during the refining process (catalytic cracking, fluid coking, blowdown systems, VDU condensers, sulfur recovery), and fugitive emissions from process and storage areas.

For the average emitter, the estimated PECs of 0.07 ppb (annual average) and 0.74 ppb (maximum 24-hour average) are calculated. For the largest emitter (8883 kg/year), the estimated PECs are 0.20 ppb (annual average) and 2.1 ppb (maximum 24-hour average) (see Appendix 6, A2.5 for details).

Chemical industry

Release estimates of formaldehyde to air reported to NPI by the chemical manufacturing industry for the years 2001-2002 and 2002-2003 indicate average emissions of 651 kg from 25 facilities and 399 kg from 27 facilities, respectively, with individual facility emissions ranging between 0 and 6960 kg (Table A7-4, Appendix 7).

Not surprisingly, formaldehyde manufacturing facilities contributed the bulk of emissions reported by the chemical industry (13 445 kg). Emissions estimates to air from formaldehyde manufacturing for 2001-2002 are shown separately in Table A7-5, Appendix 7.
Most of the formaldehyde consumed in Australia each year (~50 000 tonnes) is manufactured here by four chemical manufacturing companies at five sites (Section 7.1 and 7.3).

Formaldehyde emissions from the manufacturing process fall into three main categories: vapour emissions derived from processing and storage (majority, see Section 8.1), liquid effluent contaminated with formaldehyde, and solid wastes containing formaldehyde. Most air emissions occur via stacks, although some fugitive vapour emissions (for example, from storage tanks and discharge areas) may be released directly into the air.

One formaldehyde manufacturer conducted monitoring of stack emissions at discharge points in 2001. It is indicated that process emissions were released from 2 stacks on-site, one for tail gas fed from boilers, and the other for exhaust from the resin distillation process. Tail gas is used as boiler fuel and is discharged only during start-up. The gas passes through two process absorbers prior to release to the atmosphere to remove water, formaldehyde, and methanol from the hydrogen/nitrogen gas mixture. Discharges from the resin distillation process pass through scrubbers prior to release to the atmosphere. It was reported that the only significant stack emissions were 0.72 kg/day from the Resin Reactor 1 (efflux velocity 5.9 m/s). All other sources had emission rates at least 35 times lower than this.

For the average facility (651 kg/year), the maximum estimated annual average PEC was 0.05 ppb and the maximum 24-hour average was 0.41 ppb. For the largest formaldehyde manufacturing plant (6960 kg/year), the maximum estimated annual average PEC was 0.57 ppb and the maximum 24-hour average was 4.4 ppb (see Appendix 6, A2.6 for details).

**Miscellaneous industries**

A number of miscellaneous industries including food manufacturing, farming, textile manufacturing, hospitals and nursing homes, and waste disposal facilities reported formaldehyde emissions to air in 2001-2002. For most of these industries, emission rates were low. The total annual emissions of formaldehyde from all facilities in this category were 3255 kg (i.e. 1085 tonnes x 0.3%, refer to section 8.1.1 and Figure 13.1), and the average for an individual facility was 79 kg. The highest emissions reported for this category were from waste disposal services, with one company reporting 1099 kg/year emissions.

For an average emitter, the estimated PECs were 0.14 ppb (annual average) and 1.2 ppb (maximum 24-hour average). For the largest emitter, the estimated PECs were 2.0 ppb (annual average) and 17 ppb (maximum 24-hour average). (see Appendix 6, A2.7 for details)

**Summary**

Based on the NPI emissions estimates for formaldehyde, point source emissions contributed between 14% to 16% of the total yearly emissions reported to NPI from all sources in 2001-2003. Most emissions from industry were incidental emissions arising from combustion process. Of the industry emissions, the formaldehyde manufacturing industry contributed about 1.2% (13445 kg out of 1085 tonnes) of the total in 2001-2002.
The estimated maximum annual average and maximum 24-hour average PECs for each industry category are shown in Table 13.1. It should be remembered that these PEC predictions have been derived using data from the NPI database in which most of the data has been estimated. As such, the PEC predictions should be interpreted cautiously owing to uncertainties in the initial release estimates. In addition, not all industrial sources report to the NPI.

### Table 13.1: Annual estimated average and maximum 24-hour average PECs for point source emissions of formaldehyde for each industry category (in ppb)

<table>
<thead>
<tr>
<th>Type of industry</th>
<th>Maximum Annual Average PEC</th>
<th>Maximum 24-hour Average PEC</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Average emitter</td>
<td>Largest emitter</td>
</tr>
<tr>
<td>Mining</td>
<td>1.8</td>
<td>H1.8 (expected)</td>
</tr>
<tr>
<td>Wood &amp; paper</td>
<td>4.8</td>
<td>16 (2*)</td>
</tr>
<tr>
<td>Electricity supply</td>
<td>0.11</td>
<td>0.10</td>
</tr>
<tr>
<td>Materials manufacture</td>
<td>2.1</td>
<td>0.78</td>
</tr>
<tr>
<td>Petroleum</td>
<td>0.07</td>
<td>0.20</td>
</tr>
<tr>
<td>Chemical manufacture</td>
<td>0.05</td>
<td>0.57</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>0.14</td>
<td>2.0</td>
</tr>
</tbody>
</table>

*refined estimates by EML Pty Ltd

#### 13.1.2 Diffuse source emissions

**Urban air**

Urban levels of formaldehyde due to diffuse urban emissions were determined by CSIRO from a re-analysis of detailed urban airshed modelling of ambient pollutant concentrations in Melbourne previously undertaken by CSIRO for EPA Victoria (Hurley et al., 2001). The details are provided in Appendix 6, Section A3. The re-analysis generated 24-hour averages to supplement the original modelling of annual average concentrations. The results provide the best available estimate of urban concentrations away from significant local sources, such as industry or large roads. The estimated maximum annual average formaldehyde concentration is 1.6 ppb (Hurley et al., 2001) and the maximum 24-hour average is 13 ppb (see Table 13.2).

When determining the impact of an industrial source located in an urban area, it is common practice (EPA Victoria, 1985) to add the maximum PEC for the industrial source to a typical urban background concentration, represented by the 70th percentile, rather than the maximum 24-hour average urban background, which is unlikely to occur at the same time as the maximum source impact.
Analysis of the cumulative probability distribution from the PPCR modelling indicated that the 70th percentile 24-hour average PEC was 2.2 ppb.

**Table 13.2: PECs of formaldehyde for Melbourne from urban airshed modelling**

<table>
<thead>
<tr>
<th>Averaging time</th>
<th>Maximum PEC</th>
<th>70th percentile PEC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Annual average</td>
<td>1.6 ppb</td>
<td>-</td>
</tr>
<tr>
<td>24-hour average</td>
<td>13 ppb</td>
<td>2.2 ppb</td>
</tr>
</tbody>
</table>

**Roads**

Maximum formaldehyde concentrations due to roadway emissions were determined by CSIRO from modelling of emissions from a 6-lane dual carriageway freeway. The details are provided in Appendix 6, Section A4. The modelling results at three distances from the edge of the freeway are listed in Table 13.3. They show a rapid decrease in concentrations with distance from the edge of the freeway.

**Table 13.3: Formaldehyde PECs for typical large urban freeway (150 000 cars per day) modelled using AUSROADS**

<table>
<thead>
<tr>
<th>Location</th>
<th>Maximum annual average PEC</th>
<th>Maximum 24-hour average PEC</th>
</tr>
</thead>
<tbody>
<tr>
<td>At edge of freeway</td>
<td>0.77 ppb</td>
<td>2.3 ppb</td>
</tr>
<tr>
<td>20 m from edge of freeway</td>
<td>0.37 ppb</td>
<td>1.06 ppb</td>
</tr>
<tr>
<td>100 m from edge of freeway</td>
<td>0.15 ppb</td>
<td>0.50 ppb</td>
</tr>
</tbody>
</table>

**13.1.3 Natural background concentrations**

Formaldehyde is formed naturally in the atmosphere and biosphere by a variety of processes, the most important of which are oxidation of methane and isoprene. As such, background concentrations also need to be incorporated in calculation of the PECs. Assuming natural methane oxidation is the only source, Lowe et al. (1980) predicted natural background concentrations of formaldehyde in the atmosphere in the order of 0.4 ppb at the ground surface, decreasing to about 0.1 ppb at an altitude of 5 km. This agrees with measurements in clean marine air at Cape Grim (northern Tasmania) by Ayers et al. (1997), who reported a 24-hour average of 0.4 ppb in summer.

The US EPA (1993) predicted that in remote areas, oxidation of methane combined with oxidation of biogenic hydrocarbons, such as isoprene, produced background concentrations of about 0.6 ppb during daylight hours. In contrast, measurements in the Latrobe Valley in Australia from rural sampling sites showed 2-hour average concentrations between 2 and 3 ppb with a recommended representative summer regional background concentration of 2 ppb (Carnovale & Ramsdale, 1988). This result indicates a significant contribution from the oxidation of isoprene, which is much smaller in the non-summer months. This would reduce the annual average below 2 ppb. Thus, for the purpose of this
assessment, it is assumed that natural background formaldehyde concentrations are 2 ppb (maximum 24-hour average) and 1 ppb (annual average).

13.1.4 Combining PECs from all sources

Table 13.7 summarises the contribution from the various sources modelled and the estimated natural background concentration. The PECs from the wood and paper industries have been separated from the other industries because they are all located away from major urban centres.

The total PECs (without the wood and paper industries) represent an expected extreme worst-case formaldehyde concentration in an urban area. It includes the 70th percentile PEC due to diffuse urban sources, the natural background concentration, the worst-case contribution from an urban freeway, and the worst-case contribution from a nearby industry. The total PECs are 5.5 ppb (annual average) and 23.5 ppb (maximum 24-hour average).

13.1.5 Measured data

Monitoring data are available from a number of locations and environments in Australia, predominantly Victoria, Queensland, South Australia and Western Australia.

Table 13.4 provides ambient formaldehyde levels (24 h average) measured at two sites in Brisbane, which have been monitored for over two years by the Queensland EPA (Pattearson, 2002).

Table 13.4: Ambient formaldehyde concentrations in the Brisbane CBD and at Wynnum, QLD (in ppb)

<table>
<thead>
<tr>
<th>Location</th>
<th>Season</th>
<th>Minimum</th>
<th>Maximum</th>
<th>Median</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wynnum</td>
<td>Summer</td>
<td>1.5</td>
<td>10.6</td>
<td>4.5</td>
<td>4.6</td>
</tr>
<tr>
<td>Brisbane CBD</td>
<td></td>
<td>1.4</td>
<td>5.9</td>
<td>2.7</td>
<td>2.9</td>
</tr>
<tr>
<td>Wynnum</td>
<td>Autumn</td>
<td>1.2</td>
<td>10.7</td>
<td>5.5</td>
<td>5.6</td>
</tr>
<tr>
<td>Brisbane CBD</td>
<td></td>
<td>0.8</td>
<td>7.1</td>
<td>2.6</td>
<td>2.8</td>
</tr>
<tr>
<td>Wynnum</td>
<td>Winter</td>
<td>3.0</td>
<td>17.8</td>
<td>7.5</td>
<td>7.7</td>
</tr>
<tr>
<td>Brisbane CBD</td>
<td></td>
<td>0.9</td>
<td>7.7</td>
<td>3.0</td>
<td>3.5</td>
</tr>
<tr>
<td>Wynnum</td>
<td>Spring</td>
<td>1.8</td>
<td>13.5</td>
<td>4.9</td>
<td>5.3</td>
</tr>
<tr>
<td>Brisbane CBD</td>
<td></td>
<td>1.2</td>
<td>6.9</td>
<td>3.0</td>
<td>3.2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Location</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Wynnum</td>
<td>All data</td>
<td>1.2</td>
<td>17.8</td>
<td>5.3</td>
<td>5.7</td>
</tr>
<tr>
<td>Brisbane CBD</td>
<td></td>
<td>0.8</td>
<td>7.7</td>
<td>2.8</td>
<td>3.1</td>
</tr>
</tbody>
</table>

CBD, central business district

The data indicate consistently higher formaldehyde concentrations at the Wynnum monitoring site than in the central business district (CBD) of Brisbane. The Wynnum site is situated in a residential area adjacent to a petroleum refinery. The predominant source of formaldehyde in the CBD is motor vehicle emissions.

The data in Table 13.4 show that formaldehyde concentrations are highest in winter. The higher pollution levels in winter are a feature peculiar to Brisbane, owing to its geographical position. The city is surrounded by mountain ranges on
three sides. In winter, pollutants are trapped by the mountains due to a predominance of light winds, whereas in summer, strong north-easterly winds carry accumulated pollutants away to the south and southeast (AATSE, 1997).

Table 13.5 provides measured concentrations of formaldehyde from sites located in urban, rural and industrial areas in eastern Australia. The measurements reflect both seasonal and diurnal variations in formaldehyde concentrations in the air.

The highest average (hourly average from continuous monitoring for 1 month) concentrations of formaldehyde (18 ppb) were measured at Edwardstown (SA), where levels were almost double those observed in central Adelaide. Edwardstown is an industrial area adjacent to an urban area; hence, significant industrial sources of formaldehyde are likely to be contributing to the air levels. In central Adelaide, the major source of formaldehyde is motor vehicle emissions (EPA Victoria, 1999a). In comparison with other studies in Table 13.5, the mean concentrations in Adelaide, both city and industrial area, are higher. No information is given in the reference documents as to the reason for the higher levels detected in Adelaide.

Formaldehyde concentrations measured over a 2-hour period in the Latrobe Valley varied between sites, but were only slightly higher in the township of Traralgon than in the rural areas. The principal local sources of pollution in Traralgon are power stations, a paper mill, motor vehicles, domestic fires and burning off. Average concentrations were also higher in the afternoon than in the morning in the valley, suggesting a significant afternoon source, such as photochemical conversion of naturally produced isoprene compensating for the expected greater atmospheric mixing in the afternoon (EPA Victoria, 1999a).

Table 13.5: Measured concentrations of formaldehyde in the air at various locations in Australia (EPA Victoria 1999a & b)

<table>
<thead>
<tr>
<th>Location</th>
<th>Site</th>
<th>Sampling time</th>
<th>Mean Concentration (ppb)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Latrobe Valley</td>
<td>Traralgon town</td>
<td>2-h mean (am)</td>
<td>3.0 (0.1 - 5.3)</td>
</tr>
<tr>
<td></td>
<td>2-h mean (pm)</td>
<td></td>
<td>4.0 (1.1 - 4.4)</td>
</tr>
<tr>
<td></td>
<td>4 rural sites</td>
<td>2-h mean (am)</td>
<td>2.4 - 2.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2-h mean (pm)</td>
<td>2.6 - 3.1</td>
</tr>
<tr>
<td>Melbourne</td>
<td>Southeastern Arterial Freeway</td>
<td>May-July 1994</td>
<td>3.26 (0.4 - 6.5)</td>
</tr>
<tr>
<td>Castlereagh, NSW</td>
<td>Waste Management Centre – landfill for</td>
<td>Aug.-Sept. 1995</td>
<td>5.1 (max. 7.68)</td>
</tr>
<tr>
<td></td>
<td>industrial liquid, sludge and solid waste</td>
<td>24-h average</td>
<td></td>
</tr>
<tr>
<td>Brisbane</td>
<td>Fort Lytton National Park, near oil</td>
<td>July-Oct. 1992</td>
<td>7.5 ppb (max. 14.1)</td>
</tr>
<tr>
<td></td>
<td>refineries</td>
<td>30 min. average</td>
<td></td>
</tr>
<tr>
<td>Adelaide</td>
<td>Central Adelaide (city)</td>
<td>Nov.-Dec. 1994</td>
<td>10 (max. 20)</td>
</tr>
<tr>
<td></td>
<td>Edwardstown (industrial area)</td>
<td>hourly average</td>
<td>18 (max. 50)</td>
</tr>
<tr>
<td>Wagerup WA</td>
<td>Aluminium smelter</td>
<td>20 minutes</td>
<td>Background: 2.0 (max 5.1)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Event: 2.3 (max 7.18)</td>
</tr>
</tbody>
</table>
Formaldehyde concentrations near point sources at Castlereagh (landfill) and Fort Lytton (near an oil refinery) were higher than those measured near a Melbourne freeway, where emissions are derived largely from motor vehicles.

Air quality monitoring data collected from high traffic areas in South Australia (Agar et al. 2000 & 2001, as cited in NEPC, 2002) show overall mean levels of formaldehyde between 10 ppb (12-day mean) and 18 ppb (34-day mean) with a peak of 135 ppb (1 hour average). Data collected near an industrial source (Mitchell et al. 1994, as cited in NEPC, 2002) showed an overall mean of 20 ppb (2-month mean). Air monitoring studies in the Melbourne CBD and a major road in Malvern, Victoria, recorded values ranging between 0.4 and 7.6 ppb (EPA Victoria, 1994, as cited in NEPC, 2002).

Recently, the levels of formaldehyde and a range of other carbonyl compounds have been monitored in Wagerup Western Australia. Wagerup is an aluminium smelter located about 130 kilometres south of Perth near the rural township of Waroona. The refinery has an annual production capacity to 2.35 million tonnes. The maximum atmospheric levels of formaldehyde were 5.1 and 7.18 ppb for background and event samples (samples taken when refinery odour was present throughout sampling), respectively (DoE WA, 2004). The average values for background and event sampling were 2.0 ppb (7 samples) and 2.3 ppb (6 samples), respectively.

In general, the monitoring data suggest that, while the emissions estimates indicate diffuse sources of formaldehyde contribute the highest overall emissions on a kg/year basis, the concentrations of formaldehyde in the air will be highest close to industrial point sources, particularly those located in urban environments.

Concerns have been raised recently regarding the use of ethanol in fuels and the potential impact on formaldehyde emissions from vehicles. Orbital Engine Company undertook a vehicle testing study for the Department of the Environment and Heritage in order to assess the impact of gasoline containing 20% ethanol (by volume) on the Australian passenger vehicles. The study concluded that formaldehyde emissions were essentially unchanged in 5 new vehicles using gasoline containing 20% ethanol compared to gasoline only. The findings are similar to other studies as described in a recent review by Orbital Engine Company (2002).

Table 13.6 shows a summary of formaldehyde levels found in ambient air from a range of locations in Canada. Of the 3842 samples analysed, 32 were below the limit of quantification (LOQ) of 0.042 ppb. The maximum measured 24-hour average concentration was 22.9 ppb (IPCS, 2002).

13.1.6 Summary

The maximum likely annual average and maximum likely 24-hour average PECs of formaldehyde have been modelled for urban air (Table 13.7). These values include diffuse urban sources and the natural background level and are calculated next to a major urban freeway and near the largest urban industrial source.

In deriving the PECs, it was necessary to make a number of simplifying assumptions, particularly in the point source modelling. The results should be interpreted cautiously owing to uncertainties in both the NPI emission estimates and in the details of the source configurations used in the point source modelling.
The better quality of the modelling for near-road and urban areas means that this modelling is the most reliable. In spite of the uncertainties in deriving the PEC, the predicted values are comparable to measured data from monitoring sites.

Table 13.6: Formaldehyde levels in ambient air in Canada

<table>
<thead>
<tr>
<th>Duration of sampling</th>
<th>Concentration (ppb)</th>
<th>Type and number of sites</th>
</tr>
</thead>
<tbody>
<tr>
<td>24-h</td>
<td>&lt; LOQ to 22.9</td>
<td>8 urban sites</td>
</tr>
<tr>
<td>24-h</td>
<td>10.03</td>
<td>2 suburban sites</td>
</tr>
<tr>
<td>24-h</td>
<td>7.59</td>
<td>2 rural sites (influenced by urban/industrial activities)</td>
</tr>
<tr>
<td>24-h</td>
<td>8.23</td>
<td>4 rural sites (regionally representative)</td>
</tr>
<tr>
<td>1 month to 1 year average</td>
<td>7.3 to 0.65</td>
<td>For the above sites</td>
</tr>
<tr>
<td>24-h average measured over 3 months</td>
<td>1.4 to 3.67</td>
<td>Near a forest products plant</td>
</tr>
</tbody>
</table>

The CSIRO modelled maximum annual average of 5.5 ppb is consistent with the values of 3.1 ppb for Brisbane CBD and 5.7 ppb for Wynnum (Table 13.4). The CSIRO modelled maximum 24-hour average of 23.5 ppb is consistent with the most extensive data from Brisbane (Table 13.4) with values up to 17.8 ppb, and from Canada (Table 13.6) with values up to 22.9 ppb.

Table 13.7: Summary of PECs in air of formaldehyde at current emission rates

<table>
<thead>
<tr>
<th>Source</th>
<th>Annual average PEC</th>
<th>Maximum 24-hour average PEC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urban concentrations away from significant local sources, such as industry or large roads</td>
<td>1.6 ppb</td>
<td>13 ppb</td>
</tr>
<tr>
<td>Natural background</td>
<td>1 ppb</td>
<td>2 ppb</td>
</tr>
<tr>
<td>Edge of large urban freeway</td>
<td>0.77 ppb</td>
<td>2.3 ppb</td>
</tr>
<tr>
<td>Maximum predicted impact from a single industry (except for wood and paper industries)</td>
<td>2.1 ppb</td>
<td>17 ppb</td>
</tr>
<tr>
<td>(Maximum impact from wood and paper industries which are all located outside major urban areas)</td>
<td>(16 ppb)</td>
<td>(119 ppb)</td>
</tr>
<tr>
<td>(2 ppb*)</td>
<td>(37 ppb*)</td>
<td></td>
</tr>
<tr>
<td>Total (not including possible impact from wood and paper industries)</td>
<td>5.5 ppb</td>
<td>23.5 ppb</td>
</tr>
</tbody>
</table>

* refined estimates by EML Pty Ltd
13.2 Indoor air concentrations

The indoor air environment includes residential buildings and commercial buildings, such as schools, offices, hotels etc.

13.2.1 Residential buildings

There are many types of residential buildings in Australia. In this report, the residential buildings are defined as two major categories: conventional or established homes and mobile homes. Mobile homes include caravans/motor homes and manufactured homes, such as park cabins.

Australian studies

Several studies focussing on the formaldehyde levels in Australian homes are available and summarised in Table 13.8. Levels of formaldehyde in conventional homes range from 0.1 ppb to 109 ppb, with average levels lying between 15 ppb to 30 ppb in studies measuring from 90 minutes to 4-day average. Two recent studies reporting 7-day averages (Ayers et al., 1999; Sheppeard et al., 2002) indicated lower formaldehyde levels (< 4 ppb).

Monitoring data indicate that formaldehyde levels are higher in mobile homes than in established conventional residences (Table 13.8). Recently measured concentrations in occupied caravans ranged from 8 ppb to 175 ppb (average 29 ppb). In this study, 2 of the 60 caravans investigated exceeded 100 ppb (Dingle et al., 2000). The same study found that in unoccupied caravans, the formaldehyde levels ranged from 10 ppb to 855 ppb (average 100 ppb). Although the data is limited, concentrations appear to have decreased from the levels detected in previous surveys (McPhail, 1991; Dingle et al., 1992), which may be attributable to changes in resin technology and improved manufacturing controls for product emissions (Houghton et al., 2002). Formaldehyde concentrations in mobile homes are high because they tend to have a high content of formaldehyde emitting materials (such as subfloors, cabinets, shelves, hardwood wall panelling, laminated flooring and doors), while their sizes are relatively small, i.e. higher load factor. In the study by Dingle et al. (2000), up to 80% of the unoccupied caravans were less than 1100 m³ in size, with 70% of the caravans having only one room. Meyer & Hermanns (1985) reported the load factor of a typical mobile home was approximately 1.4 m²/m³ compared to a typical load factor for conventional homes ranging from 0.3 to 1.1 m²/m³.

Information from the Recreational Vehicle Manufacturers Association of Australia (RVMAA) indicates that approximately 18 000 caravans/motor homes are manufactured in Australia per year. RVMAA represents approximately 90% Australian caravan/motor home manufacturers. The total number of manufactured homes manufactured in Australia is estimated to be about 2000 per year. There are few imported manufactured homes or caravans (less than 1000 a year).

Overseas data

Formaldehyde levels in Australian conventional and mobile homes are consistent with those reported in other countries. Average levels in French, Canadian and Finnish conventional homes are 21 ppb (Gonzalez-Flesca et al., 1999), 30 ppb (IPCS, 2002; Guggisberg et al., 2003), and 33 ppb (Jurvelin et al., 2001),
respectively. Recent papers showed that average indoor levels of formaldehyde are 6.9 ppb in Sweden (27 urban dwellings) (Sakai, 2004); 14.5 ppb in 123 residential homes across 6 cities in Hungary (Erdei, 2004); and 28.6 ppb in 61 residential homes in Paris, France (Clarisse et al., 2003). In the US, formaldehyde levels range from 45 ppb to 140 ppb in conventional homes and 90 ppb to 460 ppb in mobile homes, depending on location (Godish, 1992). However, recent data in US showed levels of formaldehyde in new mobile homes ranging from 21 ppb to 73 ppb (Hodgson et al., 2000; Hodgson et al., 2002; Sherman, 2004) and new conventional homes ranging from 13 ppb to 52 ppb (Hodgson, 2000). In UK, the average indoor formaldehyde levels are 24 ppb (summer) and 22 ppb (winter) found in 37 new homes (IEH, 2004). The markedly lower formaldehyde levels in occupied caravans compared to unoccupied caravans (29 ppb vs. 100 ppb) observed in the Dingle et al. study (2000), were also seen in a Danish twin apartment study (35 ppb vs. 154 ppb; Wolkoff et al., 1991).

Residential levels of formaldehyde can vary significantly by region and/or with climate conditions, although this is not obvious in the limited Australian studies. For example, low formaldehyde levels (mean 14 ppb) have been reported in Sweden conventional homes (Norback, 1995), while Lemus et al. (1998) found that more than half the homes monitored in South Louisiana USA had levels > 100 ppb. Also, mean levels as low as 7 ppb were measured in Denmark and Greece (cited in Dingle & Franklin, 2002) while these can be up to 60 ppb in Germany (Seifert et al., 2000).

**Sources of indoor formaldehyde**

Indoor sources of formaldehyde have been studied in Australia (Brown, 1997; EA, 2001; CASANZ, 2002; Houghton et al., 2002). Major sources of formaldehyde are pressed wood products (such as particleboard and plywood that are used in building construction and furnishing materials), cooking and heating appliances (such as gas stoves, fuel burning appliances and unflued gas heaters) and tobacco smoke. Other indoor sources include permanent press fabrics, paper products, and various home and personal care products (such as household cleaners, disinfectants, fabric softeners, and cosmetics). However, the off-gassing or release of formaldehyde during use of these products is usually intermittent and unlikely to contribute significantly to the indoor formaldehyde levels.

Pressed wood products that are bonded with formaldehyde based resins have been recognised as emitters of formaldehyde (Kelly, 1999; Jiang, 2002). In Australia, the pressed wood products typically used are plywood (used for panelling, furniture and other products), particleboard (used for shelving, countertops, floor underlayment, some laminated flooring, furniture) and MDF (used for cabinets, furniture, doors and some laminated flooring). Building materials and furnishings generally release formaldehyde continuously at low levels while sources relating to activities carried out in the home release formaldehyde intermittently. Thus, pressed wood products are likely to be the major source of formaldehyde in homes where large quantities are installed, especially as seen in mobile homes (McPhail, 1991; Dingle et al., 2000). Similar findings are reported in overseas studies, such as Hodgson et al. (2002).

Australian mobile home manufacturers confirmed the use of pressed wood products in mobile homes. The majority of Australian made caravans use thin interior plywood for internal linings, such as ceilings and walls, and thick
plywood for flooring. Particleboard, MDF and plywood are also used in mobile homes.

It is believed that the majority of the plywood used in manufacturing mobile homes in Australia is imported. A market-leading supplier of imported plywood confirmed that exterior plywood is used for manufacturing furniture and cupboards and interior plywood is used for wall and ceiling linings. Australian made plywood products are mainly used for flooring. ABS statistics for the 2003-2004 financial year indicate that the total importation volume of thin interior bonded overlaid wall panelling, commonly used in mobile homes, was 5481 m$^3$.

With regard to emissions from combustion appliances, the amount of formaldehyde generated will depend on the type of appliance (e.g. space heaters, ranges, ovens, stoves, furnaces, and fireplaces), how well the appliance is installed, maintained, and vented, and the kind of fuel it uses (e.g. natural gas, LPG, kerosene, oil, coal and wood). For example, a study by the Australian Government Department of the Environment and Heritage (Environment Australia, 2002) on emissions from domestic solid fuel burning appliances indicated that the emission factors for formaldehyde vary among different fuel types (eucalypt, softwood and manufactured wood) and the average is 2.4 g/kg dry fuel mass. Formaldehyde emissions from unflued gas heaters in a chamber study ranged from $< 10$ µg/m$^3$ to 2100 µg/m$^3$ (Brown et al., 2004). Based on the available monitoring studies, emissions from combustion sources are likely to be a minor contributor to indoor formaldehyde levels (Garrett et al., 1997; Dingle & Franklin, 2002), although Garrett et al. (1997) and Sheppeard et al. (2002) did note that the highest recorded formaldehyde level in their studies was associated with an unvented or unflued gas heater.

Unflued gas heaters are recognised as primary residential heaters in Australia, and the Australian Government Department of the Environment and Heritage has recently undertaken a domestic setting assessment of their emissions, including formaldehyde. One of the aims of the study was to gather data on the concentrations of indoor air pollutants in homes attributable to unflued gas appliances (Natural Heritage Trust, 2004). Samples were collected in 6 houses in NSW and 6 houses in VIC where no new particleboard or furnishings had been fitted, and where both new and old unflued gas heaters were installed. Two samples were taken in each house: 24-hour period and during heater operating period (normally 3 hours). The average levels of formaldehyde were 32 µg/m$^3$ (30 ppb) (24-h average) and 84 µg/m$^3$ (70 ppb) during heater operation. The study found that concentrations exceeded 100 µg/m$^3$ (80 ppb) on three sampling occasions and these were recorded during heater operating periods.

Another potential source of indoor formaldehyde is tobacco smoke. Some studies indicate that tobacco smoke does not appear to increase formaldehyde levels significantly in indoor environments (Cumming, 1991; IPCS, 2002). Australian studies have reported lower levels in houses with smokers compared to houses without smokers (Garrett et al., 1997; Dingle & Franklin, 2002). The cause was not investigated, but the authors suggest that ventilation in smokers’ houses could be enhanced by frequently opening doors and windows when smoking indoors (Dingle & Franklin, 2002).

High indoor formaldehyde levels have also been associated with use of urea formaldehyde foam insulation (UFFI). However, this product is rarely used in
Australia today. In the early 1980s, 72,000 Australian dwellings installed UFFI as an energy conservation measure in their walls or ceilings. By 1987 this practice had significantly declined (Brown, 1987).

In addition, new carpets and newly painted surfaces may also contribute to indoor formaldehyde levels, although their contribution has not been adequately investigated (Wieslander, 1997; Brown, 1998; Brown, 2001; Rumchev et al., 2002).

**Factors affecting indoor formaldehyde levels**

It has been observed in several studies that the age of a building is a predictor of indoor formaldehyde concentrations (Table 13.8). Studies have shown that formaldehyde levels decreased exponentially with increased age of the home (Yu et al., 1999a; Brown, 2002), and higher values consistently occurred in homes of less than 10 to 20 years old (Godish, 1995; Garrett et al., 1997; Sheppeard et al., 2002; Dingle & Franklin, 2002). This is proposed to be due to the decrease in formaldehyde release from sources, such as pressed wood products with age (Brown, 1999). Levels of emissions in existing houses, therefore, have the potential to become elevated after renovation, particularly with use of high formaldehyde emitting materials.

Building ventilation is another important factor that affects indoor formaldehyde levels. Limited data from Australian buildings indicate that ventilation rates have become lower in residential buildings constructed in recent years due to energy conservation measures, particularly in homes built since the 1980s (Brown, 1997). Homes constructed without vents in the walls have been reported to experience a significantly higher level of formaldehyde than those with fixed wall vents (4.18 ppb vs. 2.87 ppb, Sheppeard et al., 2002).

Ventilation in homes can also be affected by habitual activities, including opening of windows and doors. Sheppeard et al. (2002) reported a significant difference in formaldehyde levels between homes with different habits of opening widows when using a heater (2.18 ppb “usually opening” vs. 2.74 ppb “sometimes opening” vs. 4.39 ppb “never opening”). Similarly, McPhail (1991) found average levels of 214 ppb in new caravans when opened (ventilated) and 705 ppb when closed (restricted ventilation). The significant difference in concentrations between occupied caravans and unoccupied caravans (29 ppb vs. 100 ppb) may also be a result of increased ventilation associated with occupant activities (Dingle et al., 2000). The authors of this study also explained their findings of lower concentrations in summer compared to winter (29 ppb vs. 36 ppb) by the increased ventilation due to more window, door, and vent openings in summer.

In conventional homes, it has been postulated that lower air exchange rates in bedrooms compared to the rest of the home may contribute to slightly elevated levels of formaldehyde in bed rooms, as reported by Garrett et al. (1997), Brown (2002); Dingle & Franklin (2002), and Rumchev et al. (2002). In testing the performance of passive samplers, Gillet et al. (2000) also described differences between rooms in six homes, with the highest levels recorded in kitchens. However, a causal link was not discussed and the study utilised only a small number of samples.

In addition to ventilation patterns, increased temperature and humidity may influence the amount of indoor formaldehyde by catalysing the hydrolysis of the
N-methylol groups, and to a lesser extent, the methylene ether linkages in the urea formaldehyde resin which further contribute to the release of formaldehyde from pressed wood products (Yu & Crump, 1999a). Consistent with this, a significant positive relationship between formaldehyde levels and indoor temperature has been established in a number of building surveys (Table 13.8), although this seasonal effect may be confounded by reduced ventilation rates during winter. Diurnal variations in formaldehyde may also be explained by changes in indoor temperature and humidity. Godish (1992) reported a doubling in formaldehyde concentration for every 5-6°C rise in temperature and an increase of approximately 1% in concentration for every 1% rise in relative humidity.

Overall, indoor concentrations of formaldehyde are the result of the interaction of many factors. These include emission sources, the age and use patterns of these sources, the load factor of the building, temperature, humidity, and ventilation rates and patterns. Pressed wood products appear to be the highest formaldehyde emitting sources. Consequently, the worst-case scenario for indoor formaldehyde levels could be created by minimum ventilation, maximum temperature, maximum humidity, and high source loadings.

13.2.2 Non-residential buildings

Non-residential buildings include offices, schools, hospitals, recreational or public buildings. There are two major categories: conventional or established buildings and relocatable buildings. Relocatable buildings (also called demountables) include classrooms, offices, hospital buildings and prisons.

Formaldehyde levels in non-residential buildings have received little investigation in Australia. Table 13.9 shows the monitoring data in both conventional and relocatable offices. Based on a small number of samples, the formaldehyde levels reported for conventional offices were less than 50 ppb (Gillett et al., 2000), whereas those in relocatable offices were found ranging from 420 to 830 ppb, with a mean of 710 ppb (Dingle et al., 1992; Gillett et al., 2000). These results are comparable with the data for residential buildings, including the observation of elevated levels in mobile homes.

No air monitoring data in Australian relocatable classrooms are available. Limited recent overseas data found that the average (7 hours to 5 days passive sampling) formaldehyde levels in conventional classrooms range from 15 ppb to 22 ppb and in relocatable classrooms range from 18 ppb to 34 ppb (Shendell et al. 2004). It also concluded that the main sources of formaldehyde are from interior finish materials and furniture. Information from Australian relocatable building manufacturers confirmed the use of pressed wood products, mainly plywood for wall lining and ceilings, particleboard, MDF and plywood for floor, and MDF for cupboards. Several manufacturers claimed to use products not containing formaldehyde resins, such as colorbond sandwich panel, plasterboard, cement sheet and Weather tex for ceiling or wall lining. The total number of relocatable buildings manufactured in Australia is not available, but anecdotal information suggests that the number is increasing. There are reportedly no imported relocatable buildings in Australia.
<table>
<thead>
<tr>
<th>Building type &amp; Location</th>
<th>Sample number</th>
<th>Sampling &amp; Analysis</th>
<th>Formaldehyde level (ppb)</th>
<th>Factors affecting formaldehyde levels</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conventional homes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NSW – Sydney</td>
<td>63</td>
<td>24 hr passive sampling, or CSIRO chromotropic acid method</td>
<td>28.9</td>
<td>↑: unflued gas heating and smoking, presence of new carpets, particleboard flooring or with renovations ←: heater type</td>
<td>McPhail, 1991</td>
</tr>
<tr>
<td>Sydney</td>
<td>18</td>
<td>7 day passive sampling, HPLC</td>
<td>3.8</td>
<td>1.1 – 25.5</td>
<td>Ayers et al., 1999</td>
</tr>
<tr>
<td>Sydney and 5 rural areas</td>
<td>139</td>
<td>7 day passive sampling, HPLC</td>
<td>3.4</td>
<td>0.1 – 46.2</td>
<td>Sheppeard et al., 2002</td>
</tr>
<tr>
<td>VIC – Latrobe Valley (rural)</td>
<td>80</td>
<td>90 min bubbler sampling, NIOSH chromotropic acid method</td>
<td>19.7</td>
<td>6 – 73</td>
<td>Godish, 1995</td>
</tr>
<tr>
<td>Latrobe Valley (rural)</td>
<td>1133</td>
<td>4 day passive sampling, HPLC</td>
<td>15.7</td>
<td>&lt; 0.2 – 109</td>
<td>Garrett et al., 1997</td>
</tr>
<tr>
<td>WA – Perth</td>
<td>100</td>
<td>3-4 day passive sampling, HPLC</td>
<td>26.0</td>
<td>0 – 97</td>
<td>Dingle et al., 1992</td>
</tr>
</tbody>
</table>
### Table 13.8: Formaldehyde monitoring data in Australian homes (continued)

<table>
<thead>
<tr>
<th>Building type &amp; Location</th>
<th>Sample number</th>
<th>Sampling &amp; Analysis</th>
<th>Formaldehyde level (ppb)</th>
<th>Factors affecting formaldehyde levels</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perth (initial sampling)</td>
<td>185, 160</td>
<td>3 day passive sampling, HPLC</td>
<td>22.8, 21.4</td>
<td>3 – 92</td>
<td>↑: home age (&lt; 10 years), seasonal variation (summer &gt; winter), bedroom (not significant) ↓: houses with smokers (not significant.) ↔: presence of building materials, gas cookers or heaters, the type of structure (house, flat or semi-detached), the number of months the doors and windows were left open, the number of occupants</td>
</tr>
<tr>
<td>(≈6 months later)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Perth (asthma associated)</td>
<td>88, 104</td>
<td>8 hr passive sampling, HPLC</td>
<td>31.7, 20.0</td>
<td>Not reported</td>
<td>↑: temperature, seasonal variation (summer &gt; winter), presence of unflued gas heater and new carpet</td>
</tr>
<tr>
<td>(controls)</td>
<td></td>
<td>liquid chromatography</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NSW – (location not stated)</td>
<td>24</td>
<td>24 hr passive sampling, or CSIRO chromotropic acid method</td>
<td>346</td>
<td>67 – 1000</td>
<td>↑: new caravan, closed windows</td>
</tr>
<tr>
<td>WA – Perth (occupied caravan)</td>
<td>20</td>
<td>3-4 day passive sampling, HPLC</td>
<td>90</td>
<td>20 - 280</td>
<td></td>
</tr>
<tr>
<td>WA – Perth (occupied caravan)</td>
<td>60</td>
<td>3-5 day passive sampling, HPLC</td>
<td>29</td>
<td>8 – 175</td>
<td>↑: new caravan, seasonal variation (winter &gt; summer) ↔: temperature and humidity</td>
</tr>
<tr>
<td>(unoccupied caravan)</td>
<td>132</td>
<td></td>
<td>100</td>
<td>10 – 855</td>
<td></td>
</tr>
</tbody>
</table>

* Results may not be comparable due to differences in sampling and analytical methodology.

↑ significant increase;
↓ significant decrease;
↔ = no difference.

Limited data suggest that formaldehyde levels may be higher in new offices than established ones. Formaldehyde concentrations in a test chamber containing new office furniture were up to 158 ppb (4-hour average) and 192 ppb (1-day average) (Brown, 1999). An average level of 48.6 ppb was also observed in a newly constructed office with new furniture, which was higher than established offices (Gillett et al., 2000). The elevated formaldehyde levels may be attributed to the presence of pressed wood products. In addition, office materials and equipment, such as carbonless copy paper, photocopiers, laser printers, together with insulation materials and soft furnishings, can contribute to indoor levels (Brown, 1999). While emission rates have been estimated for some of these sources (Brown, 1999; Kelly, 1999), there are insufficient data for estimating total releases, which are expected to vary considerably.

Similar to the home environments, ventilation may also influence the level of formaldehyde in office buildings. For example, poor ventilation has been suggested to be associated with complaints of “sick building syndrome” (Brown, 1997).

### 13.2.3 Estimation of indoor to outdoor ratio

Table 13.10 summarises the results of studies that have measured both indoor and outdoor formaldehyde levels. These studies indicate that indoor levels can be up to 16-fold higher than outdoor levels.

---

**Table 13.9: Formaldehyde monitoring data in Australian offices**

<table>
<thead>
<tr>
<th>Building type &amp; Location</th>
<th>Sample number</th>
<th>Sampling &amp; Analysis</th>
<th>Formaldehyde (ppb) Average Range</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conventional offices</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WA – Perth</td>
<td>3</td>
<td>3-4 day passive sampling, HPLC</td>
<td>21</td>
<td>15 – 70</td>
</tr>
<tr>
<td>VIC – (location not stated) offices with new furniture</td>
<td>2</td>
<td>3 day passive sampling, HPLC</td>
<td>48.6</td>
<td>Not reported</td>
</tr>
<tr>
<td>Relocatable offices</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WA – Perth</td>
<td>12</td>
<td>3-4 day passive sampling, HPLC</td>
<td>710</td>
<td>420 – 830</td>
</tr>
</tbody>
</table>

---

*Priority Existing Chemical Assessment Report No. 28*
Table 13.10: Summary of studies comparing average concentrations of formaldehyde levels indoors (conventional homes) with corresponding outdoor levels

<table>
<thead>
<tr>
<th>Location</th>
<th>Region</th>
<th>Formaldehyde (ppb)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Latrobe Valley, Australia</td>
<td>Rural</td>
<td>15.7</td>
<td>Garrett et al., 1997</td>
</tr>
<tr>
<td>Columbus, America</td>
<td>Urban</td>
<td>8.2</td>
<td>Johnson et al., 2004</td>
</tr>
<tr>
<td>Canada (16 sites in 6 provinces)</td>
<td>Rural, urban, &amp; suburban</td>
<td>30.0</td>
<td>IPCS, 2002</td>
</tr>
<tr>
<td>Helsinki, Finland</td>
<td>Urban</td>
<td>33.3</td>
<td>Jurvelin et al., 2001</td>
</tr>
<tr>
<td>Nancy, France</td>
<td>Urban</td>
<td>20.8</td>
<td>Gonzalez-Flesca et al., 1999</td>
</tr>
</tbody>
</table>

13.3 Formaldehyde concentrations in water and soil

13.3.1 Concentrations in water

Emissions of formaldehyde to water may be expected to occur via sewage treatment facilities during production of formaldehyde and formaldehyde products and during use of consumer products containing formaldehyde. Atmospheric formaldehyde may reach surface water when washed out of the atmosphere in rain.

Concentrations in the sewer

In the NICNAS survey (Section 7.3), the majority of industries responding to the NICNAS survey reported either no emissions of formaldehyde to the sewer, or only dilute emissions resulting mainly from equipment cleaning. Formaldehyde contaminated effluent is released to sewer under licensed trade waste agreements where it subsequently undergoes treatment at the local wastewater treatment plant. Trade waste agreements generally allow concentrations of between 50-200 mg/L of formaldehyde to be disposed of via the municipal treatment plant, depending on the jurisdiction. Most emissions to the municipal sewer occur via on-site treatment facilities, with some companies indicating their effluent is analysed for formaldehyde prior to release. One formaldehyde manufacturer indicated excess formaldehyde is neutralised on-site, with < 20 mg/L going to trade waste in the last two years. Another formaldehyde manufacturing company indicated typical levels of 0.5-5 mg/L released to storm water.

As a worst-case treatment plant situation, the NPI Emissions Estimation Technique Manual for sewage and wastewater treatment (NPI, 1999b) provides typical concentrations of formaldehyde in raw sewage of 0.2 \( \mu \)g/L. The data are from a large industry-intensive city (i.e. Melbourne).
Concentrations in rain and surface water

The amount of formaldehyde reaching surface water in rain is difficult to determine, but is expected to vary from region to region, depending on air quality. More formaldehyde may be expected to reach surface waters located within polluted urban areas.

The Department of Human Services in South Australia (DHSSA, 2003) has undertaken monitoring of formaldehyde levels in water from rainwater tanks in suburban areas surrounding metal foundries. In 1998, 26 samples were collected and analysed from several suburbs including Torrensville, Underdale, Flinders Park and West Hindmarsh. Formaldehyde levels were found to range between 3 and 5.9 µg/L, which is below the level set out in the Australian Drinking Water Guidelines of 500 µg/L (NHMRC/ARMCANZ, 1996). In 2002, additional testing of rainwater found levels remained between < 3 and 6 µg/L.

Measured concentrations of formaldehyde in atmospheric water (rain, snow, fog) from various locations have been reported in the IPCS (1989) and CICAD (IPCS, 2002) reports. In rain, formaldehyde concentrations ranged from 0.44 µg/L near Mexico City to 3003 µg/L in Venezuela during vegetation burning-off season. Concentrations in Venezuela during the non-burning season averaged 321 µg/L. Other reported concentrations in rain included 174 and 77 µg/L in Germany, 142 µg/L in Ireland, and 8 µg/L in the central equatorial Pacific. In snow, concentrations between 18 and 901 µg/L were measured in California. In fog, concentrations of 480 to 17 027 µg/L were found in the Po Valley in Italy with a mean of 3904 µg/L (3.9 ppm).

The US National Research Council (NRC, 1981) estimated washout of atmospheric formaldehyde to the sea surface to be 1-6 µg/cm² sea surface per year, with washout rates over land being higher. Atkinson (1990) estimated a washout ratio (concentration in rain/concentration in air) of 73 000 at 25°C. Zafiriou et al. (1980) estimated rainout of formaldehyde from the atmosphere of 0.010 g/m²/y or about 1% of that produced from methane oxidation in a remote marine environment in the central equatorial Pacific. These data suggest that rainout would contribute relatively low levels of formaldehyde, which would be further significantly diluted in the receiving water.

There are very little data available on measured concentrations of formaldehyde in natural surface water. In Canada, formaldehyde concentrations in surface water from the North Saskatchewan River averaged 1.2 µg/L, with peak values of 9.0 µg/L. In effluent, the highest reported concentrations were 325 µg/L (1-day mean) and 240 µg/L (4-day mean) measured from one of four treatment plants reporting releases. In groundwater, concentrations ranged from below the detection limit (50 µg/L) to 690 000 µg/L at a contaminated site close to a formaldehyde production facility (IPCS, 2002).

Predicted Environmental Concentration (PEC) in water

Due to its high biodegradability and low residence time, formaldehyde is not expected to reach significant levels in water. NPI estimates indicated releases of formaldehyde to land and water of 1000 kg in 2001-2002. If we assume release of this amount into a single metropolitan sewage treatment plant at one location with a daily effluent production of 4 - 10⁸ L, the PEC in the sewer would be 1.4 µg/L. This value assumes 80% biodegradation (an average estimate, Section 8.2.2) in
the sewer and a population in the city of 2 million, each using 200 L of water per day. The PEC would be further diluted in the receiving waters. We assume 10-fold dilution in oceans (PEC = 0.14 µg/L) and no dilution in rivers. Emission levels reported for 2002-2003 were 200 times lower than in 2001-2002, this would be reflected in a 200-fold reduction of the PEC to 0.7 ng/L.

13.3.2 Concentrations in soil and sediment

In the absence of data, no meaningful PEC can be determined for soil. However, the levels of formaldehyde entering the soil are expected to be negligible. Formaldehyde emissions to soils are most likely to occur through disposal of solid wastes containing formaldehyde. In the NICNAS survey, a number of companies indicated they disposed of small amounts of solid waste containing formaldehyde into landfill. These wastes consisted mainly of solidified resin waste and sludge from on-site treatment facilities, and amounts were in the order of tens of kg. However, it is noted that the NICNAS survey covered only a small proportion of the formaldehyde industry, and hence this amount of waste may not reflect the total waste from the whole industry.
14. Public Exposure

14.1 Direct exposure

14.1.1 Cosmetic and consumer products

Aqueous formaldehyde is commonly encountered at low concentrations in cosmetics and personal care products in people’s everyday life. Because of its bactericidal and hence preservative properties, formaldehyde and its derivatives (including formaldehyde releasing products) are often added to a number of cosmetics and personal care toiletries, such as skin cleanser, moisturiser, shampoo, conditioner, shower gel, liquid hand wash, mouthwash and toothpaste (more details in Section 7.3.3). While some cosmetics, such as nail hardeners and nail polishes, may have higher levels of formaldehyde (up to 1%), the majority of other externally applied cosmetics and toiletries (either rinse-off or non rinse-off products) contain less than 0.2% free formaldehyde (see Table 7.6).

Skin contact is the principal route of direct exposure, but exposure can also occur via eyes, mucous membranes, and respiratory epithelium. Small amounts of aqueous formaldehyde are also likely to be ingested during use of oral hygiene products. Therefore, although at low concentrations, direct exposure to formaldehyde is expected to be widespread and repeated with total exposure varying greatly, depending on the formulation and product type, route of exposure, individual habits and practices (such as frequency of use, duration of use, amount of product per application and demographics of use/misuse) and accidental exposure.

Direct skin and inhalation exposure can also occur from the use of formaldehyde solvents in hobby activities (such as in DIY film processing), some paints and coatings in decorating activities, home care and cleaning products in housekeeping (such as fabric softeners, dishwashing liquids, and floor/carpet cleaners), and paper products (such as paper towels and grocery bags). The concentrations of free formaldehyde in the majority of these products are generally low (< 0.2%) and exposures are likely to be intermittent.

Because of its high reactivity with biological macromolecules and rapid metabolism, formaldehyde exposure via the skin and inhalation is unlikely to cause systemic toxicity. Thus, the main concern as a consequence of cosmetic and consumer exposure remains at site of contact. Although concentrations of formaldehyde in these products are generally low, the direct exposure via cosmetic and consumer products is expected to be widespread and repeated.

14.1.2 Smoking

Tobacco smoke is a source of formaldehyde emissions. In Australia, the ABS 2001 survey indicates that 24% of the adult population were current smokers, 26% were ex-smokers and 49% have never smoked (ABS, 2002). The average daily consumption of cigarettes among smokers (aged 16 years and over) was reported to be 17.8 in a survey conducted in Victoria in 1996-97 (Trotter et al., 1998).
Environmental tobacco smoke is a combination of sidestream smoke, which passes directly from the burning tobacco into the air, and exhaled mainstream smoke from the smoker. Formaldehyde levels in mainstream smoke were reported at 70-100 μg/cigarette, depending on tobacco type and brand (based on US EPA 1992 data; NHMRC, 1997), with higher concentrations observed in non-filter cigarettes. Concentrations in sidestream smoke are often higher than in mainstream smoke due to its lower combustion temperature (IPCS, 2002). A sidestream smoke and mainstream smoke ratio, therefore, can vary between 0.1 and ~ 50 (NHMRC, 1997).

14.2 Indirect exposure

14.2.1 Indoor air

The principal route of indirect exposure is inhalation. Newton (2001) reported that Australians, whether at home, work, school, recreation or in-vehicle, spend up to 96% of their time indoors. According to a draft Exposure Assessment Handbook by enHealth Council (2003), approximately 42% of women and 22% of men spend more than 20 hours indoors each day. Given that there are a certain number of hours required to be indoors for sleep, approximately 18% of men and 6% of women spend less than 12 hours indoors each day. For both men and women, the greatest proportion of the populations spend more than 20 hours indoors each day and the proportion increases with age, being most marked in those over 65 years of age. Total individual exposure, thus, is likely to be closely associated with the time spent indoors. Groups of people who may be exposed to indoor air for the longest periods of time include the young, the elderly, and the chronically ill. Women tending to young families also spend more time at home.

There is consistent evidence that formaldehyde levels are higher indoors than outdoors and the indoor to outdoor ratios range from 7-fold to 16-fold (see Table 13.10). Typically, the average levels of formaldehyde in the indoor air of established conventional homes and offices are about 15 ppb to 30 ppb. Higher formaldehyde levels have been recorded in mobile homes and relocatable buildings. In Australia, recent limited monitoring data showed an average formaldehyde level of 29 ppb (range 8 to 175 ppb) in occupied caravans and 100 ppb (range 10 to 855 ppb) in unoccupied caravans. No monitoring data in manufactured homes, such as park cabins, is available. It is estimated that approximately 160 000 Australians lived in mobile homes either permanently or while on holiday in 1996, with approximately 68 000 Australians living permanently in caravan parks (CASANZ, 2002). By 1999, over 250 000 caravans were registered in Australia, with the warmer climate states (such as Queensland, Western Australia and the Northern Territory) having a greater share of caravan park residents (ABS, 2000; CAZANZ, 2002). It is possible that people living in caravans in these States/Territories may be exposed to higher levels of formaldehyde, due to relatively high humidity and temperature. There are no recent Australian monitoring data for relocatable buildings including offices and classrooms. However, limited previous data showed high levels of formaldehyde in relocatable offices (range from 420 ppb to 830 ppb, with a mean of 710 ppb). In addition, new buildings or buildings with new furniture are likely to have higher formaldehyde levels as indicated by limited measured data and some studies considering age of building (Table 13.8). Therefore, people in these
buildings would also be expected to be exposed to higher levels of formaldehyde. There is no information on the total number of relocatable buildings in Australia.

14.2.2 Ambient air

In Australia, mean levels of formaldehyde in ambient air range from 2 ppb to 18 ppb, with industrial areas reporting the highest levels. The data are limited, however, the values are in general agreement with the estimated annual average value of 5.5 ppb and the maximum 24-hour average value of 23.5 ppb for Australian urban environment using modelling technique (Table 13.7). A major source of formaldehyde in ambient air is the incomplete combustion of hydrocarbon fuels, especially from domestic heating and motor vehicles (Section 8.1). Industrial emissions are the major point sources, predominantly from mining, wood and paper industry, electricity supply and chemical and material manufacturing (Section 13.1.1). Thus, public exposure via ambient air may be higher in a heavily populated area or near some industries and during rush hour commutes, although there are no extensive measurements for these areas. Forest fires and other natural combustion sources can also emit formaldehyde to the ambient air (IPSC, 2002).

14.2.3 Drinking water, food, and soil

No data are available on the concentrations of formaldehyde in Australian drinking waters. In water, formaldehyde is formed by ozonation and chlorination of naturally occurring humic substances, contamination by accidental spills, or deposition from the atmosphere (IPCS, 2002). In ozonated drinking water concentrations of up to 30 µg/L have been reported overseas (NHMRC/ARMCANZ, 1996).

Information on formaldehyde concentrations in food in Australia is not available. According to overseas data, formaldehyde occurs in small amounts in almost all common foods, ranging from 1-90 mg/kg (IPCS, 1989), and adult dietary intake is estimated at 11 mg/day with drinking water contributing less than 10% of this amount (NHMRC/ARMCANZ 1996). Accidental contamination of food may also occur through fumigation, the use of formaldehyde as a preservative, or through cooking (IPCS, 1989).

Based on its low estimated Koc, high water solubility and its susceptibility to biotic and abiotic degradation (see Section 5), formaldehyde is not expected to significantly adsorb to soil particles and sediments, and thus it is likely to be present at negligible levels in these compartments.

Therefore, public exposure to formaldehyde via drinking water, food and soil is expected to be low.
15. Occupational Exposure

15.1 Routes of exposure

An evaluation of available information on Australian use scenarios indicates that workers are potentially exposed to formaldehyde by both inhalation and skin contact. Ingestion is unlikely to be a route of exposure in the occupational environment.

Exposure to formaldehyde may result from inhalation of vapour as formaldehyde presents mainly as a gas in the occupational environment. Heating or agitation of formaldehyde products may lead to an increased generation of vapour. Another source of formaldehyde vapour at workplaces is off-gassing from formaldehyde resins that are used widely in a number of industries, predominantly, the wood panel industry. Additionally, formaldehyde can be released as a thermal degradation product during processing of some materials under heat, such as plastics.

Inhalation of aerosol droplets from accidental releases or some application modes, such as spraying or brushing, is also possible. Formaldehyde containing particles can be inhaled when paraformaldehyde or formaldehyde resin powder is being used in the workplace. Formaldehyde resins can also be attached to carriers, such as wood dust, to be inhaled.

Dermal exposure may occur from spills or splashes of formaldehyde in solutions and exposure of the skin to aerosol droplets. No information was available on the potential dermal absorption of formaldehyde fumes.

Occupational exposure to formaldehyde is discussed for manufacture, importation and transportation, formulation and repackaging, and use of formaldehyde products in the following sections. Published data for atmospheric monitoring of formaldehyde levels in workplaces were reviewed by IARC (1995), but the majority of them were conducted before 1990. A literature search for post-1990 monitoring data was conducted and the results are summarised in the following sections. Recent Australian air monitoring data at workplaces were collected during this assessment and are also summarised.

15.2 Methodology for assessing occupational exposure

Together with the use profiles described in Chapter 7, information on occupational exposure and control measures obtained from industry were evaluated.

A reasonable amount of air monitoring data was received during the assessment, especially for formaldehyde and formaldehyde resin manufacture, and use of some formaldehyde resin products (mainly wood products). Due to the limited amount of monitoring data for other use scenarios, all data are presented in Tables 15.1 to 15.9 with information on number of samples, exposure duration, sampling and analytical methods, results, and relevant comments. However, it is noted that there are limitations for some data presented, for example, the reported single sample results are likely to involve a large coefficient of variation.
Due to lack of measured exposure data in some use scenarios, such as film processing, the EASE (Estimation and Assessment of Substance Exposure) model (version 2.0 for Windows), developed by the United Kingdom Health and Safety Executive (UK HSE, 2000) was used to estimate occupational exposure. The detail on this model and modelling results are summarised in Appendix 8.

15.3 **Formaldehyde manufacture**

Formaldehyde manufacture involves a series of continuous and enclosed processes. The four manufacturers reported a total of approximately 120 employees working in processes with potential exposure to formaldehyde, such as production and maintenance, quality control, laboratory and storage. This figure includes workers in the resin manufacturing plants as workers have dual roles in formaldehyde and resin production at most sites.

The potential for exposure to formaldehyde during manufacture is limited due to the fully enclosed nature of the processes. However, workers’ exposure could result from truck loading and/or drum filling and from situations where there is a need to break open or enter the enclosed system, such as sample collecting and testing, equipment cleaning and maintenance.

During formaldehyde manufacture, samples are manually taken from formaldehyde plants or storage tanks through a tap at the bottom of the formaldehyde absorber tower/storage tank using a jar, about three to four times per shift. The storage tanks are located outside buildings at all formaldehyde manufacturing sites. Analysis of the samples is done for parameters, such as pH and presence of by-products in a quality control laboratory under a fume hood, and usually takes less than 5 minutes.

Equipment maintenance is conducted regularly during formaldehyde manufacture. It was reported that typically the reaction chamber and the absorber tower are cleaned only occasionally. The frequency of the catalyst replacement varies and ranges from once in one to three years depending on the manufacturing efficiency. Specially trained personnel conduct the maintenance tasks while the manufacturing operation is shut down. Higher formaldehyde levels may occur during maintenance tasks, particularly in confined spaces, but are generally short-term and limited to work situations that require the use of respiratory protective equipment.

Exposure during storage is limited as formaldehyde storage tanks and pipes are of stainless steel with insulation and heat tracing where required. Storage tanks are above ground, bunded and closed except for vent pipes. The potential for exposure during transfers using piping systems is likely to be low, but could be high during manual drum filling.

It was reported that local exhaust ventilation is fitted at tank sampling and drum filling points at two sites. Laboratories are equipped with fume hoods. Operators wear overalls, helmet, gloves, safety glasses and boots during normal duties at production plants. Respiratory protection equipment is available and used where exposures are likely to be high, such as reaction chamber/absorber tower cleaning (confined spaces) and dealing with major spills.
**Measured exposure data**

Recent air monitoring data (1998-2002) measured at two sites of a formaldehyde manufacturer were provided and are summarised in Table 15.1. The majority of the personal sample results (40 out of 41) were less than or equal to 0.2 ppm during 8 to 12 hours shifts. A result of 0.5 ppm (measured for about half an hour) was found during formaldehyde drum filling.

The majority of 8h TWA static samples (13/16) and short-term (15 minutes) static samples (24/28) were less than or equal to 0.2 ppm. Four out of 16 peak static samples had readings above 0.5 ppm and up to 2 ppm. There were two high peak static readings (2.67 and 6.08 ppm) measured at a formaldehyde plant bund and a storage area. No information on the interpretation of these high readings is available.

Recent overseas air monitoring data were not identified for formaldehyde manufacture. In the 1980s, the mean levels of formaldehyde in workroom air at formaldehyde manufacture plants ranged from 0.2 ppm to 0.7 ppm (IARC, 1995).

**15.4 Importation and transportation**

Information obtained from importers indicates that formalin and products containing formaldehyde are imported in a variety of packaging types and sizes depending on products. The imported products are transported mainly by road from the arrival port to warehouses. From there they are transported for resale to customers either by a normal transport company or a “Dangerous Goods” approved transport company depending on the label requirements. Some imported products are transported to customers directly. Some importers use the imported products to formulate end products on site or contract the formulation to toll manufacturers.

In the case of storage, all importers reported that the imported products are stored in accordance with state and territory Dangerous Goods requirements. No information is available on the total number of workers handling formaldehyde products during importation and transportation.

In the situation that the imported formaldehyde products are transferred unopened to the sites of use, the only potential for exposure to formaldehyde during transport and storage are accidental leakages and spills. Workers potentially exposed due to accidental release include dockworkers, road transport drivers, employees at the storage sites, and emergency workers. However, the likelihood of exposure is low.

Due to off-gassing of formaldehyde resin products, such as from furniture and pressed wood panels, the levels of formaldehyde in transport containers can build up. Consequently, workers may be exposed to high levels of formaldehyde when they enter the containers (a confined space). An incident was reported when workers opened a truck container of trestle tables imported from overseas and immediately fell sick and vomited. Air monitoring of the container (using NIOSH Method 2016) was conducted for a number of substances and found only formaldehyde levels (3.1 mg/m³) exceeded the current occupational exposure standard (Personal communication, 2004).
<table>
<thead>
<tr>
<th>Type of sampling</th>
<th>No. of samples</th>
<th>Location/activity</th>
<th>Duration</th>
<th>Test method</th>
<th>Results# (ppm)</th>
<th>Comment</th>
<th>Year of monitoring</th>
</tr>
</thead>
<tbody>
<tr>
<td>Personal</td>
<td>8</td>
<td>FA operator</td>
<td>12 h</td>
<td>3M method 3721 monitor</td>
<td>5 ≤0.1</td>
<td></td>
<td>1998-2002</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3 &gt;0.1-0.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>33</td>
<td>Maintenance staff/</td>
<td>8-12 h</td>
<td>3M method 3721 monitor</td>
<td>24 ≤0.1</td>
<td></td>
<td>1998-2002</td>
</tr>
<tr>
<td></td>
<td></td>
<td>chemists/technician/</td>
<td></td>
<td></td>
<td>8 &gt;0.1-0.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>FA truck loading staff</td>
<td></td>
<td></td>
<td>1 &gt;0.2-0.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>FA drum filling</td>
<td>29 min.</td>
<td>AMCOSH method C6.4</td>
<td>1 = 0.5</td>
<td></td>
<td>2001</td>
</tr>
<tr>
<td>Static</td>
<td>16</td>
<td>FA plant bund/storage/</td>
<td>8 h</td>
<td>Interscan machine</td>
<td>11 ≤0.1</td>
<td></td>
<td>2001-2002</td>
</tr>
<tr>
<td></td>
<td></td>
<td>truck loading area</td>
<td></td>
<td></td>
<td>2 &gt;0.1-0.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1 &gt;0.2-0.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2 &gt;0.3-0.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>28</td>
<td>FA drum filling point/</td>
<td>15 min.</td>
<td>Interscan machine</td>
<td>18 ≤0.1</td>
<td></td>
<td>2001-2002</td>
</tr>
<tr>
<td></td>
<td></td>
<td>sample point/storage area</td>
<td></td>
<td></td>
<td>6 &gt;0.1-0.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2 &gt;0.3-0.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2 &gt;0.5-2</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>16</td>
<td>FA plant bund/storage/</td>
<td>Peak</td>
<td>Interscan machine</td>
<td>3 ≤0.1</td>
<td></td>
<td>2001-2002</td>
</tr>
<tr>
<td></td>
<td></td>
<td>truck loading area</td>
<td></td>
<td></td>
<td>1 &gt;0.1-0.2</td>
<td>a reading of 2.67 ppm</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1 &gt;0.2-0.3</td>
<td>at a plant bund and</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5 &gt;0.3-0.5</td>
<td>6.08 ppm at a</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>4 &gt;0.5-2</td>
<td>formaldehyde storage</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>area were found</td>
<td></td>
</tr>
</tbody>
</table>

FA, formaldehyde; AMCOSH, Advice Measurement and Control in Occupational Safety and Health (detection limit 0.06 ug).
# The results are presented as the number of samples in a series of result bands.
15.5 Formulation and repackaging

No information was available on the total number of workers handling formaldehyde during formulation and repackaging. At the majority of workplaces surveyed, one to three operators are involved in each stage of the batch formulation process (weighing, loading, mixing and equipment cleaning). The number of workers involved in the decanting process varies depending on batch sizes. Usually one to two operators are involved in repackaging.

15.5.1 Resin manufacture

Eleven formaldehyde resin manufacturers provided information, however, it is likely that there are more in Australia.

The likelihood of exposure is low for most workers during resin manufacture as the reactions involving formaldehyde occur in enclosed systems. Also the majority of production sites have enclosed systems for transferring formalin into the reactor and resin decanting. However, operators could be exposed to formaldehyde during abnormal operations, such as mechanical failure of hoses or seals and failure to ensure all hatches and chutes are closed. Exposure could also occur during the following activities or events: sample collection and testing, truck loading and unloading, filling of drums, equipment cleaning and maintenance, opening of tanks and equipment, and spills. However, these activities either take a short period to undertake (such as sampling), or are infrequent or accidental. In addition, workers are required to wear safety glasses, gloves, overalls and safety footwear when handling formaldehyde or formaldehyde resins at all sites. Full-face air supplied respirators or breathing masks are worn during truck loading at two companies. Local exhaust ventilation and general ventilation are used for loading and packaging areas at the majority of workplaces.

At large resin manufacturing sites, typically 10 to 20 samples per shift are taken manually from sample ports of the reactors and tested for viscosity and pH. Sampling takes less than 5 minutes. Small amounts of resin (as samples) are also made in the laboratory approximately 2 times per shift and take 4 to 5 hours to test each time. All laboratories have fume hoods. Local exhaust ventilation is also available in a laboratory which houses experimental resin reactors.

Equipment cleaning and maintenance are conducted regularly during formaldehyde resin manufacture. The frequency of the reaction tower cleaning varies from site to site and ranges from once every 6 months to 3 years. The tasks are conducted by specially trained personnel while the manufacturing operation is shut down. The required personal protective equipment (PPE), such as air-supplied breathing apparatus for working in confined spaces, is used.

The potential for inhalation and skin exposure of workers during resin manufacture is likely to be higher at worksites where manual charging of formalin from drums or paraformaldehyde prills from sealed bags, and manual drum filling of resins are undertaken.
Measured exposure data

Recent personal and static air monitoring data (1998-2003) during formaldehyde resin manufacture was provided by a number of companies and is summarised in Table 15.2a and Table 15.2b, respectively.

Almost all long-term personal monitoring results (except at site number 7 in Table 15.2a) were δ 0.5 ppm, with the majority of them (88 out of 95) δ 0.2 ppm. One high reading (1.96 ppm) was measured when formaldehyde vapours were released during flushing of the formaldehyde pump and opening of tanker hatches by a driver. The data from site number 7, which has the most number of samples (176), showed a similar pattern, with 76 out of 89 showing readings δ0.5ppm, in which 63 readings were δ0.2ppm. The company claimed that exposures in the plant operating environment and laboratory were typically less than 0.1 ppm for the duration of 12 hour (operators) and 8 hour (laboratory staff) shifts. Similarly, most of the short-term measurement results were less than 0.5 ppm (66 out of 87).

The long-term static data showed that 46 out of 50 sample results are δ 0.2 ppm (Table 15.2b). The majority of short-term static measurements were also δ 0.2 ppm, with 11 out of 74 samples in the result band of > 0.5 ppm to 2 ppm. Weekly static monitoring (100 samples in total between year 2001 and April 2003) was undertaken at site number 9 and most of readings were < 0.2 ppm. The company reported that the highest routine exposures are 0.5 ppm to 1 ppm during sampling and testing.

Information from industry indicates that where high exposures are recorded, an investigation into likely causes is initiated and corrective actions are carried out. For example, the results measured near the scrubber extraction at site number 8 (with limited details on methodology) were high (4 - 4.3 ppm), but were reduced to lower than 2 ppm after the ventilation system was improved (Table 15.2b).

No recent overseas air monitoring data on formaldehyde levels during resin manufacture were identified. Earlier data reported mean concentrations of formaldehyde during resin manufacture vary from < 1 ppm to 14 ppm, with majority < 3 ppm (IARC, 1995). Therefore, the current levels of formaldehyde in Australia are much lower compared with overseas data of two decades ago.

15.5.2 Formulation of formaldehyde products other than resins

Forty-eight formulators of formaldehyde products other than resins provided information, however, it is likely that there are more formulators in Australia.

There are a variety of formulation processes, ranging from open tanks to enclosed systems, using formalin or products containing formaldehyde as raw materials (see Table 7.4). Operators are likely to be exposed by skin contact during manual charging of mixing vessels, mixing and inspection, filling of product containers and equipment cleaning. There is also a potential for inhalation exposure, especially during charging of formalin into open vessels, heated blending processes and high-speed mechanical stirring in open tanks. Sampling is usually conducted at the end of mixing and samples are taken through a sampling tap at the bottom of the mixing vessel. Equipment is cleaned between different products by hosing with pressured water or using cleaning solvents.
### Table 15.2a: Summary of personal monitoring data during formaldehyde resin manufacture

<table>
<thead>
<tr>
<th>Site*</th>
<th>Activity</th>
<th>No. of samples</th>
<th>Duration</th>
<th>Test method</th>
<th>Results# (ppm)</th>
<th>Comment</th>
<th>Year</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>resin operators/QC chemist</td>
<td>32</td>
<td>12 h</td>
<td>3M method 3721 monitor</td>
<td>25 &gt;0.1</td>
<td></td>
<td>1998-2001</td>
</tr>
<tr>
<td></td>
<td>maintenance/R&amp;D chemist/laboratory staff/truck loading</td>
<td>25</td>
<td>8 h</td>
<td>3M method 3721 monitor</td>
<td>17 &gt;0.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>worker</td>
<td></td>
<td></td>
<td></td>
<td>8 &gt;0.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>kettle charging/ changing bag &amp; filters/drum filling</td>
<td>14</td>
<td>19-210 min</td>
<td>Pre-calibrated Dupont Sampling pump</td>
<td>11 &gt;0.1</td>
<td></td>
<td>1998-2001</td>
</tr>
<tr>
<td></td>
<td>kettle charging</td>
<td>1</td>
<td>1 h</td>
<td>Method MA-1159</td>
<td>0.07</td>
<td>Method MA-1159 is an internal method by Leeder Consulting Pty Ltd.</td>
<td>2003</td>
</tr>
<tr>
<td>3</td>
<td>NR</td>
<td>1</td>
<td>6 h</td>
<td>AMCOSH 50056</td>
<td>&lt;0.01</td>
<td></td>
<td>2001</td>
</tr>
<tr>
<td>4</td>
<td>plant operator/laboratory staff/maintenance workers</td>
<td>6</td>
<td>7-12 h</td>
<td>passive Dosi-tube</td>
<td>&lt;0.1</td>
<td></td>
<td>2001</td>
</tr>
<tr>
<td>5</td>
<td>tankers unloading/kettle drumming/drop solid resin to</td>
<td>12</td>
<td>35-700 min</td>
<td>NIOSH 2016</td>
<td>6 &gt;0.1</td>
<td>A reading of 1.96 ppm when formaldehyde vapours released during flushing of the formaldehyde pump and opening of tankers hatches by the driver.</td>
<td>1999-2001</td>
</tr>
<tr>
<td></td>
<td>cooling floor/ kettle operation</td>
<td></td>
<td></td>
<td></td>
<td>3 &gt;0.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3 &gt;0.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>plant operators/technical personnel/maintenance workers</td>
<td>89</td>
<td>8-12 h</td>
<td>passive dosimeter badges analysed by LC</td>
<td>46 &gt;0.1</td>
<td>3 readings in the band &gt;0.5-2ppm were due to plant breakdown. 2 readings where workers worn full-face canister.</td>
<td>2000 - 2002</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>17 &gt;0.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>7 &gt;0.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>6 &gt;0.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>13 &gt;0.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td></td>
<td></td>
<td></td>
<td>combination of passive dosimeter badges, Drager tubes and a direct read, hand held electronic formaldehyde device</td>
<td>33 &gt;0.1</td>
<td>One reading of 3.6 ppm was due to opening formaldehyde storage oven. For other 4 readings of &gt;2ppm workers worn respirators.</td>
<td>1999-2002</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>11 &gt;0.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5 &gt;0.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5 &gt;0.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>16 &gt;0.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5 &gt;2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Mixing/pack off</td>
<td>5</td>
<td>300-400 min</td>
<td>Dosi tube/impinger</td>
<td>3 &gt;0.1</td>
<td>One reading of 2 ppm was due to technical activity. No details for other readings &gt;0.5 to 2ppm.</td>
<td>1999-2002</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2 &gt;0.2</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

NR, not reported; LC, liquid chromatography; AMCOSH, Advice Measurement and Control in Occupational Safety and Health; QC, quality control; R&D, research and development; NIOSH, National Institute for Occupational Safety and Health; TWA, time-weighted average.

*Site 4 does not have personal monitoring data. # The results are presented as the number of samples in a series of result bands.
Table 15.2b: Summary of static monitoring data during formaldehyde resin manufacture

<table>
<thead>
<tr>
<th>Site</th>
<th>Location</th>
<th>No. of samples</th>
<th>Duration</th>
<th>Test method</th>
<th>Results# (ppm)</th>
<th>Year of</th>
<th>Year of</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Laboratory/workshop/control room/resin kettle</td>
<td>14</td>
<td>8 h</td>
<td>Interscan machine</td>
<td>8 &gt;0.1</td>
<td>2001-2002</td>
<td></td>
</tr>
<tr>
<td></td>
<td>resin load out/drum filling/laboratory/office/car park/sampling</td>
<td>53</td>
<td>15 min.</td>
<td>Interscan machine</td>
<td>43 &gt;0.1</td>
<td>2001-2002</td>
<td></td>
</tr>
<tr>
<td></td>
<td>NR</td>
<td>14</td>
<td>Peak</td>
<td>Interscan machine</td>
<td>4 &gt;0.1</td>
<td>2001-2002</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>alongside charging chute/filter/laboratory/ Mezzanine level/ Kuno unit</td>
<td>16</td>
<td>19-360 min.</td>
<td>Pre-calibrated Dupont Sampling pump</td>
<td>13 &gt;0.1</td>
<td>1998-2001</td>
<td></td>
</tr>
<tr>
<td></td>
<td>kettle charging</td>
<td>1</td>
<td>1 h</td>
<td>Method MA-1159</td>
<td>0.03</td>
<td>2003</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>resin operator - filter wash</td>
<td>1</td>
<td>15 min.</td>
<td>AMCOSH method C6.4</td>
<td>0.3</td>
<td>NR</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>process building (central column)/ Mezzanine floor/near drums</td>
<td>4</td>
<td>24 h</td>
<td>NR</td>
<td>3 &gt;0.1</td>
<td>2001</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>kettle charging/kettle drumming/drop solid resin to cooling floor/near kettle/laboratory (next to fume hood)</td>
<td>11</td>
<td>48-250 min.</td>
<td>NIOSH 2016</td>
<td>10 &gt;0.1</td>
<td>1999-2001</td>
<td></td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>5 min.</td>
<td>Kitigawa detector</td>
<td>2 &gt;0.3</td>
<td>1999-2001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>scrubber extraction</td>
<td>NR</td>
<td>NR</td>
<td>NIOSH 3500 &amp; AS2365.6 (1995)</td>
<td>4-4.3</td>
<td>1999</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>control room/laboratory/top of reactors/scrubbers/tank farm/ sample pots/reactor room/truck loading station/Mezzanine level</td>
<td>5</td>
<td>12 h</td>
<td>NIOSH 2541</td>
<td>4 &gt;0.1</td>
<td>2002</td>
<td></td>
</tr>
<tr>
<td></td>
<td>100 (weekly)</td>
<td>&lt;5 min.</td>
<td>Dragger colorimetric tubes (limit of detection 0.2 ppm)</td>
<td>&lt;0.2 (majority)</td>
<td>2001-2003</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

NR, not reported; AMCOSH, Advice Measurement and Control in Occupational Safety and Health; NIOSH, National Institute for Occupational Safety and Health; AS, Australian Standard.
*Sites 3 and 7 do not have static monitoring data. #The results are presented as the number of samples in a series of result bands.
The majority of formulators reported that workers wear safety glasses and gloves when handling formalin and products containing formaldehyde. At some workplaces workers are required to wear overalls, safety footwear and aprons. Respiratory protection equipment, such as half-face masks, full-face visors and full-face powered air respirators, are available at most of the workplaces, and are worn when exposure is likely to be high, such as manual loading and open mixing.

Exhaust ventilation above the mixing tank is used at the majority of the workplaces. Some sites have roof exhaust ventilation and industrial fans. Six formulators reported that the workplace relies on natural ventilation only and four did not provide any information on engineering controls.

Inhalation exposure of workers during formulation is likely to be high at worksites with an open mixing process and no exhaust ventilation. However, since formulation is a batch process, exposure will only occur on the days when formaldehyde products are formulated. As seen in Table 7.4, the duration of the formulation process varies between companies, but is usually an intermittent process of a few hours.

**Measured exposure data**

Limited personal and static monitoring data collected during formulation of biocide, film processing and consumer products were provided and are summarised in Table 15.3. There were few long-term personal samples, and most of those reported were < 0.2 ppm. Short-term sampling results ranging from 0.3 ppm to 2 ppm were measured during raw material weigh-up, equipment cleaning and maintenance. Static results measured at a blending platform, mixing tank opening and filling line range from 0.5 ppm to 1.5 ppm, with measurement duration of 10 minutes. One static reading of 2.24 ppm was found during bulky box tank filling.

No overseas air monitoring data on formaldehyde levels during formulation of formaldehyde products were identified.

**15.5.3 Repackaging**

Exposure of workers to formaldehyde is limited to accidental spills or leakages during repackaging as the processes are usually fully or partially enclosed, of relatively short duration and usually infrequent. Moreover, PPE (such as gloves, overalls, safety glasses or goggles) is reportedly used at five repackaging companies. Three of them have exhaust ventilation in place whereas the other two rely on general ventilation and use of industrial fans. Both skin and inhalation exposures to formaldehyde are likely during the manual repackaging of paraformaldehyde powder because formaldehyde-containing dusts can be generated during the process. However, one company reported that all activities are conducted in a booth fitted with an extraction fan system and all workers wear respirators with gas and particle cartridges, rubber gloves, goggles, hair cover and dust coat. Therefore, the potential exposure to formaldehyde during repackaging is considered low.
<table>
<thead>
<tr>
<th>Formulation</th>
<th>Type of sampling</th>
<th>No. of samples</th>
<th>Location/activity</th>
<th>Duration</th>
<th>Test method</th>
<th>Results (ppm)</th>
<th>Comment</th>
<th>Year of monitoring</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biocide manufacture</td>
<td>personal</td>
<td>NR</td>
<td>filter/packaging of products</td>
<td>6 h</td>
<td>pumps with carbon active</td>
<td>0.003</td>
<td></td>
<td>2001</td>
</tr>
<tr>
<td></td>
<td></td>
<td>NR</td>
<td>raw material weigh-up</td>
<td>18 min.</td>
<td>passive samplers</td>
<td>0.28</td>
<td></td>
<td>2002</td>
</tr>
<tr>
<td>Film processing products</td>
<td>personal</td>
<td>12</td>
<td>Line setting/packaging</td>
<td>47-486 min.</td>
<td>AMCOSH method C6.4</td>
<td>6 &gt;0.1</td>
<td>Higher readings (&gt;0.3 ppm) were measured during packaging/filling</td>
<td>1998</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>/mixing/filling</td>
<td></td>
<td></td>
<td>4 &gt;0.1-0.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1 &gt;0.3-0.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2 &gt;0.5-2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Consumer product</td>
<td>personal</td>
<td>1</td>
<td>operator</td>
<td>3.5 h</td>
<td>Pre-calibrated SKM sampling</td>
<td>0.08 (8h TWA)</td>
<td></td>
<td>2001</td>
</tr>
<tr>
<td>formulation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>pump and HPLC</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>static</td>
<td>2</td>
<td>pumping area/Mezzanine level</td>
<td>3.55 h</td>
<td>NR</td>
<td>0.01</td>
<td></td>
<td>2001</td>
</tr>
<tr>
<td>Consumer product</td>
<td>static</td>
<td>2</td>
<td>blending platform</td>
<td>10 min.</td>
<td>Kitigawa atmosphere monitor</td>
<td>0.5, 0.53</td>
<td></td>
<td>2001-2002</td>
</tr>
<tr>
<td>formulation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>mixing tank opening</td>
<td>10 min.</td>
<td></td>
<td>1.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>filling line</td>
<td>10 min.</td>
<td></td>
<td>0, 0.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>bulky box tank filling</td>
<td>10 min.</td>
<td></td>
<td>2.24</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

NR, not reported; HPLC, high performance liquid chromatography; AMCOSH, Advice Measurement and Control in Occupational Safety and Health; TWA, time-weighted average.
# The results are presented as the number of samples in a series of result bands, or individual results if only one or two samples are available.
15.6 End uses of formaldehyde products

15.6.1 Formaldehyde resins

Information from industry submissions indicates that most of the formaldehyde resins used in Australia contain < 0.2% free formaldehyde. However, some contain up to 13% free formaldehyde. In addition, formaldehyde gas can be released if resin is heated to temperatures where it decomposes or when in contact with high humidity levels.

Information from the Plywood Association of Australia (PAA) indicates that approximately 500 operative staff work in the processes of glue mixing, glue spreading, panel lay-up and pressing. These workers are potentially exposed to formaldehyde-containing adhesives used in plywood mills. No information is available on the total number of workers handling formaldehyde resins in the manufacture of particleboard and MDF. The concentrations of free formaldehyde in the resins used in particleboard and MDF manufacture range from < 0.2% to 0.5% and are up to 5% in the plywood industry.

During particleboard and MDF manufacture, operators are likely to be exposed to formaldehyde from the hot press procedure and onwards (see Figure 7.5), as the formaldehyde resins are handled in enclosed systems in the processes before the hot press. However, potential exposure could occur in situations where there is a need to break open or enter the enclosed system, such as equipment cleaning and maintenance. Typically, filters in the resin storage tanks are cleaned manually 1 to 2 times per shift and the task takes about 10 minutes each time. The storage tanks are cleaned yearly for melamine urea formaldehyde resin and once in 3 to 4 years for urea formaldehyde resin.

These pressed wood products are used industrially in furniture and cabinet making and also have do-it-yourself (DIY) applications. The atmosphere created by machining the products, such as fitting, sawing/cutting and sanding, contains a mixture of wood dusts, free formaldehyde, dust particles onto which formaldehyde is absorbed and, potentially, the resin binder itself and derivatives. Therefore, workers are likely to inhale airborne formaldehyde.

The use process of formaldehyde resins in other industries (Section 7.3.3) involves basically dilution and/or mixing with other ingredients and then a drying process to make the resin set. Some uses, such as paper treating and coating, also involve hot pressing after drying. The methods of application of the resin product vary largely and include dipping/bathing (paper treating and coating, textile finishing and leather tanning), brushing (composite construction), spraying (fibreglass industry and anti-graffiti wall sealer manufacture), use of mop/bristle rollers, and use of mechanical equipment, such as print screen and dyeing machines in textile printing and dyeing. The duration of use varies from a couple of minutes per day to continuous use. Information on the total number of workers in these industries is not available.

Information from industry submissions and the NICNAS survey indicates that half the number of companies using formaldehyde resins or products containing formaldehyde resins have local or roof exhaust ventilation in place. Others rely on general ventilation. Basic PPE (gloves, safety glasses and clothing) is worn at
most sites during handling of formaldehyde resin products. Some reported use of 
respiratory protection during glue mixing. One company using formaldehyde 
resin in making hardboards reported that it does not use any PPE.

In summary, workers’ exposure to formaldehyde during use of the resin is likely 
to be higher if the resin contains high levels of free formaldehyde, especially by 
inhalation during the hot press process. Skin exposure is also possible during use 
of the resin in a variety of industries due to the manual handling involved and the 
high viscosity of some adhesive resins.

**Measured exposure data**

Monitoring data (1999-2004) at pressed wood manufacturing sites were provided 
by PAA and AWPA, and are summarised in Table 15.4.

In plywood mills, most long-term personal monitoring results (61/71) were < 0.3 
ppm. No short-term personal monitoring data was provided. Most of the long-
term static data (31/34) showed levels < 0.3 ppm. It appears that formaldehyde 
levels are higher at mills using urea formaldehyde resin. In particleboard and 
MDF mills, limited data showed that most of long-term samples were < 0.3 ppm 
(5/8 for personal samples, 12/17 for static samples). No short-term data was 
available.

AWPA has an air monitoring program in place for wood working companies in 
VIC, NSW and QLD which use the wood panel products manufactured by its 
members. The data in 2001-2003 were provided and are summarised in Table 
15.4. The majority of personal samples (154 out of 159) showed formaldehyde 
levels < 0.2 ppm in workers’ breathing zones.

A limited number of companies using formaldehyde resins in non-wood working 
industries provided air monitoring data and the details are also shown in Table 
15.4. Long-term personal sampling during core making indicated that most 
samples (13/17) were >0.3 ppm. It was noted that the formaldehyde levels 
reduced significantly after process modification. Static levels of up to 0.5 ppm 
were measured in coater rooms using paints containing formaldehyde, whereas 16 
static samples in the firelighter manufacturing site were all < 0.5 ppm (the limit of 
detection).

Recent overseas air monitoring data (Makinen & Kangas, 1999; Chung et al., 
2000; Gillett et al., 2000; Poźniak et al. 2001; Westberg et al., 2001; Fransman et 
al. 2003) measuring formaldehyde levels during use of formaldehyde resin 
products are summarised in Table 15.5. The majority of the personal samples 
showed less than 0.2 ppm formaldehyde around workers’ breathing zone. Similar 
results were found in static samples, with only six out of 198 samples above 0.3 
ppm (maximum 0.5 ppm). These results are in agreement with the Australian 
data.

The earlier overseas data summarised by IARC (1995) reported the levels of 
formaldehyde in plywood, particleboard and paper mills, furniture factories, and 
other wood product plants, such as match mill, wooden container mill and parquet 
plant. Most of the results (mean) were below 2 ppm (ranging from 0.08 ppm to 
1.7 ppm). However, some high levels of formaldehyde (2 ppm to 7.4 ppm) were 
measured in wood and paper industries, mainly during glue preparation, hot 
pressing, sawing, and paper impregnation with formaldehyde resins.
<table>
<thead>
<tr>
<th>Use</th>
<th>Type of sampling</th>
<th>No. of samples</th>
<th>Activity/Location</th>
<th>Duration</th>
<th>Sampling method</th>
<th>Results# (ppm)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plywood mill using urea formaldehyde</td>
<td>Personal</td>
<td>17</td>
<td>All activities from veneer glue spreading, pre-lay and hot press operating.</td>
<td>6-8 h</td>
<td>NIOSH Method 2016</td>
<td>9 &lt;0.2 3 &gt;0.2-0.3 5 &gt;0.3</td>
<td>PAA (measured in 2002-2004)</td>
</tr>
<tr>
<td>Phenol formaldehyde adhesive</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plywood mill using phenol formaldehyde</td>
<td>Personal</td>
<td>54</td>
<td>All activities from veneer glue spreading, pre-lay and hot press operating.</td>
<td>6-10 h</td>
<td>NIOSH Method 2541</td>
<td>11 &lt;0.1 38 &gt;0.1-0.3 5 &gt;0.3</td>
<td>PAA (measured in 1999-2004)</td>
</tr>
<tr>
<td>Static</td>
<td></td>
<td>34</td>
<td>Hot press, spreader infeed, glue loft operating</td>
<td>8 h</td>
<td>NIOSH Method 2541</td>
<td>31 &lt;0.3 3 &gt;0.3-0.4</td>
<td></td>
</tr>
<tr>
<td>Particleboard and MDF mills</td>
<td>Personal</td>
<td>8</td>
<td>Press operators, sanders, forming station, press outfeed, laboratory technician</td>
<td>5 h</td>
<td>NIOSH Method 5700 (NIOSH, 1994)</td>
<td>2 &lt;0.1 1 &gt;0.1-0.2 2 &gt;0.2-0.3 3 &gt;0.3-0.5</td>
<td>AWPA (measured in 2004)</td>
</tr>
<tr>
<td>Static</td>
<td></td>
<td>17</td>
<td>Glue blending line, warehouse, cut to size saw, trim saw, sanders, forming station, press outfeed</td>
<td>5 h</td>
<td></td>
<td>8 &lt;0.1 2 &gt;0.1-0.2 2 &gt;0.2-0.3 3 &gt;0.3-0.5 2 &gt;0.5-2</td>
<td>AWPA (measured in 2004))</td>
</tr>
<tr>
<td>Working with particleboard and MDF</td>
<td>Personal</td>
<td>30</td>
<td>Wood cutting, routing at 7 sites (1 construction site using MDF, 1 TAFE college workshop and 5 furniture manufacturing sites)</td>
<td>5 h</td>
<td>NIOSH Method 5700 (NIOSH, 1994)</td>
<td>26 &lt;0.08 4 &gt;0.08</td>
<td>AWPA (measured in 2001)</td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>Personal</td>
<td>66</td>
<td>Wood cutting, routing at 12 furniture manufacturing sites</td>
<td>5h</td>
<td></td>
<td>64 &lt;0.06 2 = 0.14</td>
<td>AWPA (measured in 2002)</td>
</tr>
<tr>
<td></td>
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<td></td>
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</tr>
<tr>
<td></td>
<td>Personal</td>
<td>61</td>
<td>Wood cutting, routing at 14 furniture manufacturing sites</td>
<td>5h</td>
<td></td>
<td>58 &lt;0.2 5 &gt;0.2-0.5</td>
<td>AWPA (measured in 2003)</td>
</tr>
<tr>
<td>Use</td>
<td>Type of sampling</td>
<td>No. of samples</td>
<td>Activity/Location</td>
<td>Duration</td>
<td>Sampling method</td>
<td>Results# (ppm)</td>
<td>Reference</td>
</tr>
<tr>
<td>---------------------</td>
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</tr>
<tr>
<td>Workshop using</td>
<td>static</td>
<td>27</td>
<td>No details</td>
<td>Spot</td>
<td>Formaldehyde meter</td>
<td>27 &lt;0.04</td>
<td>Personal communication, 2001</td>
</tr>
<tr>
<td>particleboard</td>
<td></td>
<td></td>
<td></td>
<td>testing</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Relocatable building</td>
<td>1</td>
<td></td>
<td>Cutting and working</td>
<td>8h</td>
<td>NIOSH method 2541</td>
<td>&lt;0.03</td>
<td>Personal communication, 2001</td>
</tr>
<tr>
<td>personal manufacture</td>
<td></td>
<td></td>
<td>with MDF boards</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Core making</td>
<td>personal</td>
<td>5</td>
<td>core oven, core</td>
<td>8 h</td>
<td>AS2986</td>
<td>3 &lt;0.1</td>
<td>NICNAS survey (1999 data)</td>
</tr>
<tr>
<td></td>
<td></td>
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<td>blower, auto-pour</td>
<td></td>
<td></td>
<td>2 &gt;0.3 – 0.5</td>
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<td></td>
<td></td>
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<td>channel, mould-line</td>
<td></td>
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<td></td>
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<td>basement, melt-</td>
<td></td>
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<td>Core making</td>
<td>Personal</td>
<td>10</td>
<td>hot box process –</td>
<td>8h</td>
<td>Passive diffusion</td>
<td>1 &lt;0.2</td>
<td>Barton, 1998</td>
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<td></td>
<td></td>
<td></td>
<td>core unloading</td>
<td></td>
<td>formaldehyde vapour monitor</td>
<td>4 &gt;0.5-1</td>
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</tr>
<tr>
<td></td>
<td></td>
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<td></td>
<td>2 &gt;1-2</td>
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<td></td>
<td></td>
<td></td>
<td>core racking</td>
<td></td>
<td></td>
<td>3 &gt;2</td>
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</tr>
<tr>
<td>Personal</td>
<td>static</td>
<td>2</td>
<td>Warm box process –</td>
<td>8h</td>
<td>NIOSH 2541</td>
<td>0.59, 0.65</td>
<td>NICNAS survey (1997 data)</td>
</tr>
<tr>
<td>Using formaldehyde</td>
<td></td>
<td></td>
<td>core unloading</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>resin paints</td>
<td></td>
<td></td>
<td>8h</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Firefighter</td>
<td>static</td>
<td>16</td>
<td>throughout the</td>
<td>8 h</td>
<td>Kitigawa formaldehyde</td>
<td>&lt;0.5</td>
<td>NICNAS survey (2002 data)</td>
</tr>
<tr>
<td>manufacture</td>
<td></td>
<td></td>
<td>process</td>
<td></td>
<td>detector tubes (No. 171SB)</td>
<td>(limit of detection)</td>
<td></td>
</tr>
<tr>
<td>Quality control</td>
<td>static</td>
<td>20</td>
<td>No details</td>
<td>Spot</td>
<td>Formaldehyde meter</td>
<td>20 &lt;0.2</td>
<td>Personal</td>
</tr>
<tr>
<td>laboratory</td>
<td></td>
<td></td>
<td></td>
<td>testing</td>
<td></td>
<td></td>
<td>communication, 2003</td>
</tr>
<tr>
<td>Quality control</td>
<td>static</td>
<td>2</td>
<td>bench top with</td>
<td>6 h</td>
<td>air sampling pump</td>
<td>&lt;0.2</td>
<td>NICNAS survey (1999 data)</td>
</tr>
<tr>
<td>laboratory</td>
<td></td>
<td></td>
<td>local extraction</td>
<td></td>
<td></td>
<td>(limit of detection)</td>
<td></td>
</tr>
</tbody>
</table>

NR, not reported; NIOSH, National Institute for Occupational Safety and Health; AS, Australian Standard; MDF, medium density fibreboard; PAA, Plywood Association of Australasia; AWPA, Australian Wood Panel Association.

# The results are presented as the number of samples in a series of result bands where necessary.
<table>
<thead>
<tr>
<th>Use</th>
<th>Type of sampling</th>
<th>No. of samples</th>
<th>Activity/location</th>
<th>Duration</th>
<th>Test method</th>
<th>Results# (ppm)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Processing PF resins</td>
<td>personal</td>
<td>NR</td>
<td>production of friction linings and abrasive materials in 24 workplaces</td>
<td>NR</td>
<td>GC-MSD and HPLC</td>
<td>0.003-0.09</td>
<td>Posniak et al., 2001</td>
</tr>
<tr>
<td>Plywood mill</td>
<td>personal</td>
<td>22</td>
<td>Dryers, composes, during pressing and finishing process</td>
<td>15 min.</td>
<td>NIOSH 2016</td>
<td>0.07 (mean)</td>
<td>Fransman et al., 2003</td>
</tr>
<tr>
<td>Plywood manufacturing in a New Zealand mill</td>
<td>static</td>
<td>10</td>
<td>Dryers, composes, during pressing and finishing process</td>
<td>7.5-19 h</td>
<td>NR</td>
<td>10 &lt;0.1</td>
<td>PAA (measured in 2004)</td>
</tr>
<tr>
<td>Use of PF glue (&lt;0.4% free FA) in plywood mill</td>
<td>personal</td>
<td>49</td>
<td>Patching/machine feeding/forklift driving/scaring/assembly/hot pressing/glue preparation/finishing/carrying plywood piles areas where glue was directly used (13 sites)</td>
<td>8 h</td>
<td>air sampling pumps and HPLC</td>
<td>26 ≥0.1</td>
<td>Makinen &amp; Kangas, 1999</td>
</tr>
<tr>
<td>Machining MDF - sawing and sanding</td>
<td>static</td>
<td>48</td>
<td>Work room</td>
<td>30 min.</td>
<td>Kitagawa 710 tubes</td>
<td>0.01-0.10</td>
<td>Chung et al. 2000</td>
</tr>
<tr>
<td>Carpentry workshop</td>
<td>static</td>
<td>48</td>
<td>Working bench</td>
<td>30 min.</td>
<td>impregnated filters</td>
<td>0.02-0.14</td>
<td>Gillett et al. (2000)</td>
</tr>
<tr>
<td>Glue/paint used in graphics</td>
<td>static</td>
<td>4</td>
<td>Not indicated</td>
<td>&gt; 3 days</td>
<td>Passive sampler method and HPLC</td>
<td>0.03-0.06</td>
<td>Gillett et al. (2000)</td>
</tr>
<tr>
<td>Sand foundry and static die-casting foundry</td>
<td>personal</td>
<td>46</td>
<td>moulding/core making/pouring/shake-out/static die-casting/core knock-out</td>
<td>8 h</td>
<td>diffusive samplers and GC</td>
<td>0.007-0.12</td>
<td>Westberg et al., 2001</td>
</tr>
<tr>
<td></td>
<td>static</td>
<td>20</td>
<td>NR</td>
<td>8 h</td>
<td></td>
<td>0.007-0.12</td>
<td></td>
</tr>
</tbody>
</table>

NR, not reported; MSD, mass-selective detection; HPLC, high performance liquid chromatography; GC, gas chromatographic; FA, formaldehyde; PF, phenol formaldehyde; MDF, medium density fibreboard; NIOSH, National Institute for Occupational Safety and Health; PAA, Plywood Association of Australasia.
# The results are presented as the number of samples in a series of result bands.
In 1997, UK HSE initiated a review on the health effects of exposures arising from machining MDF. As part of the review, HSE carried out a hazard assessment and an exposure survey and researched the characteristics of MDF dust including formaldehyde. The published document (UK HSE, 1999) summarised a number of human cross-sectional studies published in 1988 to 1994 where the majority of these measured free formaldehyde levels (both static and personal sampling) at workplaces using wood panel products. The average formaldehyde level within workers’ breathing zone ranged from < 0.01 ppm to 0.4 ppm, with peak level up to 0.8 ppm.

The apparent decrease in the formaldehyde levels over the years may be attributed to the reduced level of free formaldehyde in resins and improvements in processes and control measures. Forensic/hospital mortuaries and pathology laboratories

15.6.2 Forensic/hospital mortuaries and pathology laboratories

Formalin solutions containing 4% formaldehyde are commonly used in the forensic/hospital mortuaries and pathology laboratories including histopathology and anatomical laboratories. However, solutions containing higher levels of formaldehyde (up to 32%) are also handled during dilution. Information on the total number of workers involved in these industries is not available.

There is a potential for dermal exposure to formalin products through spills or splashes onto skin or eyes during some processes, such as manual dilution of the concentrated formalin solutions and dispensing solutions from storage tanks to specimen jars. There is also potential for inhalational exposure to formaldehyde fumes during fixing and accessioning human tissues and organs. The exposure durations in these industries vary considerably. Some workers, such as hospital staff and doctors, would have brief exposure when placing human tissues into specimen jars filled with formalin solutions whereas fixing and accessioning human tissues and organs conducted by staff at most forensic/hospital mortuaries and pathology laboratories are daily activities. The exposure duration for students studying tissues and organs fixed by formalin solutions varies, but is limited to the period they are in laboratories.

Information from the industry indicates that the majority of laboratories/mortuaries are equipped with local exhaust systems and some also have an exhaust system for the whole area. Considering the degree of manual handling that the process involving formaldehyde can entail in these industries, the ergonomic design of the laboratory/mortuary and fume cabinet and their effectiveness, combined with work procedures and training, are important factors influencing the potential for exposure. Examination gloves, safety glasses and laboratory gowns are worn at the majority of laboratories/mortuaries. It was reported that respirators are also used when preparing large amounts of formalin solutions used in forensic medicine areas or anatomy laboratories.

Measured exposure data

Limited Australian personal and static monitoring data during use of formalin products in mortuaries, hospitals and pathology laboratories were available (Table 15.6a and Table 15.6b, respectively).
Long-term (37-428 min.) personal sampling results ranged from 0.02 ppm to 0.66 ppm in an anatomy laboratory without local exhaust ventilation (Table 15.6a), with the majority of results below 0.3 ppm. However, more recent limited data measured in a pathology laboratory showed levels up to 3 ppm. Formaldehyde levels decreased significantly after control measures were implemented. Data measured in the mid 1980s in hospital mortuaries and pathology laboratory showed higher results, ranging from 0.4 ppm to 4.8 ppm, with measurement durations between 23-77 min.

Limited long-term static data showed results of 0.2 ppm to 2.66 ppm in an anatomy dissection laboratory. Short-term static data in pathology laboratories showed most levels < 0.3 ppm, but some up to 1.5 ppm. Static spot testing data measured in forensic medicine mortuaries and anatomy laboratories showed levels that ranged from < 0.1 to up to 2 ppm, with most < 0.3 ppm (425 out of 593, about 70% samples).

Recent overseas personal monitoring data (Dufresne et al., 2002; Ryan & Burroughs, 2003; Akbar-khanzadeh & Pulido, 2003) found average levels of formaldehyde ranging from 0.2 to 0.9 ppm in 71 samples in total (Table 15.7).

Recent overseas static monitoring data (Table 15.7) showed average levels of formaldehyde ranging from 0.5 to 1.5 ppm in anatomy laboratories (no local exhaust ventilation in one laboratory) when dissection was being undertaken (Keil & Konecny, 2001). One study showed lower average levels of < 0.2 ppm in 18 samples (Ryan & Burroughs, 2003). No details on the sampling locations and ventilation were reported in the study with the highest reading of 9.34 ppm (Kim et al., 1999). Regular spot testing for 3 to 4.5 hours (Koda et al., 1999) showed levels of 0.2 to 0.4 ppm formaldehyde around workers’ breathing zone in a laboratory without specimen storage, although no local exhaust ventilation was in place. In a laboratory with large specimen storage and no local exhaust ventilation, formaldehyde levels were higher than 2 ppm in 21 spot testing samples, particularly when dissection started and when windows were closed.

Earlier overseas data reported that the mean concentrations of formaldehyde in the workroom air of anatomical theatres, pathology and hospital laboratories, and autopsy services ranged from 0.5 to 1.1 ppm. In studies of autopsy services, one study reported an average level of 4.2 ppm in 23 static samples collected, and another with 27 personal samples reported an arithmetic mean level of formaldehyde at 1.3 ppm in an autopsy service (IARC, 1995).

15.6.3 Embalming

Formaldehyde solutions containing up to 40% formaldehyde and paraformaldehyde prills/powder are commonly used in embalming in funeral homes, medical/anatomy laboratories and mortuaries. There are about 1700 funeral homes in Australia. However, not all funeral homes conduct embalming in Australia. The Australian Funeral Director Association (AFDA) reported that there are approximately 350 embalmers in Australia. AFDA represents 60% funeral homes in Australia. British Institute of Embalming (BIE) and New Zealand Embalming Association (NZEA) represent 40%. Embalming activities are less frequent in medical laboratories and mortuaries.
<table>
<thead>
<tr>
<th>Use</th>
<th>Activity/Location</th>
<th>No. of samples</th>
<th>Duration</th>
<th>Sampling method</th>
<th>Results# (ppm)</th>
<th>Comment</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anatomy dissection</td>
<td>general laboratory duty laboratory</td>
<td>4</td>
<td>241-428 min.</td>
<td>NIOSH 3500</td>
<td>0.02-0.08</td>
<td>no local exhaust ventilation.</td>
<td>Cattarin, 1997</td>
</tr>
<tr>
<td></td>
<td>human dissection</td>
<td>20</td>
<td>59-366 min.</td>
<td></td>
<td>0.03-0.34</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>wet specimen observation</td>
<td>6</td>
<td>37-79 min.</td>
<td></td>
<td>0.22-0.66</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anatomy dissection</td>
<td>Dissection laboratory (pre modification)</td>
<td>2 (same person)</td>
<td>2 h</td>
<td>Radiello passive sampler</td>
<td>1.66, 3.15</td>
<td>Modification includes use of ‘Infutrace’*</td>
<td>Personal communication, 2004</td>
</tr>
<tr>
<td></td>
<td>(post modification) Tissue preservation</td>
<td>1</td>
<td>NR</td>
<td>Passive monitor and HPLC</td>
<td>0.46</td>
<td></td>
<td>Dingle &amp; Franklin. 2002</td>
</tr>
<tr>
<td>Pathology laboratory</td>
<td>Disposal of tissue specimens down a trap</td>
<td>1</td>
<td>48 min.</td>
<td>NIOSH Chromotropic Acid Method</td>
<td>4.8</td>
<td></td>
<td>Personal communication, 1986</td>
</tr>
<tr>
<td></td>
<td>Fixing organs in formalin</td>
<td>1</td>
<td>73 min.</td>
<td></td>
<td>0.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Tissue dissections and tissue sample preparation, formalin solution dilution</td>
<td>1</td>
<td>55 min.</td>
<td></td>
<td>2.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Tissue dissections and tissue specimen examination</td>
<td>1</td>
<td>35 min.</td>
<td></td>
<td>1.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hospital mortuaries</td>
<td>Dilute formalin solution, lung perfusion mock up, pouring and decanting formalin solutions and organ washing</td>
<td>3</td>
<td>23-32 min.</td>
<td>ACGIH Bisulphite Addition Method 1 = 0.4 1 = 0.6 1 = 1.6</td>
<td></td>
<td></td>
<td>Personal communication, 1986</td>
</tr>
<tr>
<td>Sterilising dialysis machines</td>
<td>Flush a dialysis machine using 40% formalin</td>
<td>1</td>
<td>23 min.</td>
<td>NIOSH Chromotropic Acid Method</td>
<td>0.4</td>
<td></td>
<td>Personal communication, 1987</td>
</tr>
</tbody>
</table>

NIOSH, National Institute for Occupational Safety and Health. NR, not reported; ACGIH; the American Conference of Governmental Industrial Hygienists; HPLC; High Performance Liquid Chromatography.

# The results are presented as individual results (where only one sample). *'Infutrace’ is a commercial product which claims to neutralise formaldehyde in the cadavers by spraying it onto cadavers.
<table>
<thead>
<tr>
<th>Use</th>
<th>Activity/Location</th>
<th>No. of samples</th>
<th>Duration</th>
<th>Sampling method</th>
<th>Results# (ppm)</th>
<th>Comment</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anatomy dissection laboratory</td>
<td>dissection class</td>
<td>18</td>
<td>10 min.</td>
<td>direct reading device</td>
<td>14 &lt;0.25, 4 0.5-1.5</td>
<td>Result bands as provided</td>
<td>Personal communication, 1999</td>
</tr>
<tr>
<td>Dissection room</td>
<td>3</td>
<td>2 h</td>
<td>Radiello passive sampler</td>
<td>0.8, 0.75, 0.39</td>
<td>(pre modification), (post modification)</td>
<td>Personal communication, 2004</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>modification includes use of ‘Infutrace’*</td>
<td></td>
</tr>
<tr>
<td>Cold room</td>
<td>3</td>
<td>12 h</td>
<td></td>
<td></td>
<td>0.91, 1.33, 2.66</td>
<td>(pre modification), (post modification)</td>
<td></td>
</tr>
<tr>
<td>Cold room</td>
<td>1</td>
<td>15 min</td>
<td></td>
<td></td>
<td>1.08</td>
<td>(post modification), room door left open</td>
<td></td>
</tr>
<tr>
<td>Prosectorium</td>
<td>1</td>
<td>2 h</td>
<td></td>
<td>formaldehyde meter</td>
<td>0.23</td>
<td>(post modification)</td>
<td></td>
</tr>
<tr>
<td>Anatomical pathology laboratory</td>
<td>Cut-up bench</td>
<td>563</td>
<td>Spot testing</td>
<td>formaldehyde meter</td>
<td>200 &gt;0.1, 113 &gt;0.1-0.2, 86 &gt;0.2-0.3, 70 &gt;0.3-0.5, 70 &gt;0.5-2, 24 &gt;2; &lt;0.1 to &gt;4</td>
<td>The meter was close to workers’ breathing zone every 3-5 min. during a shift</td>
<td>Personal communication, 2002-2003</td>
</tr>
<tr>
<td>Dissection room (over night 6pm to 6am)</td>
<td>180</td>
<td>spot testing</td>
<td>formaldehyde meter</td>
<td>11 &gt;0.1, 5 &gt;0.1-0.2, 3 &gt;0.2-0.3, 1 &gt;0.3-0.5, 3 &gt;0.5-2</td>
<td>Higher levels detected at beginning of the testing (6pm) when air conditioning was off and then gradually decreased</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Forensic medicine mortuary</td>
<td>around proctors’ desks</td>
<td>23</td>
<td>spot testing</td>
<td>formaldehyde meter</td>
<td>11 &gt;0.1, 5 &gt;0.1-0.2, 3 &gt;0.2-0.3, 1 &gt;0.3-0.5, 3 &gt;0.5-2</td>
<td>local exhaust ventilation available.</td>
<td>2001-2002 (NICNAS survey)</td>
</tr>
<tr>
<td>dissection room</td>
<td>7</td>
<td>spot testing</td>
<td>formaldehyde meter</td>
<td>3 &gt;0.1, 3 &gt;0.1-0.2, 1 &gt;0.2-0.3</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Formaldehyde**
<table>
<thead>
<tr>
<th>Use</th>
<th>Activity/Location</th>
<th>No. of samples</th>
<th>Duration</th>
<th>Sampling method</th>
<th>Results# (ppm)</th>
<th>Comment</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pathology laboratory</td>
<td>Tissue preservation</td>
<td>20</td>
<td>NR</td>
<td>Passive monitor and HPLC</td>
<td>0.14-3.01 (mean = 0.98)</td>
<td></td>
<td>Dingle et al. 2002</td>
</tr>
<tr>
<td>Hospital mortuaries</td>
<td>On the bench next to the formalin pump</td>
<td>1</td>
<td>68 min.</td>
<td>NIOSH Chromotropic Acid Method</td>
<td>0.8</td>
<td></td>
<td>Personal communication, 1986</td>
</tr>
<tr>
<td>Hospital mortuaries</td>
<td>Workbench next to biopsy storage area</td>
<td>2</td>
<td>237-239 min.</td>
<td>ACGIH Bisulphite Addition Method</td>
<td>0.3 and 0.4</td>
<td></td>
<td>Personal communication, 1986</td>
</tr>
<tr>
<td></td>
<td>Dissection area</td>
<td>1</td>
<td>227 min.</td>
<td>ACGIH Bisulphite Addition Method</td>
<td>1.6</td>
<td>Brain sections impregnated with 40% formalin solution were left sitting covered with a towel.</td>
<td></td>
</tr>
<tr>
<td>Sterilising dialysis machines</td>
<td>Flush a dialysis machine using formalin (40%)</td>
<td>2</td>
<td>21-22 min.</td>
<td>NIOSH Chromotropic Acid Method</td>
<td>1 = 0.8</td>
<td></td>
<td>Personal communication, 1987</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1 = 0.3</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

NR, not reported; HPLC, High Performance Liquid Chromatography; NIOSH, National Institute for Occupational Safety and Health; ACGIH, the American Conference of Governmental Industrial Hygienists.

The results are presented as the number of samples in a series of result bands.
<table>
<thead>
<tr>
<th>Use</th>
<th>Type of sampling</th>
<th>Activity/Location</th>
<th>No. of samples</th>
<th>Duration</th>
<th>Test method</th>
<th>Results (ppm)</th>
<th>Comment</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pathology laboratories</td>
<td>static</td>
<td>pathologists/technicians in hospital A</td>
<td>185</td>
<td>spot testing every 2-3 min. for 4.5 hours</td>
<td>photoacoustic infra-red detection</td>
<td>All in range 0.2-0.4</td>
<td>no local exhaust ventilation, without specimen storage</td>
<td>Koda et al., 1999</td>
</tr>
<tr>
<td></td>
<td></td>
<td>pathologists/technicians in hospital B</td>
<td>62</td>
<td>spot testing every 2-3 min. for 3 hours</td>
<td>photacoustic infra-red detection</td>
<td>41 &lt;2 0.6-1 ppm (n=13) when windows were open 21 &gt;2 when dissection started, reached 8.6 ppm when windows were closed.</td>
<td>no local exhaust ventilation, with a large specimen storage. Irritated eyes, nose and throat and cough reported.</td>
<td></td>
</tr>
<tr>
<td>Biology laboratories</td>
<td>personal</td>
<td>Students in animal health training</td>
<td>18</td>
<td>3 h</td>
<td>NIOSH 3500</td>
<td>0.2-0.5 (average range)</td>
<td></td>
<td>Dufresne et al., 2002</td>
</tr>
<tr>
<td>Gross anatomy laboratories</td>
<td>personal</td>
<td>Medical students doing dissecting operations</td>
<td>21</td>
<td>3 h</td>
<td>NIOSH 3500</td>
<td>0.9 (mean)</td>
<td></td>
<td>Akbar-khanzadeh and Pulido, 2003</td>
</tr>
<tr>
<td></td>
<td>static</td>
<td>centre of laboratory and other locations (vary each day)</td>
<td>33</td>
<td>3 h</td>
<td>NIOSH 3500</td>
<td>0.6 (mean)</td>
<td></td>
<td>Keil &amp; Konceny, 2001</td>
</tr>
<tr>
<td>Gross anatomy laboratories</td>
<td>static</td>
<td>centre of laboratory and 50 other locations (vary each day)</td>
<td>50</td>
<td>3-4 h/d</td>
<td>NIOSH 3500</td>
<td>0.50-1.49 (mean)</td>
<td>no local exhaust ventilation</td>
<td></td>
</tr>
<tr>
<td>Anatomy laboratories</td>
<td>personal</td>
<td>Students and instructors</td>
<td>19</td>
<td>2-4 h</td>
<td>NIOSH 2016 Passive dosimeter</td>
<td>0.42 (mean)</td>
<td>Air conditioned room, No windows, doors open sometimes. burning eyes and nose or watery eyes reported.</td>
<td>Ryan &amp; Burroughs, 2003</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>13</td>
<td>2-4 h</td>
<td>NIOSH 2016 Active sampler</td>
<td>0.21 (mean)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>static</td>
<td>In a middle of a dissection table &amp; in a corner of the room</td>
<td>6</td>
<td>2-4 h</td>
<td>NIOSH 2016 Passive dosimeter</td>
<td>0.21 (mean)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>12</td>
<td>2-4 h</td>
<td>NIOSH 2016 Active sampler</td>
<td>0.16 (mean)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cadaver dissection</td>
<td>static</td>
<td>NR</td>
<td>48</td>
<td>1-2 h</td>
<td>NIOSH 3500</td>
<td>0.16-9.34 (no details for using result bands)</td>
<td>eye soreness and lacrimation reported</td>
<td>Kim et al., 1999</td>
</tr>
</tbody>
</table>

NR, not reported; NIOSH, National Institute for Occupational Safety and Health.
Embalmers are likely to be exposed by skin contact through spills or splashes onto skin or eyes during handling processes, such as dilution, arterial and cavity embalming, cleansing and disinfections of body surfaces and orifices, and equipment cleaning by hosing. Spills and splashes during embalming were observed at the site visited. The likelihood of exposure by inhalation is also high during embalming, such as during dilution of concentrated formalin solutions, application of the solution by spraying, and handling viscera covered with paraformaldehyde powder. The duration of embalming a body varies depending on body conditions and customers’ requirements, but usually takes 1 to 3 hours. The majority of the embalmers operate on a daily basis and exposure durations vary from 1 to 10 hours a day. Other staff involved in handling embalmed bodies, such as body dressing up and body lifting, may also be exposed to formaldehyde.

Ventilation systems installed in embalming rooms vary at different funeral homes. In general, new premises are fitted with an airflow system that blows fumes away from embalmers, together with an exhaust fan to extract the fumes. Whereas, in old buildings, usually only an exhaust fan is available. It was reported that some mortuaries do not have exhaust ventilation. Embalmers wear PPE during embalming including safety goggles, surgical gloves, theatre gown, disposable apron and rubber boots. Respiratory protection equipment is not usually used, although half masks and air-supplied respirators are available at some funeral homes.

**Measured exposure data**

Limited personal and static Australian air monitoring data during embalming in funeral homes and anatomy dissection laboratories were provided and are summarised in Table 15.8a and 15.8b, respectively.

Four samples with results of 0.1 to 0.6 ppm were measured during embalming in an anatomy dissection laboratory and one sample of 1 ppm in a hospital mortuary (measurement durations range from 43 minutes to 2 h) (Table 15.8a). Recent air monitoring data measured in funeral homes for 30 minutes showed 8 out of 13 personal samples gave results of >0.5 ppm, with a highest reading of 3.9 ppm. Short-term monitoring data showed levels of < 0.4 ppm in 4 out of 5 samples in one study, however, the product used contained only 1.4% formaldehyde. Another short-term monitoring result was 1.39 ppm (15 min).

Static monitoring data (Table 15.8b) are available for only 4 samples collected during embalming in funeral homes and showed higher levels for old data (1.1 ppm in 1986) compared to more recent data (0.21, 0.32 and 0.69 ppm).

Earlier overseas data showed arithmetic means of 0.3 to 0.9 ppm formaldehyde in 71 personal samples during embalming, but one study measured a mean level of 2.58 ppm formaldehyde in 25 personal samples (IARC, 1995). Results (arithmetic means) from a large number of static samples (128 samples plus unknown number of samples from 6 funeral homes and 23 mortuaries) ranged from 0.5 to 2.16 ppm in workplaces where embalming was conducted (IARC, 1995).

Recent overseas monitoring data showed levels of formaldehyde < 0.2 ppm during embalming (Table 15.7), but the data is limited.
<table>
<thead>
<tr>
<th>Use</th>
<th>Activity/location</th>
<th>No. of samples</th>
<th>Duration</th>
<th>Sampling method</th>
<th>Results (ppm)</th>
<th>Comment</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anatomy dissection laboratory</td>
<td>Embalming</td>
<td>3</td>
<td>43-52 min.</td>
<td>NIOSH 3500</td>
<td>0.07-0.56</td>
<td>sampling conducted during tasks.</td>
<td>Cattarin, 1997</td>
</tr>
<tr>
<td>Anatomy dissection laboratory</td>
<td>Embalming (post modification)</td>
<td>1</td>
<td>2 h</td>
<td>Radiello passive sampler</td>
<td>0.39</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pathology laboratory</td>
<td>Tissue preservation</td>
<td>NR</td>
<td>8 h</td>
<td>Passive monitor and HPLC</td>
<td>0.3-2.66 (mean = 0.98)</td>
<td></td>
<td>Dingle &amp; Franklin, 2002</td>
</tr>
<tr>
<td>Hospital mortuaries</td>
<td>Embalming post-mortem body using a 1.5% formalin solution</td>
<td>1</td>
<td>66 min.</td>
<td>NIOSH Chromotropic Acid 1 Method</td>
<td></td>
<td></td>
<td>Personal communication, 1986</td>
</tr>
<tr>
<td>Embalming at a funeral home</td>
<td>Embalming (post-autopsy)</td>
<td>1</td>
<td>78 min.</td>
<td>NIOSH Chromotropic Acid 1 Method</td>
<td></td>
<td></td>
<td>Personal communication, 1986</td>
</tr>
<tr>
<td></td>
<td>Embalming (non-post mortem)</td>
<td>2</td>
<td>91-99 min.</td>
<td></td>
<td>1 = 0.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Embalming at funeral homes washing body, fluid injection, Aspirating body cavity and filling/open/flushing body cavity.</td>
<td>10</td>
<td>30 min.</td>
<td>Glass fibre filter impregnated with 2,4-Dinitrophenylhydrazine</td>
<td>1 &lt;0.1 1 = 1.0 4 &gt;0.1-0.5 2 &gt;0.5-1 3 &gt;1-2</td>
<td>The known concentrations of formaldehyde in embalming solutions range from 0.9 to 28%</td>
<td>McGarry and Coward, 2003-2004</td>
</tr>
<tr>
<td></td>
<td>Body preparation and arterial flushing, Body 3 cavity injection</td>
<td>30 min.</td>
<td></td>
<td>Solid Sorbent Tube (10 % (2-hydroxymethyl)piperidine on XAD-2)</td>
<td>1 = 1.1 1 = 2.4 1 = 3.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Embalming at Funeral homes Embalming in a room with LEV, worker worn PPE (surgical gloves, face shield and a plastic apron)</td>
<td>5</td>
<td>15 min. (STEL)</td>
<td></td>
<td>OSHA Method 64 (Active sampling method)</td>
<td>3 = 0.05ppm 1 = 0.17 1 = 1.83</td>
<td>Using a product containing 1.4% formaldehyde. The higher readings were measured when the embalmer was temporarily between the body and the exhaust fan.</td>
<td>Tkaczuk et al., 1993</td>
</tr>
<tr>
<td>Embalming at Funeral homes Embalming in a room with LEV</td>
<td>1</td>
<td>15 min.</td>
<td></td>
<td>NIOSH 2541</td>
<td>1 = 1.39</td>
<td>Products contain 1.4% to 27.5% formaldehyde</td>
<td>Personal communication, 1999</td>
</tr>
</tbody>
</table>

NIOSH, National Institute for Occupational Safety and Health. NR, not reported. STEL, short-term exposure limit; LEV, local exhaust ventilation; OSHA; Occupational Safety and Health Authority. # The results are presented as individual results when only one sample or as the number of samples in a series of result bands. 

Formaldehyde 169
Table 15.8b: Summary of Australian static monitoring data during use of formalin solutions in embalming

<table>
<thead>
<tr>
<th>Use</th>
<th>Activity/location</th>
<th>No. of samples</th>
<th>Duration</th>
<th>Sampling method</th>
<th>Results# (ppm)</th>
<th>Comment</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Embalming at a funeral home</td>
<td>Embalming</td>
<td>1</td>
<td>72 min.</td>
<td>NIOSH Chromotropic Acid Method</td>
<td>1.1</td>
<td></td>
<td>Personal communication, 1986</td>
</tr>
<tr>
<td>Embalming at Funeral homes</td>
<td>Embalming in a room with LEV, worker worn PPE (surgical gloves, face shield and a plastic apron)</td>
<td>1</td>
<td>90 min.</td>
<td>OSHA Method 64 (Active sampling method)</td>
<td>0.21</td>
<td></td>
<td>Tkaczuk et al., 1993</td>
</tr>
<tr>
<td>Embalming at Funeral homes</td>
<td>Embalming in a room with LEV</td>
<td>1</td>
<td>4h</td>
<td>NIOSH 2541</td>
<td>0.32</td>
<td></td>
<td>Personal communication, 1999</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>15 min.</td>
<td></td>
<td>0.69</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*NIOSH, National Institute for Occupational Safety and Health. Photographic film processing*
15.6.4 Photographic film processing

Aerial and commercial film processing use products containing formaldehyde. The number of workers involved in aerial film processing is unknown, but is limited as the number of specialised film processing companies is small. However, there are more than 1000 commercial film processing machine operators in Australia using final baths and stabilisers containing formaldehyde.

Although the formaldehyde concentrations in the products used in aerial film processing are high (20% to 35%), the potential for workers’ exposure to formaldehyde is limited as the film processing is conducted in an enclosed machine. Short exposures are possible during the connection of the drum to the machine and during drum changeover. Exhaust systems inside the processing machines and floor level exhaust systems are available at aerial film processing sites. Besides the basic PPE (gloves, goggles and protective clothing), respiratory protection is also used during changing of drums at some workplaces.

Similarly, the potential for exposure to formaldehyde during the commercial film processing machines operation is limited. At the site visited, roof exhaust fans, industrial fans and general ventilation were used for ventilation. Workers wear gloves during handling formaldehyde products.

There is a potential for exposure during manual film processing when handling solutions containing 10% formaldehyde, as this operation is usually conducted in open trays in a dark room. However, the operation occurs only occasionally as trials in aerial film processing.

Measured exposure data

No Australian air monitoring data during end use of film processing products was provided. Earlier overseas data (IARC, 1995) showed a range of formaldehyde levels from < 0.01 ppm to 0.9 ppm in film processing plants, but no recent data are available. Therefore, an EASE model estimation was conducted.

Estimated data

The EASE scenario that best describes the film processing is a closed system without direct handling and system breaching for inhalation exposure, as it refers to processes in which substances remain in an enclosed system (UK HSE, 2000). The predicted inhalational exposure to formaldehyde during film processing is 0 to 0.1 ppm (0-0.12 mg/m³). The printout of the EASE modelling results is in Appendix 8.

Considering the concentrations of formaldehyde in the products, uses of PPE and the short exposure durations, it is reasonable to assume that the occupational exposure by inhalation is less than 0.1 ppm.

15.6.5 Leather tanning using formalin solutions

The leather tanning process using formalin solutions is described in Section 7.3.3 and the concentrations of formaldehyde range from 10% to 37%, although they are diluted into a 1:10 working solution for treating leather. Workers may be potentially exposed for short durations to formaldehyde during dilution and loading of the working solution. It was reported in one workplace that a local
exhaust fan is in place and all lids are closed during dilution. Workers normally wear gloves, apron, rubber boots and safety glasses. Potential for exposure is likely to be low during the leather processing as it is conducted in enclosed processing drums. In addition, leather tanning using formalin solutions takes place occasionally (a few times a year) however this use appears to be declining.

15.6.6 Sanitising treatment

Although formalin (containing 37% to 40% formaldehyde) is used as an additive to sanitise water treatment plants, this operation is only undertaken occasionally (about once a year). In addition, the concentrated formalin solutions are diluted to 1% before end use. Local exhaust ventilation is available for dilution and dispensing of the solutions. It was reported that rubber gloves, face shield and apron are used during the operation. Respirators are available for use in situations when high levels of formaldehyde fumes may occur, for example, cleaning up spills. Therefore, the potential for exposure during water treatment is considered limited.

Workers may be exposed to formalin solutions during sanitisation of bins and portable toilets, especially exposure by the skin through spills and splashes. However, based on the description of the use processes (Section 7.3.3.) including dilution, use frequency and quantities used, the potential for exposure is likely to be limited.

15.6.7 Lubricant products

Although some lubricant products contain > 0.2% formaldehyde, the working solutions are usually diluted before use and used in an enclosed system (Section 7.3.3). Therefore, the potential for worker exposure is limited to time spent in dilution of the product, which is a short and infrequent operation. Monitoring data were not identified, but the exposure to formaldehyde is considered negligible.

15.6.8 Analytical laboratories

The extent of exposure during this use is likely to be highly variable. There is potential for dermal exposure to the high concentrations of formalin commonly used in laboratories, through drips, spills or splashes onto skin or eyes while transferring formalin to and from beakers, and through contact with wastewater used to wash instruments. There is also potential for inhalational exposure to fumes during transfer of formaldehyde solution. However, exposures are minimised by a number of factors, such as confining the use of formaldehyde to fume cupboards, limited duration of use, appropriate procedures for the disposal of contaminated materials and use of PPE. Monitoring data conducted in analytical laboratories at resin and wood product manufacturing sites (Table 15.2a, 15.2b and Table 15.4) showed formaldehyde levels ranging from < 0.1 ppm to up to 0.2 ppm. A study measuring formaldehyde in chemical and dental laboratories using passive sampler technique showed results of 0.02 ppm to 0.03 ppm (Gillett, 2000). Therefore, the potential for workers’ exposure in analytical laboratories is likely to be limited.
15.6.9 Fumigation

It is unlikely that workers are exposed to formaldehyde gas during workplace fumigation, as access to the area is restricted and activation of the fumigation generators and air conditioning is remote controlled. The levels of formaldehyde are monitored and must be less than 0.2 ppm before access is allowed. Workers may be exposed to formaldehyde during transfer of the paraformaldehyde granules into gas generators and during disposal of the residue, but the durations are short for these activities. Additionally, latex gloves, overalls and airflow helmet with cartridge filter are used by operators. Moreover, this operation is infrequent. The extent of exposure during this use is considered negligible.

15.6.10 Monitoring data on other use of formaldehyde products

Recently published monitoring data studying formaldehyde levels in workplaces using formaldehyde products other than the uses discussed above are summarised in Table 15.9.

Table 15.9: Monitoring data on other uses of formaldehyde products

<table>
<thead>
<tr>
<th>Workplace</th>
<th>Test type</th>
<th>No. of samples</th>
<th>Duration</th>
<th>Test method</th>
<th>Result (mean)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Garage</td>
<td>personal</td>
<td>53</td>
<td>48 h</td>
<td>Passive sampler, HPLC</td>
<td>0.04 ppm</td>
<td>Zhang et al. 2003</td>
</tr>
<tr>
<td>Metalworking fluid</td>
<td>Personal</td>
<td>21</td>
<td>2-4 h</td>
<td>Portable pump, HPLC</td>
<td>0.04 ppm</td>
<td>Linnainmaa et al. 2003</td>
</tr>
<tr>
<td>Static</td>
<td>27</td>
<td>2-4 h</td>
<td></td>
<td>Portable pump, HPLC</td>
<td>0.04 ppm</td>
<td></td>
</tr>
</tbody>
</table>

15.7 Summary

Measured occupational exposure data in Australian workplaces are limited in major industries handling formaldehyde. The EASE modelling was used to estimate the exposure to formaldehyde for the end use in photographic film processing due to lack of monitoring data. The formaldehyde exposure levels across a number of major uses are summarised in Table 15.10.

A paper by Niemala et al. (1997) studied the trend of formaldehyde concentrations in Finnish workplaces using formaldehyde products, by analysing 1239 exposure measurements from 228 plants collected by the Register of Hygienic Measurements during 1980-1994. Industries included resin and wood panel production, furniture and carpentry industry, foundries, and textile industry. The exposure data indicated a clear reduction in the concentrations of formaldehyde while the Finnish workplace exposure standard for formaldehyde remained the same during the study period. The authors concluded that the reduction of workplace formaldehyde levels may be attributed to improved resin technology. A similar trend analysis of formaldehyde concentrations in Australian workplaces is not available.
### Table 15.10: Summary of occupational exposure data

<table>
<thead>
<tr>
<th>Use Scenario</th>
<th>Personal Monitoring (ppm)</th>
<th>Static Monitoring (ppm)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Long-term</td>
<td>Short-term</td>
</tr>
<tr>
<td>Formaldehyde manufacture</td>
<td>Most δ 0.2</td>
<td>0.5 ppm  (one sample only)</td>
</tr>
<tr>
<td>Formaldehyde resin manufacture</td>
<td>Most δ 0.2</td>
<td>Most δ 0.5</td>
</tr>
<tr>
<td>Product formulation (limited data)</td>
<td>Most δ 0.2</td>
<td>Up to 2</td>
</tr>
<tr>
<td>Pressed wood product manufacture</td>
<td>Most δ 0.3</td>
<td>No data</td>
</tr>
<tr>
<td>Wood working industry using particleboard and MDF</td>
<td>Most &lt; 0.2</td>
<td>No data</td>
</tr>
<tr>
<td>Forensic/hospital mortuaries &amp; pathology laboratories</td>
<td>Most δ 0.3  (up to 3)</td>
<td>No data</td>
</tr>
<tr>
<td>Embalming</td>
<td>Most &gt; 0.5 (up to 3.9)</td>
<td>Up to 1.4 (limited data)</td>
</tr>
</tbody>
</table>
16. Critical Health Effects for Risk Characterisation

16.1 Acute effects

In animal studies, formaldehyde is of moderate acute toxicity following inhalation (rat 4-hour LC50 value of 480 ppm (0.578 mg/m³)), oral (rat LD50 value of 800 mg/kg bw) and dermal exposures (rabbit LD50 value of 270 mg/kg bw). Information on clinical toxicology and histopathological changes from these animal studies are limited, though data from cases of human ingestion indicate that the acute toxicity of formaldehyde is related to its corrosive potential.

There are sufficient data to show that formaldehyde solution is a skin and eye irritant. Formaldehyde solution is corrosive due to the local injuries seen in humans following ingestion, together with the observance of severe eye irritation in a recent well-reported animal study with only 10 x1 of a 37% formaldehyde solution.

Eye and respiratory irritation have been reported in human epidemiology and chamber studies. Although gaseous formaldehyde is a known eye and upper respiratory tract irritant in humans, the limitations of the available data and subjective nature of sensory irritation do not allow identification of a definitive no-observed-effect level (NOEL). For the best available data, symptoms of sensory irritation have been self-reported in chamber studies at exposures between 0.25 to 3.0 ppm. Furthermore, for epidemiology studies, the unknown contribution of other substances in uncontrolled environments, mean the data are not considered reliable. In an extensive review of chamber studies by Bender (2002), it was concluded that the sensory irritation responses at levels of ε 1 ppm (1.2 mg/m³) could definitely be attributed to formaldehyde. Some individuals begin to sense irritation from 0.5 ppm (0.6 mg/m³), although the response rate is often similar to that reported in controls. Although there is limited evidence that some individuals report sensory irritation as low as 0.25 ppm (0.3 mg/m³) the data is very unreliable. Therefore, the LOEL is considered to be 0.5 ppm. Additionally, although mouse 10-minute RD50 values of 3.1 and 4 ppm (3.7 and 4.8 mg/m³, respectively) support formaldehyde being a respiratory irritant, the Alarie assay is not considered to provide reliable data for the purposes of risk characterisation.

Animal and human evidence clearly indicates formaldehyde is a strong skin sensitisier. In the EU, formaldehyde is considered a strong skin sensitisiser, having been evaluated by the EU Working Group on the Classification and Labelling of Dangerous Substances in 1995 and given a specific concentration limit of ≥0.2% for classification of solid and liquid mixtures with R43 (instead of the usual default limit of ≥1.0%).

The available human and animal data indicates formaldehyde is unlikely to induce respiratory sensitisation. Lung function tests suggest that asthmatics are no more sensitive to formaldehyde than healthy subjects. Limited evidence indicates that formaldehyde may elicit a respiratory response in some very sensitive
individuals with bronchial hyperactivity, probably through irritation of the airways.

16.2 Repeated dose effects (other than carcinogenicity)

As formaldehyde is highly reactive and rapidly metabolised at the site of contact, adverse effects are predominantly seen locally. Consequently, effects on pulmonary function, prevalence of eye, nose and throat irritation and histological changes within the nasal epithelium were investigated in populations exposed to formaldehyde in occupational and/or community environments. Overall, the data do not provide conclusive evidence that formaldehyde exposure induces effects on pulmonary function, and self-reported symptoms or irritation provide no reliable quantitative data. Conflicting results for histological changes within the nasal epithelium have been observed for workers occupationally exposed to formaldehyde. A small study by Holmstrom et al. (1989) is the most comprehensive human study available. In this study, histopathological changes were seen in the nasal epithelium of workers exposed to mean exposures of 0.25 ppm (0.3 mg/m³), with frequent short peak exposures above 0.8 ppm (0.96 mg/m³). However, overall, the weight of evidence for the histopathological changes is weak, due primarily to the limited number of investigations of relatively small populations that do not permit adequate investigations of exposure response.

Studies have also investigated effects on neurobehaviour in histology technicians exposed to formaldehyde. There is presently no convincing evidence that indicates formaldehyde is neurotoxic.

Most toxicological studies carried out in animals are inhalation studies, although data are also available for oral and dermal routes of exposure. No conclusive evidence of systemic toxicity was seen in these studies, and the health effect of concern following repeated exposure is irritation at the site of contact (i.e. skin irritation in the dermal study, and hyperplastic responses in the inhalation and oral studies). The data from inhalation studies shows a clear dose response for histological changes (cytotoxicity and hyperplasia), and indicates that effects are observed irrespective of exposure period. A NOAEL of 1 ppm (1.2 mg/m³) and a LOAEL of 2 ppm (2.4 mg/m³) were identified for histopathological changes to the nasal tract in a rat 28-month and 18-month study, respectively. For oral administration, a NOAEL of 15 mg/kg bw/day and a LOAEL of 82 mg/kg bw/day were identified for histopathological changes to the fore- and glandular stomach from a well conducted 2-year oral study in the rat. The brief details provided for the limited repeat dermal studies do not allow identification of a reliable NOAEL or LOAEL. The toxicological significance of the nasal findings is discussed in Section 10.4.

The genotoxicity of formaldehyde has been investigated in a number of in vitro and in vivo studies. The data show that formaldehyde is genotoxic in vitro, however, based on data from standard in vivo studies, formaldehyde does not appear to have systemic genotoxic potential in vivo. With regards to local effects in vivo, an increase in micronuclei in the gastrointestinal tract of rats following oral exposure are considered a consequence of cytotoxicity, though a marginal but statistically significant increase in chromosomal aberration was seen in pulmonary macrophages. Uncertainty exists in interpreting the reliability of the
data from this non-standard study. The relevance of the finding that formaldehyde is capable of producing DPX formation is discussed in detail in Section 10.5.

The available data indicate that at exposures relevant to humans, it is unlikely that formaldehyde will cause reproductive and developmental effects. In the only fertility study available, no adverse effects on fertility or parental toxicity were seen in a dietary study in minks. No effect on epididymal sperm morphology was seen in an oral mouse study, and no effects on the testes have been reported in rodents in a chronic repeated oral study and chronic inhalation studies. In a study investigating the effects of formaldehyde on testicular trace element concentrations, a reduction was seen in zinc and copper concentrations that was a secondary non-specific consequence of severe general toxicity (Ozen et al., 2002).

For developmental toxicity, there is no human evidence to indicate occupational exposure to formaldehyde is associated with low birth weight or malformations, while no reliable conclusions can be drawn from the epidemiology studies investigating spontaneous abortions. In animal studies, no developmental or maternal toxicity was observed in a dietary study in dogs. In a rat inhalation study, a slight but statistically significant reduction in foetal body weight was seen at 39 ppm (46.8 mg/m$^3$) that was a secondary non-specific consequence of severe maternal toxicity (Saillenfait et al., 1989). The NOAEC for both maternal and foetal toxicity was 20 ppm (24 mg/m$^3$).

16.3 Carcinogenicity

The relationship between formaldehyde exposure and cancer has been investigated in numerous animal and epidemiological studies. The principal carcinogenic effects observed in these studies were nasal tumours and leukaemia by inhalation.

Nasal cancers

Formaldehyde is carcinogenic in rat inhalation studies, producing an increased incidence in nasal squamous cell carcinomas. In the most comprehensive study available in the rat (Monticello et al., 1996), a significant increase in the incidence of nasal squamous cell carcinomas was observed at concentrations > 6 ppm (> 7.2 mg/m$^3$), single incidence was seen at 6 ppm and no tumours at 2 ppm (2.4 mg/m$^3$). The data suggest a difference in species sensitivity, as no significant increase in nasal tumours was seen in mice and hamsters at concentrations that were clearly carcinogenic in the rat: 14.3 ppm (17.2 mg/m$^3$) and 10 ppm (12 mg/m$^3$), respectively.

There are several epidemiological studies that show an increased risk of nasopharyngeal cancers, whereas other studies do not. Overall, although it cannot be definitely concluded that occupational formaldehyde exposure results in the development of nasopharyngeal cancer, there is some evidence to suggest a causal association between formaldehyde exposure and nasopharyngeal cancer. In addition, the postulated mode of action is considered likely to be relevant to humans and is biological plausible (see Appendix 5 for more details). Therefore, based on the available nasopharyngeal cancer data, formaldehyde should be regarded as if it may be carcinogenic to humans following inhalation exposure. In
addition, the available epidemiology exposure data are not sufficiently reliable to develop a dose-response relationship for use in risk characterisation.

**Leukaemia**

Although an increased incidence in haemolymphoreticular tumours was reported in a single questionable drinking water study in the rat, the increase was not dose-related. Furthermore, the pooling of tumour types reported as leukemia and lymphomas prevents the dose-response relationship for leukemia to be specifically determined.

An increased risk of leukaemia, occasionally significant, has been inconsistently reported in human epidemiology studies. The available data do not allow construction of a dose-response relationship for formaldehyde exposure and incidence of leukaemia. Additionally, there is currently no biologically plausible mode of action (see Appendix 5) to explain why formaldehyde would be leukaemogenic. Overall, the available human and animal data are considered insufficient to establish an association between formaldehyde exposure and leukaemia.

**Other cancers**

Only a small number of oral studies are available and no significant tumour findings were observed in the most comprehensive study available. Overall, formaldehyde solution is not considered to be carcinogenic by the oral route of exposure. No skin tumours were seen in mouse initiation/promotion studies, the only dermal data available.

Increased risks of various cancers in organs such as pancreas have been seen in some studies with no consistent pattern. The available human and animal data is insufficient to establish an association between formaldehyde exposure and these cancers.

### 16.4 Dose-response analysis

The human health effects to consider for risk characterisation are sensory irritation, skin sensitisation, cell proliferation, and carcinogenicity.

#### 16.4.1 Sensory irritation

Although sensory irritation has been reported in many human epidemiology and chamber studies, the limitations of the available data and subjective nature of sensory irritation do not allow identification of a definitive no-observed-effect level (NOEL). Extensive chamber studies confirmed that at levels of 1 ppm and greater responses can be attributed to formaldehyde exposure. The chamber studies also found that some individuals begin to sense irritation from 0.5 ppm (0.6 mg/m³), although the response rate is often similar to that reported in controls. There is limited evidence that some individuals report sensory irritation as low as 0.25 ppm (0.3 mg/m³), however, the data is very unreliable. Therefore, the lowest-observed-effect level (LOEL) is considered to be 0.5 ppm.
16.4.2 Skin sensitisation

Several animal and human studies (Marzulli & Maibach, 1974; Jordan et al., 1979; Hilton et al., 1996; Hilton et al., 1998) have been conducted to induce and/or elicit a skin sensitisation response for the purpose of hazard identification. These studies were conducted at doses to elicit a response and not designed to identify a threshold.

There is growing consensus that thresholds can be identified for skin sensitisers (Kimber et al., 1999; 2001; Boukhman & Maibach, 2001; EC, 2002a). At present, the tests that are the most appropriate to identify a threshold have not been agreed upon.

Work is also underway to categorise skin sensitisers according to their potency. For example, the EU Expert Group on Sensitisation proposed three categories of skin sensitisers (extreme, strong, moderate) based on a range of sensitisation tests (LLNA, Bueller, and human data). The Expert Group categorised formaldehyde as a strong skin sensitiser (EC, 2002b).

16.4.3 Cell proliferation

Recently, dose-response data for regenerative cellular proliferation in F344 rats was extrapolated to humans. The rat regenerative cellular proliferation data were combined with a human flux computer model (a combination of computational fluid dynamics model of the human nasal passage in three dimensions with a one-dimensional description of the entire human respiratory tract) to predict the extent and intensity of the cytotoxic responses throughout the human respiratory tract (Conolly et al., 2002). It is considered that the human model provides a reasonable basis for the prediction of irritation to the respiratory tract. It was observed that the predicted formaldehyde flux cellular proliferation relationship in rats and rhesus monkeys is similar (Kimbell et al., 2001), which suggests that rodent-primate differences in susceptibility to the cytotoxic effect of formaldehyde are small. This increases the confidence in the use of the rat data for human dose-response modelling.

The extrapolation of the rat cell proliferation data into the human model required several adjustments of the data, including the relationship between duration of exposure and intensity of cell proliferation, site-to-site variation in cell proliferation, and site-specific prediction of formaldehyde flux into tissue. Furthermore, as the dose response was J-shaped (i.e. cell proliferation rates at 0.7 and 2 ppm were below the control value), a hockey-stick shaped dose-response curve was also fitted to the cell proliferation data with an inflexion point fixed at 2 ppm so as to be conservative for risk estimation. A more detailed derivation of the model can be found in Conolly et al., (2002).

The model was used to determine how differences in activity levels (i.e. breathing rate) could affect the predicted dose response for cytotoxicity in humans at three exertion (‘working’) levels: sitting, light activity and heavy activity. For the J-shaped curve, the predicted lowest effect concentration was 1 ppm formaldehyde for the heavy working activity. For the other working levels, the predicted lowest effect concentration was 2 ppm. Using the hockey-stick curve, the predicted lowest effect concentration was 0.6 ppm for all three working levels. Both models predicted no effects at ≤ 0.5 ppm. For risk assessment purposes, it is proposed
that the more conservative value using the hockey-stick curve (i.e. 0.5 ppm) be used.

16.4.4 Carcinogenicity

The available epidemiology data are not sufficient to establish a dose-response relationship for the purposes of cancer risk characterisation. A number of risk estimation models have been used to predict human cancer risks from inhalation exposure to formaldehyde, based on the nasal tumour response in rats, including fifth-order multistage model (US EPA, 1987), third-order multistage model (US EPA, 1991), benchmark dose model (Schlosser et al., 2003) and biologically based model (Conolly et al., 2004). Based on these models, upper bound risk estimates at 0.1 ppm formaldehyde exposure range from 1300 in a million to 0.58 in a million (Schlosser et al., 2003).

It is considered that the biologically-based 2-stage clonal growth model (Conolly et al., 2004), which incorporates mechanistic data on the proposed mode of action of tumour formation in rats, provides a better estimate of the actual risk of nasal cancer over the default approach of applying standard 10 x 10 default assumptions. The model offers the potential to decrease the uncertainties inherent in the extrapolation of data, both across species (e.g. rat to human) and from high experimental bioassay concentrations to those relevant to human exposure.

The model incorporates data on normal growth curves for rats and humans, cell cycle times, and cells at risk in the different regions of the respiratory tract. Species variations in dosimetry are taken into account by computational fluid dynamic models of the rat and human noses to predict regional formaldehyde doses (flux). Lower respiratory tract flux was predicted in humans in this model using a single path mode for the nasal, oral and lung airways. The details of the 2-stage clonal growth model and selection of various parameters can be found in Conolly et al., (2004) and are summarised in Appendix 9.

Although the mechanism of action is not well understood for nasal tumour formation in rats, regenerative cell proliferation associated with cytotoxicity appears to be an obligatory step in the induction of cancer by formaldehyde. In contrast, the probability of mutation resulting from DNA protein cross-linking (DPX) is unknown. However, in this model, formaldehyde is assumed to act as a direct mutagen, with the effect considered proportional to the estimated tissue concentration of DPX. This is despite the fact that animal studies are suggestive of a threshold effect for carcinogenicity. Thus, this component of the model provides a conservative and cautionary element in recognition of a lack of a fully elucidated mechanism of action.

Maximum likelihood estimate methods were used to fit the clonal growth model to cancer incidence data. A number of sensitivity analyses were run to determine the significance of specific modelling assumptions (i.e. the probability of mutation per cell division and the growth advantage for preneoplastic cells). Age-adjusted data on the incidence of lung cancers in humans were used to calibrate the human model for background tumour incidence.

Maximum likelihood estimates of the additional carcinogenic risk for occupational and public exposures (for non-smokers) using the clonal growth model are presented in Table 16.1. The clonal growth model predicts that for 40-year occupational exposure to 0.3 ppm formaldehyde (the NICNAS
recommended occupational exposure standard), the estimated additional risk for respiratory tract cancers is approximately 0.2 in a million. While at 1 ppm (the current occupational exposure standard) the estimated additional risk is approximately 50 in a million. Similarly for public exposure, at the recommended indoor air guidance value (80 ppb), the estimated additional risk is approximately 0.3 in a million.

Table 16.1: Predicted maximum human additional risk of respiratory tract cancer due to public and occupational exposures to formaldehyde (for non-smokers)

<table>
<thead>
<tr>
<th>Formaldehyde Exposure Concentration (ppm)</th>
<th>Predicted Additional Risk Public</th>
<th>Occupational</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.001</td>
<td>$2.94 \times 10^{-9}$ (H 0.003 in 1 million)</td>
<td>Simulation not done</td>
</tr>
<tr>
<td>0.02</td>
<td>$6.02 \times 10^{-8}$ (H 0.06 in 1 million)</td>
<td>$1.86 \times 10^{-8}$ (H 0.02 in 1 million)</td>
</tr>
<tr>
<td>0.04</td>
<td>$1.23 \times 10^{-7}$ (H 0.1 in 1 million)</td>
<td>$2.70 \times 10^{-8}$ (H 0.03 in 1 million)</td>
</tr>
<tr>
<td>0.06</td>
<td>$1.90 \times 10^{-7}$ (H 0.2 in 1 million)</td>
<td>$3.58 \times 10^{-8}$ (H 0.04 in 1 million)</td>
</tr>
<tr>
<td>0.08</td>
<td>$2.60 \times 10^{-7}$ (H 0.3 in 1 million)</td>
<td>$4.50 \times 10^{-8}$ (H 0.04 in 1 million)</td>
</tr>
<tr>
<td>0.10</td>
<td>$3.30 \times 10^{-7}$ (H 0.3 in 1 million)</td>
<td>$5.48 \times 10^{-8}$ (H 0.05 in 1 million)</td>
</tr>
<tr>
<td>0.30</td>
<td>$1.25 \times 10^{-6}$ (H 1 in 1 million)</td>
<td>$1.79 \times 10^{-7}$ (H 0.2 in 1 million)</td>
</tr>
<tr>
<td>0.50</td>
<td>$2.42 \times 10^{-6}$ (H 2 in 1 million)</td>
<td>$3.38 \times 10^{-7}$ (H 0.3 in 1 million)</td>
</tr>
<tr>
<td>0.60</td>
<td>$3.09 \times 10^{-6}$ (H 3 in 1 million)</td>
<td>$4.56 \times 10^{-7}$ (H 0.5 in 1 million)</td>
</tr>
<tr>
<td>0.70</td>
<td>$4.86 \times 10^{-6}$ (H 5 in 1 million)</td>
<td>$2.20 \times 10^{-6}$ (H 2 in 1 million)</td>
</tr>
<tr>
<td>1.00</td>
<td>$3.29 \times 10^{-5}$ (H 33 in 1 million)</td>
<td>$4.92 \times 10^{-5}$ (H 50 in 1 million)</td>
</tr>
</tbody>
</table>

180 year lifetime continuous exposure at indicated ppm.
280 year lifetime continuous exposure to a background environmental background level of 4 ppb with 40 years occupational exposure (8hr/day, 5 days/week) at indicated ppm beginning at age 18 years, with a “light working” breathing pattern.

Due to public concern of childhood chemical exposure and cancers, together with the findings of relatively high levels of formaldehyde in mobile homes and relocatable buildings, a worst-case scenario risk estimation incorporating higher exposures during childhood, has been conducted using the CIIT modelling. The worst-case scenario was identified to be children who live in mobile homes and spend all their schooling time in relocatable classrooms up to 17 years of age. The details of the worst-case scenario exposure levels, respiratory ventilation rate at different activity levels and other parameters used in the modelling are in Appendix 10. The predicted additional risk of respiratory tract cancer for a full 80-year lifetime, including childhood exposure to formaldehyde under the worst-case scenario is 0.45 in a million.

The clonal growth model (Conolly et al., 2004) is considered to provide the best estimates of cancer risk. However, it is noted that this model predicts substantially lower cancer risk than other models. This is attributed to the maximised use of mechanistic data in the clonal growth model, including the
incorporation of data on normal growth curves for rats and humans, cell cycle times and cells at risk in different regions of the respiratory tract (i.e. regional formaldehyde flux). NICNAS notes that CIIT and other regulatory authorities are reviewing the 2-stage clonal growth model and developing other risk estimates for cancer. These risk estimates, when available, will be considered along with any new significant epidemiological data as an ongoing process of re-evaluation of cancer risk as part of secondary notification activities (see Chapter 19).
17. Risk Characterisation

In this section, information on the environmental and health effects of formaldehyde (Section 8 to 11) has been integrated with environmental, public and occupational exposure estimates (Section 13 to 15), to characterise the potential risks of adverse effects the chemical may cause to the environment and people of Australia. This process provides the basis for identifying areas of concern and evaluating risk management strategies.

17.1 Environmental risks

Formaldehyde is ubiquitous in the environment owing to its formation through a range of natural processes, but is frequently detected at levels higher than background concentrations because of releases through human activities. Its major anthropogenic release in Australia is into the atmosphere, from diffuse and point sources, primarily during fuel combustion. Direct release into the soil and aquatic compartments is expected to be minor resulting primarily from industrial and commercial activities. Removal through biodegradation in sewage treatment facilities will greatly reduce the amounts of formaldehyde reaching receiving waters. Biodegradation in soil by micro-organisms will prevent any accumulation in soils.

17.1.1 Atmospheric compartment

There is very limited data available on the effects of exposure of terrestrial organisms to the gas or vapour phase formaldehyde. Ecotoxicity studies indicate potentially adverse effects on some plant species over the medium term (4-6 weeks) when exposed to formaldehyde in air and fog. The most sensitive species to formaldehyde in fog was Rapeseed (*Brassica rapa*), which showed a reduction in leaf area, leaf and stem dry weight, and flower and seedpod numbers when exposed intermittently (7 hours/day, 3 days/week) for 40 days to concentrations of 14.9 ppb of formaldehyde.

No information is available on the concentrations of formaldehyde in urban fog in Australia. Data from Italy showed mean concentrations of 3.9 ppb in fog. Thus, formaldehyde could conceivably reach levels of 14.9 ppb in fog. However, toxicity studies indicated that plants were affected in the early growth phases, which occur mainly in spring or summer. In winter, when the frequency of fog incidence is expected to be highest, plants are largely dormant, and thus effects on growing seedlings is not expected to be an issue.

The worst-case PECs in an urban area was determined to be 5.5 ppb (annual average) and 23.5 ppb (maximum 24-hour average). Monitoring data indicate concentrations of formaldehyde in air vary from one location to another, and with the season and time of day. A maximum concentration of 135 ppb was measured over a 1-hour averaging period in a high traffic area in South Australia, indicating that formaldehyde may reach levels in air high enough to have adverse effects on plants, particularly in or near urban or industrial environments. However, it is unlikely that high atmospheric concentrations would be maintained for long. This
is evident from the longer-term average monitoring data, where formaldehyde concentrations are significantly lower than the short-term average concentrations.

In summary, the likelihood of a risk to non-human organisms through atmospheric exposure to formaldehyde in outdoor situations is not indicated by the available evidence.

17.1.2 Aquatic compartment

Direct release of formaldehyde into the aquatic environment occurs via municipal sewage treatment facilities largely from the chemical manufacturing industry and in consumer products. Formaldehyde may also form naturally through ozonation of humic material, deposition from the atmosphere or through contamination by accidental spills (NHMRC/ARMCANZ, 1996). Due to its high biodegradability and low residence time, formaldehyde is not expected to reach significant levels in water.

Aquatic organisms are expected to be most at risk near spills and effluent outfalls and in urban areas with high rates of fallout and washout from the atmosphere. Chemical companies manufacturing formaldehyde indicate concentrations of < 20 mg/L going to trade waste. Trade waste effluent is treated on site prior to release into the municipal sewer.

The worst-case PEC_{local} arising from industrial releases, calculated for a metropolitan sewage treatment plant using the NPI 2001-2002 release estimates, is 1.4 μg/L (Section 13.3.1). The PEC would be further diluted in the receiving water. We assume a dilution factor of 10 for oceans (PEC = 0.14 μg/L) and no dilution in rivers. Derivation of a PEC from estimated concentration (< 20 mg/L) in trade waste entering the sewer is not possible. However, the concentration will be significantly reduced through dilution in the sewer.

For aquatic organisms, the most sensitive species is *Daphnia pulex*, with the lowest reported median effective concentration (EC50) of 5.8 mg/L. The PNEC, derived from the lowest EC50 taken from a large data source and applying a safety factor of 100, is 58 μg/L. The PEC/PNEC ratio derived from the NPI industrial release estimates is $3 \times 10^{-3}$, indicating a low concern. The PEC/PNEC ratio using trade waste effluent estimates is $1.7 \times 10^{-6}$, also indicating a low concern.

It is not known how much formaldehyde is released into the sewer through use of consumer products or in rain. The available data suggest that both consumer products (which generally contain < 0.2% formaldehyde) and rainout would contribute relatively low levels of formaldehyde, which would be further significantly diluted in the receiving water.

No surface water monitoring data for formaldehyde are available in Australia. Analysis of effluent in Canada found maximum concentrations of 325 μg/L (1-day mean) near an effluent treatment plant (Section 13.3.1). The highest concentration of formaldehyde found in surface water was 9.0 μg/L (average 1.2 μg/L).

Limited Australian data show measured concentrations of formaldehyde in rainwater are between < 3 μg/L and 6 μg/L. The measured concentrations of
formaldehyde in rain at other locations in the world ranged from 0.44 µg/L and 3003 µg/L, the latter during the burning-off season.

While formaldehyde is toxic to some aquatic organisms, it is readily biodegradable (half-life ranges from 24 to 168 hours), has a low bioaccumulation potential, and organisms are able to easily metabolise it. In addition, the PEC in water is predicted to be low. As such, the impact of formaldehyde on the aquatic environment is expected to be limited, except in the case of a major pollution event, such as a spill.

17.1.3 Terrestrial compartment

Exposure to formaldehyde in soils is most likely to occur through accidental spills or leaks of aqueous formaldehyde. It may also enter the soils through disposal of solid wastes (mainly resins) containing formaldehyde.

No PEC was calculated for soils. However, levels of formaldehyde entering the soil are expected to be low. No monitoring data are available for soil concentrations in Australia. Formaldehyde is toxic to a range of micro-organisms and is known to kill viruses, bacteria, fungi, and parasites. Algae, protozoa, and microscopic fungi appear to be most sensitive to formaldehyde, with acute lethal concentrations ranging from 0.3 mg/L to 22 mg/L. Consequently, formaldehyde could be expected to negatively impact soil microbial biomass and activity if a major spill occurs.

Spills of formaldehyde on the ground would be expected to infiltrate into the soil. However, since formaldehyde is susceptible to biodegradation by a range of micro-organisms, it is expected to be readily degraded and not accumulate.

Polymerised urea-formaldehyde resins persist in the soil but do not emit formaldehyde. Partially polymerised condensation products of low molecular weight degrade gradually and release formaldehyde vapour that can be broken down by soil micro-organisms (IPCS, 1989). As such, a low risk to organisms through soil exposure to formaldehyde is indicated by the available evidence.

17.2 Public health risks

Health effects of formaldehyde are observed primarily in the tissue of first contact and are related to the level of exposure rather than to total systemic intake. Therefore, characterisation of general public health risks associated with exposure to formaldehyde is based upon analysis of the concentrations of formaldehyde in both ambient and indoor air as well as via other media (such as cosmetics and consumer products, water, and food) rather than estimates of total daily intake.

17.2.1 Public exposure

The public is exposed to formaldehyde in air primarily through the inhalation of indoor and ambient air contaminated with the chemical. The major sources of formaldehyde in ambient air are release from combustion processes, such as burning of domestic fuel transportation, and industry emissions. The sources of indoor air formaldehyde are mainly pressed wood products that emit formaldehyde, cooking and heating appliances, and tobacco smoke.
Limited measured data indicate that concentrations of formaldehyde in the ambient air are highest close to industrial point sources, particularly those located in the urban environment. The estimated environmental exposures to formaldehyde using modelling techniques indicate that the maximum likely annual average PECs of formaldehyde is 5.5 ppb and the maximum 24-h average is 23.5 ppb. The modelled values are generally in agreement with measured data (details in Section 13.1.4).

Recent studies of indoor and outdoor ratios of formaldehyde levels found indoor formaldehyde levels are about 7 to 16 times higher than outdoor levels. The measured data indicate that the average levels of formaldehyde in indoor air of established conventional homes and offices in Australia range from 15 to 30 ppb, although the data in offices are limited. Recent limited monitoring data showed average formaldehyde levels of 29 ppb (range from 8 to 175 ppb) in occupied caravans and 100 ppb (range from 10 to 855 ppb) in unoccupied caravans. No monitoring data for manufactured homes, such as park cabins, are available. Therefore, mobile homes (including caravans/motor homes and manufactured homes) appear to have higher formaldehyde levels than conventional homes. This is primarily due to use of large quantities of formaldehyde emitting materials (principally pressed wood products) in these buildings. There are no recent Australian monitoring data for relocatable buildings including offices and classrooms. However, limited data from 1992 showed high levels of formaldehyde in relocatable offices (range from 420 to 830 ppb, with a mean of 710 ppb). It has been confirmed that pressed wood products are used extensively in manufacture of these buildings. Limited data also indicates that new offices or offices with new furniture may have higher formaldehyde levels than established ones.

The public can be also exposed to aqueous formaldehyde via use of cosmetic and consumer products generally at very low concentrations, but the exposure is expected to be widespread and repeated.

17.2.2 Health impacts

The critical health effects for the characterisation of public health risk are:

- Sensory irritation via inhalation exposure to formaldehyde gas (vapour), aerosol or mist; and
- Skin sensitisation following dermal exposure to formaldehyde solutions;
- Carcinogenicity via inhalation exposure to formaldehyde gas (vapour), aerosol or mist.

Sensory irritation

Although formaldehyde is a known eye and upper respiratory tract irritant in humans, the limitations of the available data and subjective nature of sensory irritation do not allow identification of a definitive no-observed-effect level (NOEL). The lowest-observed-effect level (LOEL) is considered to be 0.5 ppm (500 ppb) which is used to derive the recommended indoor air guidance value (see section 18.2.5) and ambient air standard (Recommendation 17) of 80 ppb. The estimated maximum annual average (5.5 ppb) and the maximum 24-hour
average (23.5 ppb) formaldehyde ambient air concentrations are well below the recommended ambient air standard.

Based on measured data, indoor formaldehyde levels in conventional homes and buildings are about 15 to 30 ppb which is about 3 times lower than the recommended formaldehyde guidance level. Therefore, the risk for sensory irritation from environmental exposure to formaldehyde in conventional homes and buildings is considered to be low. However, in mobile homes (range from 8 to 175 ppb in occupied caravans and 10 to 855 ppb in unoccupied caravans) and possibly relocatable buildings (range from 420 to 830 ppb, 1992 data), formaldehyde concentrations may be close to or above the recommended indoor air guidance level. Therefore, the indoor air formaldehyde in these types of buildings is of concern for sensory irritation.

**Skin sensitisation**

Formaldehyde solution is used in a wide range of cosmetics and consumer products through which the general public can be repeatedly exposed to formaldehyde via the skin.

Formaldehyde solution is a strong skin sensitisier. Although concentrations of formaldehyde in these products are generally low (< 0.2%), dermal exposure should be minimised or prevented wherever possible because even very low concentrations of formaldehyde in solution may elicit a dermatological reaction in individuals who have been sensitised.

**Carcinogenicity**

Based on the CIIT carcinogenic risk estimation of formaldehyde to humans (Section 16.4.4), the risk for respiratory tract cancer after 80 years lifetime continuous exposure to 100 ppb formaldehyde for a non-smoker is 0.3 in a million. For the worst-case scenario (childhood spent in mobile homes and attending schools with relocatable classrooms), the predicted additional lifetime risk of respiratory tract cancer is 0.45 in a million. Therefore, the public health risk for cancer of respiratory tract due to inhalation exposure to gaseous formaldehyde is considered to be low.

The public health risk for leukemia from inhalational exposure to formaldehyde is not considered in this risk characterisation due to insufficient data to establish a causal association between formaldehyde exposure and leukaemia. Although this issue cannot be totally dismissed, the current evidence do not warrant any regulatory actions. Further research in this area is ongoing and NICNAS will maintain a watching brief.

**17.2.3 Uncertainties**

There are several uncertainties in the public health risk characterisations. Uncertainties are due to limitations in the quality of relevant animal and human toxicity and health effects data, especially the unknown contributions of other substances in uncontrolled environments and estimates of exposure in epidemiological studies. In the case of sensory irritation, further uncertainties arise from difficulties in identifying a NOEL or LOEL. The CIIT 2-stage clonal growth model for assessing the carcinogenic risk of formaldehyde is considered a more reliable estimate of cancer risk than the standard default assumptions, due to
the incorporation of as many biological data as possible. However, it also has certain limitations and assumptions which were discussed in detail in a published paper (Conolly et al., 2004). NICNAS notes that CIIT and other regulatory authorities are reviewing the 2-stage clonal growth model and developing other risk estimates for cancer. These risk estimates when available, will be considered along with any new significant epidemiological data as an ongoing process of re-evaluation of cancer risk as part of secondary notification activities.

Limited information, such as indoor air monitoring data in mobile homes and relocatable buildings, also bring uncertainties to the risk characterisation. In addition, uncertainties are inherent in the assumptions and approximations used in modelling in order to estimate the likely exposure to formaldehyde in the Australian urban ambient air.

17.2.4 Summary

Sensory irritation and skin sensitisation have been identified as the key concerns for the general public. A qualitative risk characterisation for health impacts is summarised in Table 17.1. The risk of respiratory tract cancers is considered to be low for the public.

Table 17.1: Areas of concern to the general public due to formaldehyde exposure

<table>
<thead>
<tr>
<th>Health impact</th>
<th>Area of concern</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensory irritation</td>
<td>People living in mobile homes and possibly relocatable buildings</td>
</tr>
<tr>
<td>Skin sensitisation</td>
<td>A strong sensitiser, therefore, dermal exposure should minimised or eliminated</td>
</tr>
</tbody>
</table>

17.3 Occupational health risks

17.3.1 Physicochemical hazards

Formaldehyde is a highly reactive, flammable gas and can form explosive mixtures in air. It presents a fire hazard when exposed to flame or heat. Formalin can be a flammable liquid when formaldehyde or methanol concentrations are high.

A potential fire/explosion risk exists for formaldehyde gas and solution during manufacture, transport, storage and end use. However, formaldehyde has been subject to a number of regulations, such as major hazard facilities, storage and handling regulations, and transport regulations (details see Section 18.3.1). The fire/explosion risk, therefore, is significantly reduced.

17.3.2 Occupational exposure and health impacts

Occupational exposure to formaldehyde is predominantly by inhalation and may occur in workers in a variety of industries producing and using formaldehyde products. Dermal exposure may also occur during handling of formaldehyde products.
The critical adverse effects from exposure to formaldehyde include sensory irritation, skin sensitisation and carcinogenicity.

**Sensory irritation**

As mentioned previously, the LOEL for sensory irritation in humans is 0.5 ppm.

The highest occupational exposure to formaldehyde occurs during use of formaldehyde products in embalming, due to high concentrations of formaldehyde in these products, manual handling processes, high possibility of spills and splashes, and relatively frequent and long exposure durations. Limited Australian monitoring data and available overseas data indicate that formaldehyde levels around workers’ breathing zones during embalming are often high (up to 4 ppm), with mean levels greater than 0.5 ppm. Therefore, the risk of sensory irritation is expected to be high. Lack of, or inappropriate local exhaust ventilation system may lead to greater risks.

Similarly, use of formaldehyde products in forensic/hospital mortuaries and pathology laboratories is of concern for sensory irritation, due to a similar use pattern to that of embalming. Although limited Australian air monitoring data showed that the majority of the long-term personal monitoring readings are below 0.3 ppm, higher levels of formaldehyde (up to 3 ppm) were measured in recently conducted monitoring. Higher measurements were also reported in recent overseas literature, especially in rooms without local exhaust ventilation, with windows shut, and with large specimen storage using formalin.

During formaldehyde manufacture, the majority of long-term personal and static samples were < 0.2 ppm. Only one short-term personal sample is available, with a result of 0.5 ppm measured during formaldehyde drum filling. Although most of the short-term static samples showed levels of less than 0.2 ppm, several static samples were greater than 0.5 ppm, mainly measured at formaldehyde truck loading, storage areas and drum filling points. The risk of sensory irritation during formaldehyde manufacture is generally low as the process is fully enclosed. Concerns exist in situations when formaldehyde vapour displacement occurs and where there is a need to break open or enter the enclosed system, such as sample collection and testing, equipment cleaning and maintenance.

Similar long-term personal and static monitoring results were measured during the manufacture of resin, also an enclosed process. However, 21 out of 87 short-term personal samples had results of greater than 0.5 ppm. Only two of these readings had details on activities when the measurements were taken: one was due to opening a formaldehyde storage oven and the other due to technical activity (no further details given). Some short-term static readings of greater than 0.5 ppm were also measured, mainly during sampling and testing. Therefore, concerns exist in situations during abnormal operations, such as mechanical failure of hoses or seals and during sample collection and testing, truck loading and unloading, filling of drums, equipment cleaning and maintenance, opening of tanks and equipment, and spills.

The risk of sensory irritation is also expected during repacking and formulation of formaldehyde products, other than formaldehyde resins. Open operating processes and manual handling procedures are employed at some plants during some stages of formulation, such as transfer of raw material to another container or mixing vessels, during mixing and handling of final products. This is supported
by limited air monitoring data showing short-term personal sampling results ranging from 0.3 to 2 ppm during raw material weigh-up, equipment cleaning and maintenance. However, the majority of the limited long-term personal monitoring data showed < 0.2 ppm, probably due to batch process and use of exhaust ventilation.

The risk of sensory irritation during use of formaldehyde resins is expected to be low as the free formaldehyde levels in resins are generally low. This is in agreement with recent monitoring results in Australian plants, where the majority of long-term personal samples were < 0.3 ppm. However, formaldehyde levels in air may be higher when formaldehyde-based resins are heated and/or come in contact with high humidity levels due to the volatilisation of the free formaldehyde and/or decomposition of the resin. Some uses of formaldehyde resins may lead to a higher degree of concern due to the mode of application, for example, spraying, brushing, bathing/dipping, which may generate high levels of formaldehyde in the atmosphere.

Although products containing high concentrations of formaldehyde are used in photographic film processing, the risk of sensory irritation is expected to be low because of enclosure of the film processing system, use of diluted products and infrequent breaks of the enclosed system. Exposure estimation using the EASE model resulted in low estimated levels (0 to 0.1 ppm).

The extent of potential exposure during use of formaldehyde products as laboratory reagents is likely to be variable and will depend on a number of exposure control factors, such as confining the use of formaldehyde to fume cupboards, appropriate disposal procedures and use of PPE. All laboratories reported use of formaldehyde in fume cupboards and the wearing of PPE.

Due to unlikely or negligible potential exposure to the chemical, the risk of sensory irritation is expected to be minimal in the following situations, except in cases of accidental spills or leaks of the formaldehyde products:

- importation and transportation of unopened formalin or formaldehyde products;
- leather tanning;
- sanitising treatment;
- use of lubricant products; and
- workplace fumigation.

**Skin sensitisation**

Skin sensitisation of workers can occur if there is dermal exposure to formaldehyde, which may occur as a result of manual handling of formaldehyde products during formaldehyde manufacture, formulation and repackaging, and end use. The likelihood of dermal contact in some modes of end use, such as spraying (e.g. fibreglass industry and wall sealer application), brushing (e.g. composition construction and fibreglass industry) and dipping/bathing (e.g. paper coating and film processing) is high. Because formaldehyde solutions may induce skin sensitisation and even very low concentrations of formaldehyde in solution
may elicit a dermatological reaction in individuals who have been sensitised, dermal exposure should be minimised or prevented wherever possible.

Carcinogenicity

Based on the CIIT carcinogenic risk assessment of formaldehyde to humans (Section 16.4.4), the risk for respiratory tract cancer is low (< 1 in a million) after 40 years occupational exposure to $\delta$ 0.6 ppm formaldehyde for non-smokers. Long-term occupational exposure data indicate that the formaldehyde levels within workers’ breathing zones are usually less than 0.6 ppm in almost all use scenarios, with the majority less than or equal to 0.2 ppm. Consequently, the occupational risks for respiratory tract cancers after repeated exposure to formaldehyde by inhalation is considered to be low. However, the risk of respiratory tract cancers in some occupations may be higher, such as embalmers, if there are continuously high exposures.

17.3.3 Uncertainties in occupational risk characterisation

The risk characterisation for health effects involves uncertainties which are discussed in Section 17.2.3. Additional uncertainties are inherent in the assessment of formaldehyde exposure levels among Australian workers due to limited air monitoring data for most of the use scenarios discussed. There are also uncertainties associated with the assumptions used in the EASE modelling for exposure estimation, which are discussed in Appendix 8.

17.3.4 Areas of concern in occupational settings

The key concern for workers is sensory irritation. A qualitative risk characterisation for a number of use scenarios is summarised in Table 17.2.

The risk of skin sensitisation exists when dermal exposures to formaldehyde occur. The risk of respiratory tract cancers is considered to be low for the majority of workers.

17.4 Data gaps

The following significant data gaps were identified when undertaking the risk characterisation:

- Monitoring data on indoor air formaldehyde levels, especially in mobile homes and relocatable buildings;
- Air monitoring data in some industrial settings, especially at workplaces using high concentrations of formalin products, such as funeral homes and medicine-related industries;
- Epidemiology data permitting the establishment of a more reliable human NOEL for sensory irritation;
Table 17.2: Areas of concern in occupational settings due to sensory irritation

<table>
<thead>
<tr>
<th>Scenario</th>
<th>Area of concern</th>
</tr>
</thead>
<tbody>
<tr>
<td>Embalming</td>
<td><strong>High</strong> especially at sites where there is a lack of or inappropriate local exhaust ventilation system</td>
</tr>
<tr>
<td>Forensic/hospital mortuaries and pathology laboratories</td>
<td><strong>High</strong> especially in rooms without local exhaust ventilation, with windows shut, and with large specimen storage</td>
</tr>
<tr>
<td>Formaldehyde resin manufacture</td>
<td><strong>Medium</strong> during abnormal operation and where there is a need to break open or enter the enclosed system</td>
</tr>
<tr>
<td>Repacking and formulation other than formaldehyde resin</td>
<td><strong>Medium</strong> during raw material weigh-up and transfer, open operating process (mixing, decanting etc.), equipment cleaning and maintenance</td>
</tr>
<tr>
<td>Formaldehyde manufacture</td>
<td><strong>Medium</strong> during abnormal operation and where there is a need to break open or enter the enclosed system</td>
</tr>
<tr>
<td>Use of resin products</td>
<td><strong>Medium</strong> when heated and/or in contact with high humidity, and certain modes of application that possibly generate high levels of formaldehyde in air</td>
</tr>
</tbody>
</table>
18. Risk Management

Based on the risk characterisation for the environment, general public and workers (Chapter 17), risk to human health is the major concern from exposure to formaldehyde. The critical human health effects are sensory irritation, skin sensitisation and carcinogenicity. The main public health risks are for sensory irritation in situations of high indoor air formaldehyde levels, such as mobile homes and relocatable buildings. The general public is also at risk of skin sensitisation by using numerous types of consumer products containing formaldehyde such as cosmetics. Occupational health risks for sensory irritation exist in some industries using high concentrations of formaldehyde products, such as embalming in funeral homes and pathology laboratories.

To minimise these risks, this chapter discusses current risk management controls and practices and identifies further actions that are needed for protecting the environment, general public and workers from exposure to formaldehyde. The further actions, together with information on relevant overseas risk management practices, assisted in formulating the recommendations, which are discussed in the Overview and Recommendations section of this report.

18.1 Environmental risk management

The major anthropogenic release of formaldehyde in Australia is into the atmosphere, primarily as a result of fuel combustion processes. Therefore, this section focuses on ambient air quality controls.

18.1.1 Current ambient air quality controls

Regulatory controls for hazardous air pollutants vary between countries. The most common regulatory controls employed worldwide include the setting of emission standards for stationary and mobile point sources of air pollution, overall emission reduction goals, national ambient air quality standards, action programmes targeting individual pollutants or specific environmental problems, and land-use regulations which can control the geographical areas in which air pollution sources (such as industries) can be situated. Many countries have established lists of priority air pollutants, however, only a few countries have set national ambient air quality standards for individual hazardous pollutants.

**Australia**

**Air quality initiatives**

The Australian Government Department of the Environment and Heritage (DEH) is currently implementing national policies and programs to reduce emissions from the transport, industry and residential sectors. National initiatives to reduce the impact of road transport on air quality include improving the emission performance of the Australian vehicle fleet by implementing fuel quality standards (see below), reducing in-service vehicle emissions, encouraging fuel efficient and environmentally friendly vehicles and technologies, and promoting use of alternative fuels. For industry, specific codes of practice are being
developed for spray painters/surface coaters, printers and dry cleaners to reduce evaporative emissions through the promotion and adoption of vapour recovery practices and techniques. DEH is also undertaking a number of initiatives designed to reduce wood smoke emissions. This work includes community education on the correct operating practices for wood heaters, seeking improvements to wood heater installation and emission standards, and sponsoring research to improve understanding of wood heater emissions.

A forecasting system, the Australian Air Quality Forecasting System (AAQFS), is being developed which predicts daily levels of photochemical smog, atmospheric particles and a range of other pollutants. Components of the model generate air quality forecasts for 25 pollutants (including formaldehyde) in urban and non-urban areas. This information will enable environment protection agencies and industry to test effectiveness of strategies to reduce air pollution and raise awareness of air quality as an environmental issue. EPA Victoria is providing methods for use by major Australian cities to calculate daily pollution emissions. The Bureau of Meteorology generates the high-resolution weather forecasts and CSIRO has created computer models to calculate pollution levels.

State and territory governments are undertaking similar initiatives to improve air quality by reducing emissions from a range of sources.

**Ambient air quality management**

Currently, there is no national ambient air standard for formaldehyde. The National Environmental Protection Council (NEPC), a statutory entity within the Environmental Protection and Heritage Council, sets national environmental objectives through the development of National Environment Protection Measures (NEPM). NEPM can comprise any combination of goals, standards, protocols or guidelines, which are then implemented in all Australian jurisdictions.

A NEPM for Air Toxics was proposed by the National Environment Protection Council (NEPC) in 2001, and was endorsed in April 2004 (NEPC, 2004). Air toxics include gaseous, aerosol or particulate pollutants that are present in the air in low concentrations, with characteristics such as toxicity or persistence, so as to constitute a hazard to human, plant or animal life. Formaldehyde is one of five priority hazardous air pollutants addressed in the NEPM.

The goal of the NEPM is “to improve the information base on ambient air toxics within Australia in order to facilitate the development of standards following a review of the NEPM within eight years of its making”. The stated objectives are to:

- facilitate collection of monitoring data for ambient air toxics in order to inform future risk assessments and the development of standards;
- establish a set of investigation levels which can be applied nationally to the five priority air toxics as benchmarks against which the quality of ambient air can be assessed; and
- establish nationally agreed methodologies for determining appropriate locations for air monitoring these air toxics, conducting monitoring, and reporting results of monitoring.
The NEPM includes investigation levels that will be incorporated into a guideline. These levels are intended to assist jurisdictions in the interpretation of monitoring data and to evaluate the nature and extent of any risk to health of the communities in the areas of the monitoring sites. The ‘investigation level’ set for formaldehyde in this NEPM is 40 ppb over an averaging period of 24 hours (NEPC, 2004).

Generally, state and territory governments manage ambient air quality through regulations relating to environmental protection, air quality improvement and/or pollution reduction policies. Examples of strategies include setting specific industrial emission limits, implementation of standards and codes of best practice for industrial processes, use of licence fees based on pollutant loads produced, land-use regulation, standards on emissions from solid fuel stoves and heaters and local planning approval requirement for installation of such appliances, vehicle emission testing, policies on reduced vehicle use, alternative fuel use policies, environmental tobacco smoke regulation, and bush clearing regulation.

An example of state ambient air quality management is the Victorian legal framework for protecting air quality by the Environment Protection Act 1970, which provides for the development of state environment protection policies (SEPPs). Two SEPPs regulate air quality: SEPP (Ambient Air Quality) and SEPP (Air Quality Management). Under the latter, industries are required to control emissions of pollutants by best available practices to achieve policy aims that include consideration of economic, social and environment issues in the management of emissions to the air environment (Victorian Government, 2001).

The SEPP sets two types of criteria for the assessment of emissions to the air environment – ‘design criteria’ and ‘intervention levels’. Design criteria are modelling tools to assess residual emissions from individual industrial premises after emission controls have been applied. They are used in the design stage of a facility and are not used for monitoring purposes. Intervention levels are applied to individual pollutants in relation to neighbourhood air quality and take into account cumulative sources of a pollutant within a local area (or neighbourhood). They are used with air monitoring data to assess whether air quality within a neighbourhood is acceptable, acting as triggers for possible further actions if the level is exceeded. Formaldehyde has a design criteria set at 40 $\mu$g/m³ over a 3-minute averaging time, and an intervention level set at 15 $\mu$g/m³ over a 1-hour averaging time. The intervention levels are risk-based levels adopted from the Texas Natural Resource Conservation Commission (TNRCC) Effects Screening Levels (EPA Victoria, 2001a, 2001b).

Another example of state ambient air quality management is in Western Australia (WA). With the aim of protection of human health and the environment, the Department of Environmental Protection and the Health Department of WA have embarked on a joint program to develop ambient air quality guidelines and guideline values for air contaminants of concern, with the overall objective of providing a framework and benchmark for the assessment and management of air contaminants in WA. As an interim approach for substances not listed in the NEPM for ambient air, WA adopts WHO guideline values (see below), with appropriate amendments to suit the WA context. Where there is no NEPM or WHO guideline, criteria from another jurisdiction will be adopted, once it has been assessed and determined to be applicable to the WA context (DEP, 2003).
**Overseas**

Only a limited number of overseas countries and international organisations have established ambient air standards for formaldehyde, which are summarised in Table 18.1a.

**Table 18.1a: Overseas ambient air guidelines**

<table>
<thead>
<tr>
<th>Country</th>
<th>Guideline concentration ((\text{mg/m}^3))</th>
<th>Average period</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>WHO</td>
<td>100</td>
<td>30-minute</td>
<td>WHO, 2000a</td>
</tr>
<tr>
<td>California EPA</td>
<td>94 (Acute REL)</td>
<td>Not reported</td>
<td>CEPA, 1999</td>
</tr>
<tr>
<td></td>
<td>3 (Chronic REL)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>New Zealand</td>
<td>100</td>
<td>30-minute</td>
<td>ME&amp;MH, 2002</td>
</tr>
<tr>
<td>Sweden</td>
<td>12-60</td>
<td>Annual</td>
<td>EPA Victoria, 1999a</td>
</tr>
<tr>
<td>Netherlands</td>
<td>120*</td>
<td>Hourly</td>
<td>EPA Victoria, 1999a</td>
</tr>
</tbody>
</table>

REL = reference exposure level; *maximum permissible risk level

**World Health Organisation**

The World Health Organisation (WHO) has recommended a Guideline Value for formaldehyde in ambient air of 100 \(\text{mg/m}^3\) (as a 30-minute average). For the case of especially sensitive groups within the general population that show hypersensitivity reactions without immunological signs, WHO recommends that formaldehyde concentrations be kept to a minimum and not exceed the guideline value. WHO state that the guideline is set to prevent significant sensory irritation in the general population, and is based on the lowest concentration of formaldehyde associated with nose and throat irritation in humans after short-term exposure (WHO, 2000a).

WHO guidelines are intended as guides and are not standards in themselves. WHO notes that in moving from guidelines to standards for a particular nation or region, prevailing exposure levels and environmental, social, economic and cultural conditions should be taken into account. The guidelines are considered to protect health and safety and the guidelines do not differentiate between indoor and outdoor exposure (WHO, 2000a).

**United States**

Formaldehyde is regulated federally in the United States under the Clean Air Act (CAA), which has designated the chemical as a hazardous air pollutant (HAP). The Act imposes national emission standards for HAPs in the form of source-specific technology-based control requirements i.e., technology-based standards. These emission reduction standards are set according to what is considered financially and technologically plausible, rather than being based on safe levels determined by human toxicological data. In July 2004, the US Environmental
Protection Agency (US EPA) promulgated a rule to reduce emissions of toxic air pollutants, including HAP, from facilities that manufacture plywood and composite wood products (US EPA, 2004). The purpose of the rule is to protect public health by reducing emissions of HAP from these wood-based products. The rule regulates total HAP, rather than individual air pollutants, which is the sum of the emission of six primary HAPs (including formaldehyde) emitted from manufacturing of different types of plywood and composite wood products.

The CAA allows for the setting of national ambient air quality standards, however, no national ambient air standard has been set for formaldehyde. Ambient air quality of formaldehyde is also regulated federally through mobile source emission controls enacted under the CAA. Formaldehyde, together with benzene and 1,3-butadiene, were singled out for consideration for studying the need and feasibility of controlling emissions of toxic air pollutants associated with motor vehicles and their fuels. Standards are to be set based on available technology, taking existing standards, costs, noise, energy and safety factors and will address at least benzene and formaldehyde (US EPA, 2003).

Several states in the United States have developed ambient air quality standards and different control strategies have been implemented. For example, the California Environment Protection Agency (CEPA) has established Reference Exposure Levels (RELs) for hazardous airborne substances. For formaldehyde, the acute REL has been determined to be 94 \(\mu g/m^3\) and is based on the protection of mild to moderate eye irritation (considered the most sensitive health endpoint) (CEPA, 1999). The acute REL is an exposure level that is not likely to cause adverse effects in a human population, including sensitive subgroups like asthmatics, for one hour on an intermittent basis. A chronic REL has been determined to be 3 \(\mu g/m^3\) based on a NOAEL of 32 \(\mu g/m^3\) and critical health effects of irritation, degenerative inflammatory and hyperplastic changes of the nasal mucosa in humans and animals (CEPA, 2000). Chronic RELs are intended to be protective for individuals exposed continuously over their lifetime, including periods of potentially increased susceptibility to adverse health effects, particularly during childhood and the later years of life. These RELs are then used in the health risk assessment process developed for California’s Air Toxics Hot Spot Program. The Hot Spots program aims to control point source emissions of air toxics and requires facilities, which are high priority sites determined by emissions of HAPs from individual sites, to perform detailed risk assessments which are then made available to the public (CEPA, 2002).

18.1.2 Other environmental controls

National Pollution Inventory

Formaldehyde was recently included in the list of chemicals monitored within the National Pollution Inventory (NPI) program administered by DEH. The NPI program was established in 1998 as a joint Commonwealth, state and territory initiative. Facilities using or handling more than 10 tonnes of formaldehyde are required to report their emissions to air, land and water (NPI, 2005b).

DEH developed a NPI Fact Sheet for formaldehyde in 2002 and published it on their website (DEH, 2002). This document should be reviewed and updated using the data and findings of this assessment.
Waste management

Atmospheric discharges from industrial facilities are regulated by the state/territory environment authority under license agreements. For large companies, the license requires measurement and assessment of air quality. Companies manufacturing formaldehyde are required to calculate and report emissions to the state/territory environment authority and NPI.

Formaldehyde contaminated effluent is released to sewer under licensed trade waste agreements. A trade waste agreement is a commercial contract between the discharging company and the government authority, stating the terms and conditions to be observed to discharge waste into the sewerage system. Trade waste agreements generally allow concentrations of formaldehyde between 50 to 200 mg/L to be disposed of via the municipal treatment plant, depending on the jurisdiction and the capacity of the treatment system.

National fuel standards

The Fuel Quality Standards Act 2000 (the Act) and the Fuel Quality Standards Regulations 2001 establish national standards for fuels and a framework for enforcing them. The legislation, which was fully enforceable from 1 January 2002, helps Australia to reach international fuel quality benchmarks. The new standards enable the more effective operation of petrol and diesel vehicle engines. Standards for other fuels, such as liquefied petroleum gas, compressed natural gas and biodiesel, are also being developed.

The standards regulate the supply of fuel to consumers, reduce toxic vehicle emissions and ensure that, by using clean fuels, modern vehicles fitted with advanced emissions control technologies operate at peak performance. It is anticipated that the standards will have a major impact on the amount of toxic pollutants in vehicle emissions. Reduction of overall emissions and of the pollutants, which are involved in secondary formation of formaldehyde, should also reduce formaldehyde release into the atmosphere.

Water quality guidelines

The Australian and New Zealand Guidelines for Fresh and Marine Water Quality (ANZECC & ARMCANZ, 2000) provide threshold values for a range of chemical toxicants in water used for specific purposes. The guidelines target water intended for human consumption (i.e. drinking water), aquatic ecosystems, primary industry, and recreational and aesthetics uses. There are two guidelines for threshold levels of formaldehyde: drinking water and water for use in primary industry. Drinking water quality guidelines set formaldehyde concentrations at 10 mg/L. Water used in primary industry (i.e. aquaculture) must not exceed formaldehyde concentrations of 95 mg/L, to prevent tainting of fish flesh or the flesh of other aquatic organisms intended for human consumption.

18.1.3 Further actions identified

It is considered that there are sufficient risk management guidelines in place in existing codes, standards and regulations to address aspects of environmental protection from exposure to formaldehyde. Enforcement of the new national standards for fuels should help to reduce formaldehyde emissions from motor vehicles over the longer term.
The data and findings of this report should be taken into consideration by the National Environment Protection Council (NEPC) when setting an ambient air standard for formaldehyde. Based on the available human health data, a value of 80 ppb (short sampling duration) would be appropriate.

18.2 Public health risk management

18.2.1 Current indoor air quality management

Australia

Policy initiatives

There have been several policy initiatives and major reviews addressing indoor air quality in recent years. These include:

- A review, prepared in 1997 for the Department of Environment, Sport and Heritage (now the Australian Government Department of the Environment and Heritage) as a technical paper to the State of the Environment Report 1996, presented an analysis of the state of indoor air quality in Australia (Brown, 1997);

- A National Environmental Health Strategy was released by the Department of Health and Aged Care in 1999. It aimed to improve national environmental health management by providing a framework to bring stakeholders together across the range of issues that encompasses environmental health (DOHAC, 1999). The enHealth Council was established to provide national leadership and an implementation plan was developed in 2000 which identified a range of activities at the national level. Among these, indoor air quality was identified as an actionable issue (enHealth Council, 2000);

- The Living Cities – Air Toxics Program (ATP) was established in 1999 by the then Department of Environment, Sport and Heritage to address priority urban air pollution issues and to support the development of a national strategy to monitor and manage air toxics. It highlighted indoor air quality as an emerging issue. A State of Knowledge report on Air Toxics and indoor air quality in Australia, intended as an information source for discussions on management options, was a major outcome of the first step of the ATP (EA, 2001). Formaldehyde was discussed in this report. The next step of the program is that the Australian Government Department of the Environment and Heritage (DEH) will be developing strategies to improve indoor air quality in consultation with the Australian Government Department of Health and Ageing and state and territory governments. One avenue for achieving this is through the reduction of emissions of indoor air pollutants, either by eliminating their sources or by minimising the emissions from those sources. The Air Quality section of the DEH is currently conducting an indoor air project aiming to review the current situation and to identify priority issues and possible management responses (DEH, 2004).
A strategy for action was prepared by the Clean Air Society of Australia and New Zealand (CASANZ, 2002) and a number of steps were recommended to be taken to address the issue of indoor air pollution.

All the above strategies and reviews recommend the establishment of a single government body with responsibility for indoor air quality. Other recommendations included establishing indoor air standards for the most common and serious pollutants, reduction of emissions of indoor air pollutants, harmonisation of occupational, environmental and public health standards, provision of ‘green’ labelling, increased ventilation rates and vehicle exhaust reduction strategies.

**Current activities to address indoor air issues**

There are no national standards for indoor air quality in the non-occupational environment and the responsibility for indoor air quality is not centralised in one authority. Indoor air quality is being addressed through a range of diverse activities, including guidelines, standards and building codes, state and territory government activities, and community education.

At a national level, guidelines, such as the national health guidelines prepared by Australia’s National Health and Medical Research Council (NHMRC), standards, such as those developed by Standards Australia, and certain codes, such as the Building Code of Australia (available at [www.abcb.gov.au](http://www.abcb.gov.au)), apply to indoor air quality in general.

The NHMRC recommended an indoor air quality guideline for formaldehyde in 1982. Formaldehyde became of interest to the NHMRC at that time due to concerns about urea-formaldehyde foam insulation products. Based on a review of the literature and available scientific evidence on potential health effects, including eye and respiratory tract irritation and reported carcinogenic effects in animals, an indoor air quality goal (ceiling limit) of 120 μg/m³ (100 ppb) for formaldehyde was recommended (NHMRC, 1982, 1983). However, this standard has not been implemented. The Environment Protection and Heritage Council (EPHC) considers air quality issues at a national level, however, indoor air is not currently designated as a priority project (EPHC, 2003).

Standards Australia has published a standard methodology for measuring formaldehyde levels in indoor air, AS 2365.6-1995 *Methods for the Sampling and Analysis of Indoor air – Determination of Formaldehyde –Impinger Sampling - Chromotropic Acid Method* (Standards Australia, 1995). Details are discussed in Section 6. Standards Australia has also published standards for formaldehyde emission requirements for particleboard and MDF. Details are discussed later in this section.

Australian Building Codes Board (ABCB), a joint body of all levels of government, is responsible for overseeing the Building Code of Australia (BCA) to ensure that community expectations for health, safety and amenity in the design, construction and use of buildings through building codes, standards and regulations are met. BCA specifies the Australian Standards (or equivalent) that must be met for construction of buildings, for example, structural requirements. State/territory legislations then call up the BCA. The Standards that are referenced by BCA in relation to pressed wood products are AS 1860 1998 - Installation of particleboard flooring and AS/NZS 2269 1994 - Plywood –
structural. The standards for formaldehyde emission limits are not referenced in BCA because they are product standards. In addition, state/territory legislations that call up the BCA generally do not include mobile homes and relocatable buildings in their definition of a “building”.

The Australian Government Department of Transport and Regional Services (DTRS) is responsible for setting Australian Design Rules for vehicles including caravans. They generally adopt international standards that are based on performance in regards to road safety, such as the performance of tyres, towing bars and electrical equipment. Caravans in caravan parks are not covered by these design rules.

Other government organisations, such as the Department of Treasury and the Department of Health and Ageing, also have a role in indoor air quality issues in regards to policy making for consumer product safety, including labelling and consumer awareness.

States and territories do not have specific legislation regulating indoor air quality, although various performance-based regulations impact on indoor air quality. Examples are building regulations that cite the Building Code of Australia, which references Australian Standards on openable windows and mechanical ventilation (AS 1668.2–1991 The Use of Mechanical Ventilation and Air-Conditioning in Buildings – Mechanical Ventilation for Acceptable Indoor Air Quality (Standards Australia, 1991)). This standard sets minimum requirements for preventing an excess accumulation of airborne contaminants, or objectionable odours based on needs to control body odour, food odour, air contaminants, or carbon dioxide concentrations. Another example, Education and Facility Research Group in NSW Department of Commerce, sets up standards for school facilities for new and upgraded classrooms including relocatable classrooms. One of the requirements is that MDF is not permitted for wall lining or backing to pin boards in classrooms.

**Overseas**

There is no consistent approach to management of indoor air quality between countries. The most common activities undertaken include research, provision of information, education and training, encouragement of incentive schemes (such as environmental labelling), and emissions control. Generally, countries have no specific legislation for indoor air quality, and usually no single agency with full responsibility for the issue. As in Australia, regulations whose primary purpose is other than indoor air quality are the ones that address and impact on indoor air quality, e.g. building codes, consumer product safety regulations, hazardous chemical regulations. However, the WHO has been active in encouraging the national development of indoor air quality policies through documents, such as ‘Strategic Approaches to Indoor Air Policy-Making’ (WHO, 1999) and a charter on the ‘Right to Healthy Indoor Air’ (WHO, 2000b), which provide guidance on policy-making.

Very few countries have established limit values for individual indoor pollutants. A paper by Maynard (2000) listed the countries that have established indoor limits for formaldehyde (Table 18.1b).
Table 18.1b: Overseas indoor air guidelines

<table>
<thead>
<tr>
<th>Country</th>
<th>Guideline concentration</th>
<th>Averaging period</th>
</tr>
</thead>
<tbody>
<tr>
<td>Canada</td>
<td>120 µg/m³</td>
<td>5 min.</td>
</tr>
<tr>
<td></td>
<td>For carcinogenicity:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>120 µg/m³ 'Action Level'</td>
<td></td>
</tr>
<tr>
<td></td>
<td>60 µg/m³ 'Target Level'</td>
<td></td>
</tr>
<tr>
<td>Germany</td>
<td>100 ppb (120 µg/m³)</td>
<td>Not stated</td>
</tr>
<tr>
<td>Norway</td>
<td>100 µg/m³</td>
<td>30 min.</td>
</tr>
<tr>
<td>Poland</td>
<td>50 µg/m³</td>
<td>24 h</td>
</tr>
<tr>
<td></td>
<td>100 µg/m³</td>
<td>8-10 h</td>
</tr>
</tbody>
</table>

1 the lowest concentration considered to be feasible at the present time.
2 the value that every effort should be made to reduce concentrations so that they are below it.

**Canada**

Canada established a voluntary guideline for indoor residential air quality for formaldehyde in 1987 (see Table 18.1b). The guidelines are not mandatory or enforceable, but have been approved by government departments in Canada. The guideline is considered to be the lowest level practical at which there appears to be no undue public health effects. The target value is a longer-term objective, given for carcinogenic or potentially carcinogenic substances. It recognises that a continuing effort should be made to reduce exposure to the lowest possible level, and that the goal must be considered in light of the cost and feasibility of remedial measures and technological changes. Health Canada is currently reviewing this guideline based on a review of the new data on health effects for formaldehyde.

**United States**

No indoor air quality guideline values have been established by the US federal government. Regulation of indoor air quality in the US is not centralised in a single agency. Rather, various federal and state agencies and non-governmental organisations have set their own standards. For example, in 1985, the US Department of Housing and Urban Development (HUD) established a national indoor air standard of 400 ppb for formaldehyde in manufactured homes (US CPSC, 1997). In addition, since 1985 manufactured homes must be constructed with materials that meet formaldehyde emissions limits set by HUD (see Section 18.2.1).

In California, two exposure guidelines are given: an Action Level exposure guideline, the lowest concentration considered to be feasible at the present time for formaldehyde, is set at 100 ppb (120 µg/m³), and a Target value has been set at 50 ppb (60 µg/m³) (no average testing period stated).
18.2.2 Formaldehyde emission controls from wood products

**Overseas**

**Europe**

European industry has generally adopted a formaldehyde emission classification system for particleboard and MDF that was set by Germany in 1980 and was updated in 2003 (EN312:2003 and EN612-1:2003, respectively). Under this system, products are classed E1 and E2 according to emission results obtained by a specified test method. This system has now evolved and is based on Small Chamber Test, standardized as ISO/CD 12460, which determines an equilibrium formaldehyde concentration \( (\text{mg/m}^3) \). A simpler “perforator” method (European Standard DIN EN 120) was developed to provide a more practical and rapid classification. The European product classification system is shown in Table 18.2.

**Table 18.2: Classification of particleboard and MDF in Europe**

<table>
<thead>
<tr>
<th>Class</th>
<th>Equilibrium formaldehyde concentration* ( (\text{mg/m}^3) )</th>
<th>Perforator Method ( (\text{mg/100g}) )</th>
</tr>
</thead>
<tbody>
<tr>
<td>E1</td>
<td>$\delta 124$</td>
<td>$\delta 8$</td>
</tr>
<tr>
<td>E2</td>
<td>$&gt; 124$</td>
<td>$&gt; 8-30$</td>
</tr>
</tbody>
</table>

*Equilibrium formaldehyde concentration is based on Small Chamber Test protocol ISO/CD 12460.

A number of European countries have established regulations on use of some wood-based products based on formaldehyde emission limits (EC, 2003) and they are summarised in Table 18.3.

**Table 18.3: Formaldehyde emission limits for wood-based products in European countries**

<table>
<thead>
<tr>
<th>Country</th>
<th>Regulation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Austria</td>
<td>Derived timber products, i.e. chipboards, coated chipboards, etc. shall not be placed on the market if, at equilibrium, the formaldehyde concentration caused by the derived timber product in the air of a test room exceeds 0.1 ppm.</td>
</tr>
<tr>
<td>Denmark</td>
<td>Particleboards, other wood-based products and insulation foam emitting formaldehyde - the maximum emitted formaldehyde is 0.15 mg/m³ measured in a test room of 225 litres under standard conditions.</td>
</tr>
<tr>
<td>Finland</td>
<td>Particleboards, other wood-based products, furniture and insulation foam emitting formaldehyde - the formaldehyde content of room air is not allowed to be higher than 0.15 mg/m³ in air measured according to the Finnish standard SFS 3862.</td>
</tr>
<tr>
<td>Germany</td>
<td>Coated and uncoated derived timber products (particleboard, wood core plywood, veneered board and fibreboard) may not be placed on the market if the equilibrium concentration of formaldehyde resulting from the derived timber products in the air of a test chamber exceeds 0.1 ppm.</td>
</tr>
<tr>
<td>Sweden</td>
<td>Wood-based boards must not be placed on the market, transferred or used if the emission limit of 0.13 mg/m³ is exceeded when tested in a 1 m³ test room.</td>
</tr>
</tbody>
</table>

In Europe, phenolic bonded products are exempt from testing of emission levels under the Harmonised European Standard prEN 13986. Under this standard,
phenolic bonded products may be labelled with an E1 emission level without any requirements for testing.

**Japan**

The Japan Agricultural Standards (JAS) and Japan Industrial Standards (JIS) have set formaldehyde emission limits for wood-based products. These limits, together with signs for labelling of products, were revised in 2003 (JPIC, 2004) and are shown in Table 18.4.

**Table 18.4: JAS/JIS formaldehyde emission limits for wood-based products**

<table>
<thead>
<tr>
<th>Sign</th>
<th>Former Limits</th>
<th>Revised Limits</th>
<th>Equivalent to the EU system</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Formaldehyde emission</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Average</td>
<td>Maximum</td>
<td>(star system)</td>
</tr>
<tr>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Fc0</td>
<td>&lt;0.5 mg/L</td>
<td>&lt;0.7 mg/L</td>
<td>F(3 stars)</td>
</tr>
<tr>
<td>Fc1</td>
<td>&lt;1.5 mg/L</td>
<td>&lt;2.1 mg/L</td>
<td>F(2 stars)</td>
</tr>
<tr>
<td>Fc2</td>
<td>&lt;5 mg/L</td>
<td>&lt;7 mg/L</td>
<td>F(1 star)</td>
</tr>
</tbody>
</table>

# Using Desiccator test method

The Ministry of Land, Infrastructure & Transportation (MLIT) in Japan is responsible for overseeing enforcement of Building Standards Law (BSL). In response to public health concerns associated with the “sick house” syndrome, BSL promulgated amendments to the regulation concerning formaldehyde emission from building materials that are used in interior finishing in July 2002. These materials include plywood, wooden flooring, structural panels, glued laminated lumber, LVL, MDF, particleboard, urea resin board, wallpaper and adhesives. In July 2003, the MLIT implemented the new regulations and the details of the restrictions are summarised in Table 18.5. The amendments to the BSL also included mandatory installation of ventilation equipment and restrictions related to ceiling cavities.

**Table 18.5: Japanese Building Standard Law restrictions on interior finishing materials**

<table>
<thead>
<tr>
<th>Name</th>
<th>Building Materials Stipulated in the BSL</th>
<th>Restrictions#</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>JIS/JAS systems</td>
<td></td>
</tr>
<tr>
<td>Type 1 Formaldehyde emitting building materials</td>
<td>F (1 star)</td>
<td>Use prohibited</td>
</tr>
<tr>
<td>Type 2 Formaldehyde emitting building materials</td>
<td>F (2 stars)</td>
<td>Limited area of use*</td>
</tr>
<tr>
<td>Type 3 Formaldehyde emitting building materials</td>
<td>F (3 stars)</td>
<td>Limited area of use*</td>
</tr>
<tr>
<td></td>
<td>F (4 stars)</td>
<td>No restrictions</td>
</tr>
</tbody>
</table>

# There are no restrictions on materials which have been used as parts of buildings for > 5 years. + The area size of type 2 and 3 formaldehyde emitting building materials which can be used as interior finishing materials are restricted according to the type of habitable room and the frequency of ventilation. JAS, Japanese Agricultural Standard; JIS, Japanese Industrial Standard.
United States

Standards for certain wood products used in the installation of manufactured homes were established by the US Department of Housing and Urban Development (HUD) in 1985. A limit of 0.2 ppm measured by a specified air chamber test method is set for plywood emissions and 0.3 ppm for particleboard emissions (HUD, 1999). The HUD has permitted only the use of plywood and particleboards that conform to the above specified formaldehyde emission limits in the construction of prefabricated and manufactured homes since then. Phenolic bonded plywood products are exempt from certification of formaldehyde emission levels in the US under Section II.C.3 of HUD Rule 24 CFR 3280, as the emissions are considered too low to be significant. No changes to these standards were made by a review in 2002 (http://frwebgate1.access.gpo.gov/, accessed 20/06/03).

Australia

Australian Standards and industry compliance

Australian Standards set specifications for the manufacture and application of reconstituted wood-based panels and particleboard flooring. These standards specify that products should be classed and marked according to their formaldehyde emission potential. The Australian Standards set out two alternative sets of requirements for classification of Australian wood-based products for formaldehyde emissions, corresponding to its two recognised test methods:

1) AS/NZS 4266.16:2004 Reconstituted Wood-based Panels - Methods of Test. Method 16 - Formaldehyde Emission - Desiccator Method; (Standards Australia/Standards New Zealand, 2004a) and

These requirements are summarised in Table 18.6, which shows that the Australian E1 and E2 limits using the Perforator test method are similar with the recommended E1 & E2 European standards.

Standards Australia is considering introducing lower emission limits for these types of products (< 0.5 mg/L using Desiccator method, equivalent to the Japanese 3 star limit). In addition, the European Council is working on the harmonisation of test methods including methods that determine formaldehyde content/release, such as the Perforator method, Chamber method and gas analysis method, for consistency with the ISO (EC, 2003). The Australian wood panel industry has advised that they have actively participated in the harmonisation activity. Currently, the Australian Standards recognise the Desiccator Test method and work is progressing to formalise the adoption of the ISO Small Chamber Test.
There are no standards for formaldehyde emissions from plywood products. Standards Australia is currently developing a standard for structural laminated veneer lumber (LVL), with a limit of formaldehyde emission of 0.5 mg/L (equivalent to the Japanese 3 star limit). Standards for structural plywood, interior, exterior and marine plywood will be set in near future. Meanwhile, the Plywood Association of Australia (PAA) has drafted an industry voluntary standard, adopting the Japanese limits, for the monitoring and labelling of formaldehyde emissions from plywood and other veneer based wood products that are not covered in the proposed Australian standards. PAA claims that they will implement the proposed voluntary standards and believes that over 90% of products would meet the most stringent 4 stars rating in a short time period.

Currently, PAA claims that all LVL and approximately 98% of plywood manufactured in Australia complies with the formaldehyde emission limits that are equivalent to or lower than the E1 level. This is done under a quality control program operated since 1963. This program combines process quality control and end product testing carried out by manufacturers and by a laboratory of PAA which is registered with the National Association of Testing Authorities (NATA). In 1996, a product certification scheme was introduced which was recognized by the Joint Accreditation System of Australia and New Zealand (JAS-ANZ), a government appointed quality control accreditation body. Plywood and LVL products certified by the PAA are branded with the PAA product certification stamp as well as the JAS-ANZ mark to show purchasers that the product meets relevant standards. A sample of the certification stamp can be found in Appendix 11. Currently, PAA is considering adopting the Japanese star system for product labelling.

The Australian Wood Panels Association (AWPA) states that the wood panel industry and resin suppliers have participated in formaldehyde testing and reduction programs since 1983. All wood panel companies are required to provide samples to an accredited laboratory within AWPA for testing. AWPA stated that all Australian manufactured particleboard and MDF products now

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**Table 18.6: Australian Standards for formaldehyde emissions from reconstituted wood-based products**

<table>
<thead>
<tr>
<th>Australian Standard</th>
<th>Product</th>
<th>Class</th>
<th>Requirement</th>
</tr>
</thead>
<tbody>
<tr>
<td>AS/NZS(^3) 1859.1-2004</td>
<td>Particleboard</td>
<td>E1</td>
<td>(\delta 1.5)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>E2</td>
<td>(&gt;1.5 - 5.4)</td>
</tr>
<tr>
<td>AS/NZS(^4) 1859.2-2004</td>
<td>Dry processed fibreboard (including MDF)</td>
<td>E1</td>
<td>(\delta 1.0)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>E2</td>
<td>(&gt;1.0 - 3.3)</td>
</tr>
<tr>
<td>AS/NZS(^5) 1860.1-2002</td>
<td>Particleboard</td>
<td>E1</td>
<td>(\delta 1.8)</td>
</tr>
<tr>
<td></td>
<td>flooring</td>
<td>E2</td>
<td>(&gt;1.8 - 5.4)</td>
</tr>
</tbody>
</table>

\(^1\) AS/NZS 4266.16: 2004 (Standards Australia/Standards New Zealand, 2004a)
\(^2\) AS/NZS 4266.15:1995 (Standards Australia/Standards New Zealand, 1995)
\(^3\) AS/NZS 1859.1-2004 (Standards Australia/Standards New Zealand, 2004b)
\(^4\) AS/NZS 1859.2-2004 (Standards Australia/Standards New Zealand, 2004c)
\(^5\) AS/NZS 1860.1-2002 (Standards Australia/Standards New Zealand, 2002)
meet the E1 limit. A product certification scheme similar to that for plywood products, is being used for particleboard and MDF products. A sample label of certification scheme can be found in Appendix 12 (A). Details of the reduction in formaldehyde emission levels of some Australian made wood panel products over the last 15 years were provided by the AWPA and are presented in Appendix 12 (B).

The emission status of all imported wood-based products is not known. Some importers of wood-based products state that the imported products meet international standards in regard to formaldehyde emission levels according to information from their suppliers. For example, wall panel and industrial panel plywood products from South-Eastern Asia reportedly meet the International Wood Products Association (IHPA) standards (1997) which are equivalent to E2 level using chamber method.

18.2.3 Product labelling schemes

Product labelling schemes have been developed in many countries to assist consumers in choosing “environmentally friendly”, low-emitting products to promote healthy indoor air environment. They have been supported either by environmental or health government agencies or by voluntary industry initiatives. Two examples of overseas labelling schemes that address formaldehyde emissions are the German ‘Blue Angel’ scheme and the US Carpet and Rug Institute scheme. The former, established in 1977 by the German Federal Environmental Agency, has set labels for low-emission composite wood panels that include formaldehyde-containing binding agents, and a label for low-emission wood products and wood-based products. To gain these labels, products must not exceed a concentration of 0.05 ppm for formaldehyde in the test room under specified testing conditions (RAL, 2003). The US Carpet and Rug Institute established a green labelling program in 1992, whereby manufacturers’ samples are tested and attached with a certified ‘green label’ if they meet specified limits. The current criteria for formaldehyde emissions from carpets is 0.05 mg/m²/hr (CRI, 2004).

An international standard for environmental marketing claims was developed by ISO in 1998 (ISO/DIS 14021.2). This was followed by an international best practice guide for formal eco-labelling schemes (ISO 14024) where use of an eco-labelling symbol is allowed when the scheme’s stated environmental criteria are met. In Australia, the Joint Standards Australia/Standards New Zealand Committee on environmental labelling revised the ISO standard with national modifications in 2000 and designated AS/NZS ISO 14021:2000 (Standards Australia/Standards New Zealand, 2000b). The Australian Environmental Labelling Association (AELA), an independent environmental scientific research and assessment organisation, established an Australian Ecolabel Program in 2001 as an environmental labelling program for Australia in conformance to ISO 14024. This program is the Australian member of the Global Ecolabelling Network and awards Ecolabels to products that meet or exceed voluntary environmental standards for environmental performance. Standards are established by the AELA after stakeholder consultation and assessment of market needs. A number of standards related to formaldehyde have been set and they are:

- Wool pile carpet - a formaldehyde limit of 0.01 mg/m³ of air per 1m² new carpet at the point of despatch from the factory;
Laundry and hand dishwashing detergents – shall not be formulated or manufactured with more than 0.1% by weight of formaldehyde or formaldehyde donors expressed as formaldehyde; and

Printing inks (draft) and gypsum plasterboard – shall not be formulated or manufactured with formaldehyde or have the potential to release formaldehyde during use.

AELA is developing a standard for indoor and outdoor furniture for household and commercial use. No standards have been set for wood products containing formaldehyde, although a standard for VOC in adhesive products was set as no more than 5% in weight of VOC. The adhesive products include wallpaper paste, adhesives for wall covering, flooring, tiles and other adhesives (paper, wood, office, plastic) (AELA, 2002).

It should be noted that one of the requirements for pressed wood products that are tested in accordance with the Australian Standards for formaldehyde emission limits is to label them to indicate formaldehyde emission levels.

18.2.4 Current risk management for consumer products

SUSDP

The Australian Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) (NDPSC, 2003) lists formaldehyde (excluding its derivatives) in Schedules 2 and 6, except in preparations containing ≤ 5% of formaldehyde. Schedule 2 is for human therapeutic use preparations and the required signal words required are ‘PHARMACY MEDICINE’. Schedule 6 is for poisons that are substances with a moderate potential for causing harm, the extent of which can be reduced through the use of distinctive packaging with strong warnings and safety directions on the label.

The labelling requirements for formaldehyde include the safety directions ‘Avoid contact with eyes’, ‘Avoid contact with skin’ and ‘Avoid breathing dust/vapour/spray mist’.

The recommended first aid instructions are:

- For advice, contact a Poisons Information Centre on 13 1126 or a doctor at once;
- If swallowed, do NOT induce vomiting;
- If in eyes, hold eyelids apart and flush the eye continuously with running water. Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes;
- If skin or hair contact occurs, remove contaminated clothing and flush skin and hair with running water; and
- If inhaled, remove from contaminated area. Apply artificial respiration if not breathing.

This schedule has been adopted by all jurisdictions. However, in light of recent upgrade on the carcinogenicity of formaldehyde and its known potency in causing
skin sensitisation, the current scheduling should be reviewed by the National Poison and Drugs Scheduling Committee (NDPSC).

**Cosmetics**

The majority of cosmetic products used in Australia contain $< 0.2\%$ free formaldehyde. However, some products, such as nail hardener, contain up to 1% formaldehyde. Overseas publications report the formaldehyde content of some cosmetics as high as 4.5% (in nail hardeners) and concentrations in dry skin lotions, crème rinses and bubble bath oil are in the range of 0.4% to 0.6% (IPCS, 1989).

There is no Australian standard limiting the amount of formaldehyde allowed in cosmetic products. The Australian cosmetic associations advised that the Australian cosmetics industry follows international practice based on the Cosmetic Ingredient Review (CIR) reports for formaldehyde (CIR Expert Panel, 1984) and formaldehyde donor products, such as DMDM Hydantoin (CIR Expert Panel, 1988). These reports concluded that the concentration of free formaldehyde should not exceed 0.2% and aerosolised cosmetic products containing formaldehyde should not be used. These reports have been reviewed recently and no changes have been made (CIR, 2003; CTFA, 2003).

In the EU, Annex VI (List of preservatives which cosmetic products may contain) of the Cosmetics Directive 76/768/EC (EC, 1999) requires that all finished products containing formaldehyde or substances listed in the Annex which release formaldehyde must be labelled with the warning ‘Contains formaldehyde’ where the concentration of formaldehyde in the finished product exceeds 0.05%. The maximum authorised concentration of free formaldehyde and paraformaldehyde is 0.2% in cosmetic products, except for oral hygiene products where the maximum concentration of free formaldehyde is 0.1%. Use of formaldehyde and paraformaldehyde in aerosol dispensers (sprays) are prohibited. Formaldehyde is also listed in Annex III of Cosmetics Directive 76/768/EC (a list of preservatives which cosmetic products must not contain except subject to the restrictions and conditions laid down due to toxicological concerns), which limits the maximum authorised concentration in nail hardeners to 5% (calculated as formaldehyde). The Annex also states that nail hardeners with $> 0.05\%$ formaldehyde as a preservative must carry the warning statement of ‘Protect cuticles with grease or oil. Contains formaldehyde’.

In Canada, formaldehyde is acceptable for use in non-aerosol cosmetics provided that it does not exceed 0.2%. In addition, the recommended limit for formaldehyde concentration in cosmetics is less than 0.3% except for nail hardeners, for which a maximum concentration of 5% is recommended (IPCS, 2002).

**18.2.5 Further actions identified**

**Indoor air**

Although the worst-case scenario risk estimation for respiratory cancer indicates a low risk (less than one in a million at $< 0.3$ ppm), it is prudent to eliminate or reduce formaldehyde exposure to the public wherever possible. In addition, the general public may be at risk of sensory irritation when exposed to high indoor air
formaldehyde levels. There are a number of ways to tackle this issue, such as setting an indoor air standard/guideline, formaldehyde source control and consumer awareness.

Due to lack of national indoor air standard for formaldehyde, an indoor air guidance value should be set, so that the results of monitoring studies can be considered and action taken where appropriate. A guidance value of 80 ppb is recommended. The critical health effect selected for deriving the guidance value is sensory irritation, with an identified LOEL of 0.5 ppm. Using the WHO approach for deriving guidance values for health based exposure limits (ICPS, 1994, 2005), the following uncertainty factors have been applied. The interspecies uncertainty factor is not applicable for formaldehyde, as the possible NOEL level was based on human studies. An uncertainty factor of 3.2 for interspecies variability to account for toxicodynamic differences between individuals is appropriate. The interspecies uncertainty factor of 3.2 for toxicokinetic is considered not applicable, as sensory irritation is a local effect. It is recommended that an additional uncertainty factor of 2 be applied to extrapolate from LOEL to NOEL. Thus, the overall uncertainty factor that is used for deriving the indoor air guideline value is 6.4 (3.2 x 2). Consequently, the recommended guideline value is determined to be 80 ppb (0.5 ppm/6.4). As formaldehyde is metabolised rapidly at site of contact and sensory irritation is an acute effect, the duration of sampling should be short. For example, the WHO ambient air standard has a sampling period of 30 minutes, also noting the sampling duration is usually 1 to 4 hours in the Australian Standard AS 2365.6-1995 (Standards Australia, 1995). It is important to note that as formaldehyde is classified as a Category 2 carcinogen, indoor air formaldehyde levels should be kept as low as practicable.

Regarding formaldehyde source control, the majority of pressed wood products made in Australia meet the lowest European formaldehyde emission limit (E1) which is equivalent to the Australian emission standards. It appears that in general imported pressed wood products are not tested and certified for formaldehyde emissions in Australia. The majority of these standards are not called up by the Building Code of Australia (BCA) because they are product standards. In addition, state/territory legislations that call up the BCA generally do not include mobile homes and relocatable buildings in their definition of a “building”. Therefore, manufacturers of mobile homes and relocatable buildings should use only materials that meet the Australian emission standards. In addition, Standards Australia should adopt and/or develop a standard for mobile homes and relocatable buildings including guidelines on ventilation and use of pressed wood products that meet the lowest Australian Standards formaldehyde emissions limit.

Raising consumer awareness is an important approach to addressing indoor air issues. Approaches should include publication of an Information Sheet to raise consumer awareness regarding minimising formaldehyde levels in indoor air as well as distribution of the information to mobile home owners and residents.

**Consumer products**

Although formaldehyde is listed on the SUSDP, NDPSC should consider more restrictive categories or cut-off values for consumer products including cosmetics, given its potency of causing skin sensitisation and potential carcinogenicity.
18.3 Occupational health and safety risk management

18.3.1 Current regulatory controls

Hazard classification

Formaldehyde is currently listed on the *Hazardous Substances Information System* (DEWR, 2004) and is classified as toxic by inhalation, in contact with skin and if swallowed (R23/24/25), causes burns (R34), limited evidence of a carcinogenic effect (Carcinogen, Category 3, R40), and may cause sensitisation by skin contact (R43).

Based on the human health effects assessed in this report, the current hazard classification for formaldehyde has been reviewed against the NOHSC Approved Criteria for Classifying Hazardous Substances (NOHSC, 2004). The classification of Carcinogen, Category 3 (limited evidence of a carcinogenic effect, R40) should be replaced with Carcinogen, Category 2 (may cause cancer by inhalation, R49). Classifications for the other health endpoints are confirmed in this assessment.

Occupational exposure standard

The current national occupational exposure standard for formaldehyde is 1 ppm (1.2 mg/m³), expressed as an 8 hour time-weighted average (TWA) airborne concentration, with a short-term exposure limit (STEL) of 2 ppm (2.5 mg/m³) for 15 minutes, and a sensitiser notation (NOHSC, 1995). This standard should be revised, as 1 ppm is higher than the LOEL for sensory irritation (0.5 ppm) based on the evaluation of health effects data in this assessment. Furthermore, formaldehyde is classified Category 2 Carcinogen by inhalation, therefore the level of exposure should be kept as low as possible.

The Australian exposure standard was adopted in 1990 from the American Conference of Governmental Industrial Hygienists (ACGIH) threshold limit value (TLV) established in 1985 (ACGIH, 1986). The original ACGIH documentation stated that available information indicated that irritation of eyes and nose occurs down to 1 ppm and symptoms also occur below 1 ppm. Therefore, the ACGIH considered that the TLV value of 1 ppm might not be low enough to prevent the hypersensitive person from either suffering irritation or complaints. In 1989, ACGIH proposed a TLV-ceiling level of 0.3 ppm (0.37 mg/m³) to further reduce the potential for sensory irritation for workers handling formaldehyde and formaldehyde-containing products (ACGIH, 1989). This proposal was adopted in 1992 (ACGIH, 2000a). The ceiling level designation was deemed appropriate due to the association of formaldehyde with rapid onset of irritation. The recommended limit of 0.3 ppm was based on evidence of irritation from reports of occupational exposure to formaldehyde as well as human formaldehyde exposures in other settings. In addition, ACGIH stated that the reported dose-dependent nasal squamous metaplasia observed in rats and monkeys, as well as the inadequate epidemiological data on the cancer risk to man, support maintaining exposures as low as practicable. In 2000, the ACGIH standard was revised by adding a notation for skin sensitisation (ACGIH, 2000b).

Current occupational exposure standards for formaldehyde in Australia and other countries are listed in Table 18.7.
The German Deutsche Forschungsgemeinschaft (DFG) reviewed their occupational exposure limits for formaldehyde in 2000 and assigned a maximum 8 h workplace value (MAK value) of 0.3 ppm (0.37 mg/m³). The chemical has also been assigned with Category 1 peak limitation (ceiling level), using an excursion factor of 2. This means that exposure levels should not exceed 0.6 ppm for any period longer than 5 minutes on more than 8 occasions per shift. In setting the new MAK value, the DFG took into account new data that confirmed a previous assumption that occurrence of tumours in the nasal mucosa of rats and mice may be the result of chronic proliferative processes caused by the cytotoxic effects of formaldehyde. The avoidance of cell proliferation through the irritation effect of formaldehyde on the upper respiratory tract was considered a decisive factor in setting the MAK value. They considered the database for irritation effects of formaldehyde on the upper respiratory tract insufficient to establish a MAK value, and therefore, set the level against a parameter for irritation of the eyes (a more sensitive measure). The value of 0.3 ppm was based on an extensive review by Pautenbach et al. (1997) of the literature investigating formaldehyde induced sensory irritation. This review found that daily exposures for 8 hours to maximum formaldehyde concentrations of 0.3 ppm did not result in eye irritation in nearly all workers.

Although UK HSE currently has maximum exposure limits (MELs) of 2 ppm for both 8 hour TWA and 15 minute STEL, it has been flagged that the formaldehyde exposure standard, together with 14 other chemicals, will be reviewed as part of the development of a new UK occupational exposure limit (OEL) framework (UK HSE, 2003).

**Atmospheric monitoring**

Under the National Occupational Health and Safety Commission’s (NOHSC) *Model Regulations and Code of Practice for the Control of Workplace Hazardous Substances* (NOHSC, 1994c), employers are required to carry out an assessment of the workplace for all hazardous substances. The methodology for a workplace assessment is provided in the NOHSC *Guidance Note for the Assessment of Health Risks Arising from the Use of Hazardous Substances in the Workplace* (NOHSC 1994b). When an assessment indicates the risk of exposure via inhalation is significant, atmospheric monitoring should be conducted to measure levels of the hazardous substance in the workplace as a precursor to the implementation of suitable control measures to reduce exposure. Subsequent monitoring will be required to ensure that such measures are effective. Analytical methods for the measurement of formaldehyde in air are detailed in Chapter 6.

Atmospheric monitoring programs for formaldehyde are in place at the four manufacturing sites. The NICNAS survey indicated that air monitoring was conducted at a small proportion of workplaces that use formaldehyde or formaldehyde products.

It should be noted that atmospheric monitoring might not provide an accurate estimate of total exposure in situations where significant dermal exposure occurs.
Table 18.7: Australia and overseas occupational exposure standards for formaldehyde (adapted from ACGIH, 2004)

<table>
<thead>
<tr>
<th>Country</th>
<th>TWA¹ (ppm, unless otherwise stated)</th>
<th>STEL² (ppm, unless otherwise stated)</th>
<th>CEILING (ppm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Australia</td>
<td>1</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Belgium</td>
<td></td>
<td>0.3</td>
<td></td>
</tr>
<tr>
<td>Brazil</td>
<td></td>
<td>1.6</td>
<td></td>
</tr>
<tr>
<td>China</td>
<td></td>
<td>0.5 mg/m³</td>
<td></td>
</tr>
<tr>
<td>Czech Republic</td>
<td>0.5 mg/m³</td>
<td>1 mg/m³</td>
<td></td>
</tr>
<tr>
<td>Finland</td>
<td>0.3</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Germany (MAK)</td>
<td>0.3</td>
<td>0.6</td>
<td>1</td>
</tr>
<tr>
<td>Hong Kong</td>
<td></td>
<td></td>
<td>0.3</td>
</tr>
<tr>
<td>Ireland</td>
<td></td>
<td></td>
<td>2</td>
</tr>
<tr>
<td>Japan - JSOH</td>
<td>0.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malaysia</td>
<td></td>
<td>0.3</td>
<td></td>
</tr>
<tr>
<td>Mexico</td>
<td></td>
<td></td>
<td>2</td>
</tr>
<tr>
<td>Netherlands</td>
<td>1</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>New Zealand</td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Norway</td>
<td>0.5</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Poland</td>
<td>0.5 mg/m³</td>
<td>1 mg/m³</td>
<td></td>
</tr>
<tr>
<td>Spain</td>
<td></td>
<td></td>
<td>0.3</td>
</tr>
<tr>
<td>Sweden</td>
<td>0.5</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>UK HSE (MEL)</td>
<td>2</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>US-ACGIH*</td>
<td></td>
<td></td>
<td>0.3</td>
</tr>
<tr>
<td>USA-NIOSH IDLH*</td>
<td></td>
<td></td>
<td>20</td>
</tr>
<tr>
<td>USA-NIOSH REL*</td>
<td>0.016</td>
<td></td>
<td>0.1</td>
</tr>
<tr>
<td>USA-OSHA PEL</td>
<td>0.75</td>
<td></td>
<td>2</td>
</tr>
</tbody>
</table>

¹ time-weighted average; ² short-term exposure limit; * not regulatory standards
ACGIH = American Conference of Governmental Industrial Hygienists (recommended limits); NIOSH = National Institute of Occupational Safety and Health (recommended limits); OSHA = Occupational Safety and Health Administration (statutory limits); MAK=Maximale Arbeitsplatz Konzentration (Maximum Workplace Concentration); MEL= Maximum Exposure Limit; IDLH=Immediately Dangerous to Life and Health; REL=Recommended Exposure Limits; PEL=Permissible Exposure Limit; JSOH=Japan Society for Occupational Health; UK HSE = UK Health and Safety Executive.
Health surveillance

In accordance with the NOHSC Model Regulations and Code of Practice for the Control of Workplace Hazardous Substances (NOHSC, 1994c), employers have a responsibility to provide health surveillance in those workplaces where the workplace assessment indicates that exposure to a hazardous substance may lead to identifiable substance-related disease or adverse health effects.

Formaldehyde is not listed in Schedule 3 (list of substances requiring health surveillance) and as such, there are no formal requirements for health surveillance for exposed workers. However, it was reported that health surveillance programs are in place at three formaldehyde manufacturing sites (Hexion Specialty Chemicals, Woodchem and Orica). All employees who routinely work around the plant or laboratory regularly undergo lung function tests, skin examination and an evaluation of prior and existing respiratory history.

Control of major hazard facilities

NOHSC has developed the Control of Major Hazard Facilities National Standard [NOHSC:1014(2002)] and National Code of Practice [(NOHSC:1014 (1996)] (NOHSC, 2002). A Major Hazard Facility is an area where an activity takes place involving a quantity of a material(s) which exceeds the threshold(s), as specified in Schedule 1 of the standard. Formaldehyde is listed in Schedule 1, with a threshold quantity of 50 tonnes.

The purpose of the standard is to prevent and minimise the effects of major accidents and near misses by requiring the person in control of the facility to:

- identify and assess all hazards and implement control measures to reduce the likelihood and effects of a major accident;
- provide information to the relevant public authority (state, territory or Commonwealth jurisdiction) and the community, including other closely located facilities, regarding the nature of hazards at a major hazard facility and the emergency procedures in the event of a major accident;
- report and investigate major accidents and near misses, and appropriate corrective action; and
- record and discuss the lessons learnt and the analysis of major accidents and near misses with employees and employee representatives.

The four formaldehyde manufacturers exceed the threshold quantity for a major hazard facility site. Three of them have registered with relevant state authorities and reported that a program is in place to control major accidents. Woodchem Australia Pty Ltd in NSW has notified the state authority that they may be a possible major hazard facility, however, the relevant legislation is being drafted in this state.

National storage and handling regulations

Formaldehyde meets the criteria for a dangerous good so national storage and handling regulations for dangerous goods are applicable. Storage and handling
requirements are described in the NOHSC National Standard for the Storage and Handling of Workplace Dangerous Goods (NOHSC, 2001a) and NOHSC National Code of Practice for the Storage and Handling of Workplace Dangerous Goods (NOHSC, 2001b).

Information provided by applicants and the NICNAS survey indicates that these requirements are met.

**National transport regulations**

The Australian Code for the Transport of Dangerous Goods (ADG Code) sets out requirements relating to the transport of dangerous goods by road and rail.

Formaldehyde is listed twice in the ADG Code, under UN numbers 2209 and 1198, and paraformaldehyde is listed under UN number 2213 (Table 18.8). The transport of formaldehyde gas is prohibited by the ADG Code (FORS, 1998). Non-flammable formaldehyde solutions with less than 25% formaldehyde, are not subject to the provisions of the ADG Code.

Information provided by applicants and the NICNAS survey indicates that the transport regulations have been complied by industries.

**18.3.2 Current industry controls**

According to the NOHSC National Model Regulations for the Control of Workplace Hazardous Substances (NOHSC, 1994c), exposure to hazardous substances should be prevented, or where that is not practicable, controlled to minimise risks to health. NOHSC’s National Code of Practice for the Control of Workplace Hazardous Substances (NOHSC, 1994c) lists the hierarchy of control measures, in priority order, that should be implemented to eliminate or minimise exposure to hazardous substances. These are:

- elimination;
- substitution;
- isolation;
- engineering controls;
- safe work practices; and
- personal protective equipment.

**Elimination and substitution**

Elimination is the removal of a chemical from a process and should be the first option considered when minimising risks to health. In situations where it is not feasible or practical, substitution should be considered. Substitution includes replacing with a less hazardous substance or the same substance in a less hazardous form.
Table 18.8: Summary of the information contained in the ADG Code

<table>
<thead>
<tr>
<th>UN Number</th>
<th>2209</th>
<th>1198</th>
<th>2213</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shipping name</td>
<td>Formaldehyde solution, with not less than 25% formaldehyde</td>
<td>Formaldehyde solution, flammable</td>
<td>Paraformaldehyde</td>
</tr>
<tr>
<td>Class</td>
<td>8 (Corrosive Substance)</td>
<td>3 (Flammable Liquid)</td>
<td>4.1 (Flammable Solid)</td>
</tr>
<tr>
<td>Subsidiary Risk</td>
<td>NA</td>
<td>8 (Corrosive Substance)</td>
<td>NA</td>
</tr>
<tr>
<td>Packing Group</td>
<td>III</td>
<td>III</td>
<td>III</td>
</tr>
<tr>
<td>Hazchem Code</td>
<td>2Z</td>
<td>2YE</td>
<td>1[Z]</td>
</tr>
<tr>
<td>Packaging Method</td>
<td>3.8.8</td>
<td>3.8.3</td>
<td>3.8.4.1</td>
</tr>
<tr>
<td></td>
<td>RT7 Toxic or corrosive liquid (density ≤1)</td>
<td>RT1 Flammable liquid</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>RT7 Toxic or corrosive liquid (density ≤1)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>RT8 Toxic or corrosive liquid (density &gt;1)</td>
<td></td>
</tr>
</tbody>
</table>

NA, not applicable; RT number refers to a particular kind of tank that is intended to form part of a road vehicle or to be attached to a road vehicle; Z, breathing apparatus (no risk of violent reaction); [Z], breathing apparatus for fire only; Y, breathing apparatus (with risk of violent reaction); E, when evacuation should be considered.

Information from industry submissions indicates that substitution or elimination of formaldehyde has been considered by some end users or formulators. For example, one company indicated that a substitute for the use of formalin solutions in leather tanning is being sought. Information from the CSIRO’s Department of Textile and Fibre Technology (CSIRO Leather Research Centre, 2004) indicates that use of formaldehyde or formaldehyde resins in leather tanning is declining. Some manufacturers of manufactured homes claimed that they have started to use products that do not contain formaldehyde. For example, wall plywood, wall laminates, MDF skirting and architraves are no longer used in recent manufacturing of luxury cabins. However, there are some manufacturers that still use laminate and plywood linings for budget cabins. It was reported that some pathology laboratories use formalin-free fixative products during specimen preparation. It was also reported that some consumer products (detergents and toilet disinfectant) have been reformulated to remove formaldehyde.

Formaldehyde users need to evaluate the technical issues, costs, health and safety and environmental effects of each option when considering substitution of formaldehyde. In particular, the human health and environmental effects of the substitute should be considered to ensure that formaldehyde is not being replaced by a more hazardous substance.

Overseas industries have also been trying to eliminate or substitute formaldehyde as far as possible. For example, an article by Cattarin (1997) indicated that the future direction of anatomical study at universities would be via interactive
computer learning and observation of specimens which are preserved using formaldehyde-free plastic resin. Some formaldehyde resin producers have been looking into techniques that neutralise free formaldehyde in the resin (Anon, 2004). Some hardwood plywood producers in the US have indicated that they will begin formaldehyde-free manufacturing processes (CFP, 2005).

Isolation

Isolation as a control measure aims to separate employees, as far as practicable, from the chemical hazard. This can be achieved by distance, use of barriers or enclosure. Isolation when handling formaldehyde products was reported by industry. For example, production areas are located away from control rooms and offices at some formaldehyde and resin manufacturing sites. It was reported that coating products containing formaldehyde are used in a separated coaters’ room. Formaldehyde products are often stored in special areas due to their flammability properties. At most formulation sites, isolation of the mixing process is achieved by either housing the mixing tank or containers in a separate workshop or operating at a distance from other activities.

Engineering controls

Engineering controls used in plants or processes minimise the level of hazardous substances at workplaces. They include enclosure or partial enclosure, local exhaust ventilation and automation of processes.

Manufacturers of formaldehyde and formaldehyde resins in Australia all reported enclosed and automated manufacturing processes. Local exhaust ventilation and general ventilation are used during sampling, manual loading and packaging.

Formulation processes vary in the degree to which the plant is enclosed. Of the methods reported, open mixing processes were the most common. Enclosed loading processes and automated decanting processes were reported by most formulators. Local exhaust ventilation is generally employed to ensure that the vapours are drawn away from the work area. Other types of ventilation, such as industrial fans and general ventilation, were also observed.

Best practice to be followed during formulation is total enclosure of the processes, such as transfer of formaldehyde to the mixing vessel through enclosed pipes, decanting products through closed pipelines, and use of a lid on the mixing vessel during mixing.

The types of engineering controls employed during end use of formaldehyde products vary at different sites, such as the extent of enclosure of the process and type of ventilation. Open processes are common for embalming and forensic/hospital mortuaries and pathology laboratories. The majority of these workplaces have ventilation systems in place, but some do not (see Section 15.6.3). All analytical laboratories reported use of fume hoods or ‘down draught’ extraction system. Enclosed and automated processes were reported in industries, such as film processing and leather tanning. Floor level or roof exhaust system and general ventilation were the most common engineering controls in end use industries. Engineering controls need to be evaluated to ensure they are efficient by methods such as air monitoring and airflow testing.
Safe work practices

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

Many safe work practices reported for formaldehyde relate to minimising the risks of its flammability. Among these are eliminating all sources of ignition and preventing accumulation of vapours in hollows or sumps.

Several safe work practices were reported as part of general procedures:

- Limited access to areas where formaldehyde products are manufactured or used;
- In the absence of local exhaust ventilation, use of formaldehyde products in a well-ventilated area;
- Written procedures for handling;
- Procedures to ensure workers read MSDS when using a chemical for the first time;
- Labelling/placarding of tanks;
- Methods to reduce exposure during sampling e.g. use of sampling tap; and
- Prompt cleanup of spills.

Personal protective equipment

Personal protective equipment (PPE) is used to minimise exposure to or contact with chemicals. As a general rule, PPE should be used where other control measures are not practicable or adequate to control exposure. PPE should be used in conjunction with engineering controls and not as a replacement.

For formaldehyde, PPE is primarily used to protect hands and to prevent face and eye splashes. It is usually combined with basic protection, such as boots and overalls. Aprons were reported to be used by several NICNAS survey respondents.

Gloves are generally provided at most workplaces. Types of gloves specified by some formulators and end users of formaldehyde products were PVC, nitrile, latex, impervious and rubber gloves. It is important to select gloves that are resistant to formaldehyde. Australian/New Zealand Standard AS 2161.1 (2000a) Occupational Protective Gloves Part 1: Selection, Use and Maintenance (Standards Australia/Standards New Zealand, 2000) provides guidance in selecting and use of protective gloves for handling hazardous substances. Glove manufacturers’ recommendations should be consulted when selecting protective equipment, and suitability may depend on the degree of contact with formaldehyde. For example, one compatibility table for formaldehyde rates laminated film, nitrile and unsupported neoprene glove materials as most resistant for heavy exposure (Ansell, 1998). Another source provides specific information on formaldehyde regarding permeation index numbers for permeation rates and breakthrough times for chemical protective clothing including gloves, coveralls and suits which may help industries in selecting appropriate protective clothing (Forsberg & Mansdorf, 1997).
Safety glasses and goggles were the most commonly reported eye protection. Face shields were also used or available at some workplaces.

Information from industry submissions and the NICNAS survey indicates that respiratory protection equipment is available at many workplaces, but is generally not used during daily operation. They are used where exposures are likely to be high, such as manual drum filling, entering into a confined space and dealing with major spills. The types of respiratory protection equipment used vary from site to site and range from disposable half-face mask to full-face, air-supplied respirators depending on the task and potential for exposure to formaldehyde. It was reported that full-face air-supplied respirators or breathing masks are worn for working in confined spaces, opening pipelines containing formaldehyde and dealing with major spills. They are also worn during loading and transferring formaldehyde or paraformaldehyde powder at four resin manufacturing and formulation sites. Half-face respirators with organic vapour/dust filters are used during connection of intermediate bulk containers to automated dosing system for transfer of bulk formalin at one site. Some formulators use respirators with inorganic and particulate filter during mixing. Airflow helmet with K1 cartridge filter is used during sterile area fumigation. Respirators are also used in aerial film processing during drum changing and some embalming sites. However, information from state/territory occupational health and safety authorities indicates that some respiratory protection equipment used at workplaces is not properly maintained.

It is important to use appropriate respiratory protection equipment where exposures are likely to exceed the occupational exposure standard. Australian/New Zealand Standard AS/NZS 1715: 1994 - Selection, Use and Maintenance of Respiratory Protective Devices (Standards Australia/Standards New Zealand, 1994) provides guidance in selecting and using respirators for handling hazardous substances. Also, respiratory protection equipment needs to be maintained properly.

**MSDS**

MSDS are the primary source of information for workers handling chemical substances. Under the NOHSC National Model Regulations for the Control of Workplace Hazardous Substances (NOHSC, 1994c) and the corresponding state and territory legislation, suppliers are obliged to provide an MSDS to their customers for all hazardous substances. Employers must ensure that a MSDS for any hazardous substance used in the workplace is readily accessible to employees with potential for exposure to the substance.

Formaldehyde is a hazardous substance as defined under the NOHSC Approved Criteria for Classifying Hazardous Substances (the Approved Criteria) (NOHSC, 2004).

MSDS for different types of formaldehyde products were provided by industry and assessed according to the NOHSC National Code of Practice for the Preparation of Material Safety Data Sheets (the MSDS Code) (NOHSC, 2003). The details of the MSDS assessment are provided in Appendix 13. Four groups of MSDS were assessed: MSDS for formalin, formaldehyde products, formaldehyde containing resins, and paraformaldehyde. The overall quality of the MSDS examined is reasonable. Most conveyed the statement that the chemical/product was hazardous and provided the correct identification data. The MSDS for
formalin were considered the most comprehensive, though incomplete company
details were a major deficiency. Apart from formalin MSDS, hazard information
was inconsistent and did not correlate with the concentration cut-offs. The most
common health effect omitted was skin sensitisation, followed by corrosivity.
Information on chronic effects was also omitted in some MSDS. For first aid
statements, the most common incorrect statement was to advise induction of
vomiting following oral ingestion, mainly in MSDS for paraformaldehyde.
Details of overseas exposure standards, rather than the Australian ones, were
included in some of all the four groups of MSDS. Safe handling information was
well covered except in the MSDS for formaldehyde products.

A sample MSDS for formalin, prepared in accordance with the MSDS Code, is
provided at Appendix 14. The sample MSDS is for guidance purposes only.
Under the NOHSC National Model Regulations for the Control of Workplace
Hazardous Substances (NOHSC, 1994c), manufacturers and importers have the
responsibility to compile their own MSDS and ensure that information is up-to-
date and accurate.

Labels

Under the NOHSC National Model Regulations and Code of Practice for the
Control of Workplace Hazardous Substances (NOHSC, 1994c) and the
corresponding state and territory legislation, suppliers of hazardous chemicals
used at work are obliged to provide labels in accordance with the NOHSC Code
of Practice for the Labelling of Hazardous Substances (the Labelling Code)
(NOHSC, 1994a).

Sample labels for different types of formaldehyde products were provided by
industry and assessed according to the Labelling Code. The details of the
assessment are provided in Appendix 15. The overall quality of labels is
considered satisfactory. Most labels covered the requirements apart from first aid
and emergency procedures, which were not well covered in some label groups.
Provision of the correct signal word (either Poison or Hazardous) was the most
common omission on labels other than formalin labels. Risk and safety phrases
were omitted on some labels, but there was no consistency in the phrases omitted.

Voluntary industry guidelines

Infection Control Guidelines for the Funeral Industry were prepared in 1992 by a
committee comprising of representatives of the Australian Funeral Directors
Association (AFDA), Australian Institute of Embalming (AIE), the Australian
Workers’ Union (VIC) and the Department of Human Services (VIC). This
document, which is currently being reviewed, recommends procedures which
incorporate infection control measures designed to prevent accidental infection
amongst workers including embalmers in the funeral industry. Although this
guideline does not cover other non-infectious hazards for embalmers, such as
toxic effects due to exposure to chemicals including formaldehyde, it contains
requirements on design of embalming room (such as ventilation and embalming
tables), waste disposal, equipment cleaning, and protective clothing and
equipment for embalmers and their assistants/trainees.
18.3.3 Further actions identified

This assessment has reviewed the classification of formaldehyde against the NOHSC Approved Criteria for Classifying Hazardous Substances (NOHSC, 2004), and recommends replacement of Carcinogen, Category 3 (limited evidence of a carcinogenic effect, R40), with Carcinogen, Category 2 (may cause cancer by inhalation, R49). Classifications for the other health endpoints are confirmed in this assessment.

The current national occupational exposure standard is 1 ppm 8h TWA and 2 ppm STEL. This should be revised, as predicted human additional risk of respiratory tract cancers due to occupational exposure to formaldehyde at 1 ppm is unacceptable (approximately 50 in a million for non-smokers). In addition, the LOEL level for sensory irritation is 0.5 ppm based on the evaluation of health effects data in this assessment. The current occupational exposure standard should be lowered to 0.3 ppm 8h TWA and 0.6 ppm STEL. The supporting documentation for this proposed exposure standard is provided in Appendix 16. The proposed national exposure standard will be released for public comment by OASCC after this assessment has been published.

The general workplace measures to reduce workers’ exposure to formaldehyde are in place at the majority of workplaces. However, based on the known hazards of formaldehyde, best practice should be implemented to minimise occupational exposure to formaldehyde, especially in the industries that use products containing high concentrations of formaldehyde, such as embalming and pathology laboratories. A number of deficiencies in MSDSs and labels provided by industry were identified and these need to be noted and amended as soon as possible.
19. Secondary Notification

Under Section 65 of the *Industrial Chemicals (Notification and Assessment) Act* 1989, secondary notification of formaldehyde may be required where an introducer of the chemical becomes aware of circumstances that may warrant a reassessment of its hazard and risks. Specific circumstances include:

- The function or use of formaldehyde has increased, or is likely to change significantly;
- The amount of formaldehyde introduced into Australia has increased, or is likely to increase significantly;
- The method of manufacture of the chemical in Australia has changed or is likely to change, in a way that may result in an increased risk of adverse health effects or adverse environmental effects;
- Additional information has become available to the introducer as to the adverse health effects or adverse environmental effects of the chemical.

NICNAS will re-evaluate the cancer hazard classification and risk estimates when any new significant epidemiology data and risk estimates become available.

The Director of NICNAS must be notified within 28 days of the manufacturer/importer becoming aware of any of the above or other circumstances prescribed under Section 65 of the Act.
Appendix 1

List of Organisations and Individuals Consulted During this Assessment

List of organisations

Aerosol Association
Alan Beckwith Macbro
Ameron Coatings
AstraZeneca Pty Ltd
Atotech
Australian Chamber of Commerce and Industry
Australian Consumer & Specialty Products Association
Australian Environmental Labelling Association Inc
Australian Funeral Director Association
Australian Hardboards
Australian Paint Manufacturers Federation Inc
Australian Pharmaceutical Manufacturers Association (APMA)
Australian Wood Panels Association Incorporated
BASF Akzo Nobel Automotive OEM Coatings Pty Ltd
BHP Steel - Building & Manufacturing Markets
Brims Wood Panels Pty Ltd
Castlebark Pty Ltd
Chamber of Minerals and Energy of Western Australia
Cookson Plibrico
Cosmetics, Toiletry & Fragrance Association of Australia
Commonwealth Scientific and Industrial Research Organisation
Decorative Wood Veneers Association
Department for Environment & Heritage
Department of Forensic Medicine
E.D. Oates Pty Ltd
FC Productions
Ford Motor Company of Australia Ltd
Foseco Pty Ltd
Fugro Spatial Solutions Pty Ltd
Garrett Enterprises Pty Ltd
Grant’s Fibreglass Products
Gregory & Carr Funerals
Gribbles Pathology
HiChem Industries
Jasol Australia
Kaal Acoa Pty Ltd
Klen International Pty Ltd
Leathercrafters Association of Queensland Inc
Local Government Association of NSW
Melba Industries
Minerals Council of Australia
Nuplex - Wangaratta
Oiltech Australia Pty Ltd
Pascoe Pty Ltd
Plastics and Chemicals Industries Association Inc
Plywood Association of Australia Ltd.
Processed Forest Products
Protec Pty Ltd
Royal College of Pathologist Australasia
Sheridan Australia Pty Ltd
Sherwood Paint Industries
Smithers-Oasis Australia
Surface Coatings Association Australia
Swedish Match Australia Pty Ltd
Tasman Insulation Aust. Pty Ltd
The Valspar (Australia) Corporation Pty Ltd
Tri-Tech Chemical Co Pty Ltd
United Photo & Graphic Services Pty Ltd
Wagon Paints (Aust) Pty Ltd
Virbac Pty Ltd
Packer Leather Pty Ltd
List of individuals

Ms Cecelia Wilson  
Education Facilities Research Group,  
NSW Department of Commerce

Dr Desiree Mesaros  
Indoor Air Special Interest Group,  
Clean Air Society of Australia and New Zealand

Mr Jim Houghon  
Hire Thinking Pty Ltd.

Ms Dale Gilbert  
Built Environment Research Building Division  
Public Works, QLD

Dr Lidia Morawska  
School of Physical Sciences  
Queensland University of Technology

Professor Neil Gunningham  
School of Resources, Environment and Society  
The Australian National University

Ms Cathy Clutton  
National Health and Medical Research Council

Ms Ashley Watson  
NSW Environment Protection Authority

Dr Rosemary Nixon  
Occupational Dermatology Research and Education Centre

Ms Vicky Shepperd  
NSW Department of Health

Ms Helen Cameron  
Environmental Health,  
Australian Government Department of Health and Ageing

Dr Neale Jackson  
RMIT University

Mr Jeff Simpson  
Haztech Environmental

Dr Marian Lloyd-Smith  
National Toxics Network

Ms Lyn Dennison / Ms Sue Connor  
Environment Protection Authority Victoria

Mr Jeff Angel  
Total Environment Centre

Mr Joe Smith  
Australian Pesticides and Veterinary Medicines Authority (APVMA)
Mr James Hart
National Occupational Health and Safety Commission

Mr Steve Riobbins
Workcover NSW

Mr Ross Di Corleto
Australian Institute of Occupational Hygienists Inc. (AIOH)

Mr Don Henry
Australian Conservation Foundation

Mr John Connor
Australian Conservation Foundation

Mr Cam Walker
Friends of the Earth Victoria

Associate Professor Malcolm Sim
Monash Medical School

Mr Roger Beale
National Environment Protection and Heritage Council

Professor Graham Johnston
University of Sydney

Linda Grannas
Victorian Workcover Authority

Mr Stuart Prior
Carter Holt Harvey

Mr Peter McGarry

Mr Peter O'Donnell
Gunnersen Timbermark Pty Ltd

Dr Rory Conolloy
Chemical Industry Institute of Toxicology, US
Appendix 2

NICNAS

Questionnaire for Formulators/Manufacturers of Formaldehyde Products

Who should complete this questionnaire?

This questionnaire applies to companies that formulate and/or manufacture product(s) containing formaldehyde. Simple dilution is considered as formulation.

Return Date

Please return the questionnaire by Friday 20th December 2002 to:

Formaldehyde Questionnaire
National Industrial Chemicals Notification and Assessment Scheme
GPO Box 58
Sydney NSW 2001

Need help answering the questionnaire?

If you have any queries about the questionnaire or any of the questions, please contact:

Griffin D’Costa
Phone 02 8577 8894
Fax: 02 8577 8888
Email: griffin.d’costa@nicnas.gov.au

Please complete your company information

Company Name

Address:

Contact Name: ______________ Position: ______________

Phone Number: ______________ Email: ______________

Formaldehyde
Please circle answers where appropriate

1. Which form of formaldehyde do you use to formulate products?
   (a) Formaldehyde  □  →  a) % Formaldehyde
   b) Quantity
      (Please indicate units e.g. kg/year; litres/yr etc)
   (b) Pentaformaldehyde  □  →  a) % Formaldehyde
   b) Quantity
      (Please indicate units e.g. kg/year; litres/yr etc)

2. What products do you formulate?

<table>
<thead>
<tr>
<th>Product Name</th>
<th>Typical end uses</th>
<th>% formaldehyde</th>
<th>Product rank (Yes/No)</th>
<th>Annual sales volume of product</th>
<th>Size and type of package</th>
<th>Available to public? (e.g. retail, factory-door sales)</th>
</tr>
</thead>
<tbody>
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</tbody>
</table>

Please supply copies of the MSDS and labels for these products.
3. Describe the formulation process.

The following questions need to be answered for this point:

1. How are formaldehyde raw materials stored?
2. How is formaldehyde transferred to mixing vessels?
3. Is the formulating process open or closed?
   * Hints:
     - Open (eg. open tanks)
     - Partially closed (eg. covered tanks)
     - Closed (fully sealed process including automated addition of formaldehyde to tanks)
4. How long does one batch take and how often do you undertake batches (if it is batch process)?
5. Is any heating involved?
6. How do you collect and test samples?
7. Describe decanting and packaging process (automated or manual)
8. How are the formulated products stored and/or transported to customers?


3. How is the formulating equipment cleaned?
4. Estimate numbers and activities of workers formulating formaldehyde products.

<table>
<thead>
<tr>
<th>Number</th>
<th>Process/Activities (eg. loading, mixing, decanting, packaging etc.)</th>
<th>Hrs/day</th>
<th>Days/yr</th>
</tr>
</thead>
<tbody>
<tr>
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</tbody>
</table>

5. Describe any engineering controls that are in place to reduce exposure of workers to formaldehyde eg. exhaust ventilation, industrial fans, general ventilation etc.

<table>
<thead>
<tr>
<th>Process/Activity</th>
<th>Engineering Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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</tbody>
</table>

6. Please give details of any personal protective equipment used by workers eg type of gloves, goggles, respirators, protective clothing etc.

<table>
<thead>
<tr>
<th>Process/Activity</th>
<th>Personal Protective Equipment</th>
<th>Type</th>
</tr>
</thead>
<tbody>
<tr>
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</tbody>
</table>

7. Are any other precautions taken to reduce exposure of workers to formaldehyde?

Limited access to area of use. Y/N  Written procedures for safe use. Y/N
Special labelling or placarding: Y/N  Other__________________________

8. Has atmospheric monitoring been conducted to determine levels of formaldehyde in the workplace?

Yes  No
If yes, please provide details of monitoring eg. testing equipment, methods, duration, and results. Attach report(s) if available.

9. Have there been any incidents involving spillage of formaldehyde or formaldehyde products at your workplace?

Yes  No
If yes, please give details, including how they were cleaned up.

10. Are you aware of any adverse health effects (eg. skin, eye and respiratory irritation, allergy, burns etc.) experienced at your workplace due to exposure and/or spillage of formaldehyde or formaldehyde products?

Yes  No
If yes, please give details:

11. Are education and training programs for safe use of formaldehyde in place?

Yes  No
If yes, please give details:
12. Is there any formaldehyde waste generated during formulation, including waste through the cleaning process?  

<table>
<thead>
<tr>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
</table>

If yes, what type and what % or concentrations of formaldehyde in waste? Estimate the quantity of waste.

13. What methods do you use to dispose of formaldehyde waste (please tick)?

- Blending with other products and re-use  
- Release to atmosphere  
- Incineration eg. boiler fuel  
- Other (please specify)

<table>
<thead>
<tr>
<th>Licensed discharge</th>
<th>Send to recycler</th>
<th>Waste collection</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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</tr>
</tbody>
</table>

14. How do you handle empty formaldehyde containers (please tick)?

- Rinse and/or re-use  
- Sell to drum recycler  
- Other (please specify)

<table>
<thead>
<tr>
<th>Return to supplier</th>
<th>Send to landfill</th>
</tr>
</thead>
<tbody>
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</tbody>
</table>

Thank You for Your Time and Effort!!

Please return the questionnaire with MSDS and labels to:

Formaldehyde Questionnaire  
National Industrial Chemicals Notification and Assessment Scheme  
GPO Box 58  
Sydney NSW 2001
### Appendix 3

## Summary Tables of Human Epidemiology Data

These tables are taken from the CICAD (IPCS, 2002), therefore, the references in the table do not necessarily appear consistent with the references at the back of this report.

### Summary of risk measures from case-control studies

<table>
<thead>
<tr>
<th>Cancer/study population</th>
<th>Formaldehyde exposure</th>
<th>Risk Measure (95% CI)</th>
<th>Reference (comments)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oropharynx or hypopharynx SEER³ population based - Washington State</td>
<td>≥10 years occupational exposure occupational exposure score* of ≥20</td>
<td>OR = 1.3 (0.7-2.5) OR = 1.5 (0.7-3.0)</td>
<td>Vaughan et al., 1986a (IARC Working Group noted that different proportions of interviews conducted with next-of-kin cases and controls may have affected odds ratios)</td>
</tr>
<tr>
<td>Nasopharynx SEER population based - Washington State, USA</td>
<td>exposure score* of ≥20</td>
<td>OR = 2.1 (0.8-7.8)</td>
<td>Vaughan et al., 1986a (IARC Working Group noted that different proportions of interviews conducted with next-of-kin cases and controls may have affected odds ratios)</td>
</tr>
<tr>
<td>Nasopharynx SEER population based - Washington State, USA</td>
<td>residential exposure of ≥10 years residential exposure of &lt;10 years</td>
<td>OR = 5.5 (1.6-19.4) OR = 2.1 (0.7-6.6)</td>
<td>Vaughan et al., 1986b (IARC Working Group considered living in a mobile home a poor proxy for exposure)</td>
</tr>
<tr>
<td>Nasal squamous cell carcinoma Hospital based - Netherlands</td>
<td>&quot;any&quot; occupational exposure; assessment A &quot;any&quot; occupational exposure; assessment B</td>
<td>OR = 3.0 (1.3-6.4) OR = 1.9 (1.0-3.6)</td>
<td>Hayes et al., 1986 (IARC Working Group noted that a greater proportion of cases than controls were dead and variable numbers of next-of-kin were interviewed, 10% of controls but none of cases, by telephone; noted also that, although different, results for exposure assessments A &amp; B* were both positive)</td>
</tr>
<tr>
<td>Squamous cell carcinoma of nasal cavity/paranasal sinus Danish Cancer Registry</td>
<td>occupational exposure without exposure to wood dust</td>
<td>OR = 2.0 (0.7-5.9)</td>
<td>Olsen &amp; Asnaes, 1986 (IARC Working Group noted possibly incomplete adjustment for confounding for wood dust for adenocarcinoma; felt squamous cell carcinoma less likely to be affected, since no clear association with wood dust) (Small number of cases)</td>
</tr>
</tbody>
</table>
### Summary of risk measures from case-control studies (cont.)

<table>
<thead>
<tr>
<th>Study Description</th>
<th>Exposure Category</th>
<th>Odds Ratio</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nasopharynx Connecticut Tumour Registry, USA</td>
<td>highest potential exposure category highest potential exposure category and dying at 68+ years of age</td>
<td>OR = 2.3 (0.9-6.0)</td>
<td>Roush et al., 1987</td>
</tr>
<tr>
<td></td>
<td></td>
<td>OR = 4.0 (1.3-12)</td>
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</tr>
<tr>
<td>Oral/oropharynx Population based - Turin, Italy</td>
<td>“any” occupational exposure &quot;probable or definite&quot; occupational exposure</td>
<td>OR = 1.6 (0.9-2.8)</td>
<td>Merletti et al., 1991</td>
</tr>
<tr>
<td></td>
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<td>OR = 1.8 (0.6-5.5)</td>
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</tr>
<tr>
<td>Larynx SEER population based - Washington State, USA</td>
<td>“high” occupational exposure occupational exposure of ≥10 years occupational exposure score of ≥20</td>
<td>OR = 2.0 (0.2-19.5)</td>
<td>Wortley et al., 1992</td>
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<tr>
<td></td>
<td></td>
<td>OR = 1.3 (0.6-3.1)</td>
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<td>OR = 1.3 (0.5-3.3)</td>
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<tr>
<td>Nasal cavity/paranasal sinus (squamous cell carcinoma) Population based - France</td>
<td>males with possible exposure to formaldehyde males with duration of exposure: ≤20 years &gt;20 years</td>
<td>OR = 0.96 (0.38-2.42)</td>
<td>Luce et al., 1993 (IARC Working Group noted possible residual confounding by exposure to wood dust)</td>
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<td>OR = 1.09 (0.48-2.50)</td>
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<td>OR = 0.76 (0.29-2.01)</td>
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<tr>
<td>Nasopharynx Hospital based - Philippines</td>
<td>&lt;15 years of exposure &gt;25 years since first exposure &lt;25 years of age at first exposure</td>
<td>OR = 2.7 (1.1-6.6)</td>
<td>West et al., 1993 (IARC Working Group noted no control for the presence of Epstein-Barr viral antibodies, for which previous strong association with nasopharyngeal cancer was observed)</td>
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<td></td>
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<td>OR = 2.9 (1.1-7.6)</td>
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<td></td>
<td></td>
<td>OR = 2.7 (1.1-6.6)</td>
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<tr>
<td>Lung Nested - cohort of chemical workers - Texas, USA</td>
<td>likely occupational exposure</td>
<td>OR = 0.62 (0.29-1.36)</td>
<td>Bond et al., 1986</td>
</tr>
<tr>
<td>Lung Population based - Montreal, Quebec, Canada</td>
<td>“long-high” occupational exposure (cancer controls/ population controls)</td>
<td>OR = 1.5 (0.8-2.8)/</td>
<td>Gérin et al., 1989</td>
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<td></td>
<td></td>
<td>OR = 1.0 (0.4-2.4)</td>
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<tr>
<td>Lung (adenocarcinoma) Population based - Montreal, Quebec, Canada</td>
<td>“long-high” occupational exposure (cancer controls/ population controls)</td>
<td>OR = 2.3 (0.9-6.0)/</td>
<td>Gérin et al., 1989</td>
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<td></td>
<td></td>
<td>OR = 2.2 (0.7-7.6)</td>
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<tr>
<td>Respiratory cancer Nested - cohort of Finnish woodworkers</td>
<td>cumulative exposure of &gt;3.6 mg/m²-months, without minimum 10-year induction period cumulative exposure of &gt;3.6 mg/m²-months, with minimum 10-year induction period exposure to formaldehyde in wood dust</td>
<td>OR = 0.69 (0.21-2.24)</td>
<td>Partanen et al., 1990 (IARC Working Group noted that there were too few cancers at sites other than the lung for meaningful analysis)</td>
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<td></td>
<td></td>
<td>OR = 0.89 (0.26-3.0)</td>
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<td></td>
<td>OR = 1.19 (0.31-4.56)</td>
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<tr>
<td>Lung Population based - Missouri, USA</td>
<td>potentially exposed non-smokers</td>
<td>OR = 0.9 (0.2-3.3)</td>
<td>Brownson et al., 1993</td>
</tr>
<tr>
<td>Lung Nested - cohort of US automotive foundry workers</td>
<td>occupational exposure with latency period of: 0 years 10 years 15 years 20 years</td>
<td>OR = 1.31 (0.93-1.85)</td>
<td>Andjelkovich et al., 1994</td>
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<td></td>
<td></td>
<td>OR = 1.04 (0.71-1.52)</td>
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<tr>
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<td></td>
<td>OR = 0.98 (0.65-1.47)</td>
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<tr>
<td></td>
<td></td>
<td>OR = 0.99 (0.60-1.62)</td>
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### Summary of risk measures from case-control studies (cont.)

<table>
<thead>
<tr>
<th>Multiple myeloma</th>
<th>OR</th>
<th>Multiple myeloma</th>
<th>OR</th>
<th>Non-Hodgkin’s lymphoma</th>
<th>OR</th>
<th>Ocular melanoma</th>
<th>OR</th>
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<tbody>
<tr>
<td>Incident cases in follow-up of cancer prevention study in USA</td>
<td>1.8 (0.6-5.7)</td>
<td>males with probable occupational exposure</td>
<td>1.1 (0.7-1.6)</td>
<td>potential &quot;lower intensity&quot; of exposure</td>
<td>1.2 (0.9-1.7)</td>
<td>&quot;ever&quot; exposed to formaldehyde</td>
<td>2.9 (1.2-7.0)</td>
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<tr>
<td>Danish Cancer Registry</td>
<td>1.6 (0.4-5.3)</td>
<td>females with probable occupational exposure</td>
<td>1.6 (0.4-5.3)</td>
<td>potential &quot;higher intensity&quot; of exposure</td>
<td>1.3 (0.5-3.8)</td>
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<td></td>
<td>Heineman et al., 1992; Pottern et al., 1992</td>
<td></td>
<td></td>
<td></td>
<td>Blair et al., 1993</td>
<td></td>
<td>Holly et al., 1996</td>
</tr>
</tbody>
</table>

* a SEER = Surveillance, Epidemiology and End Results programme of the US National Cancer Institute.
* b Weighted sum of number of years spent in each job, with weighting identical to estimated formaldehyde exposure level for each job.
* c Data in parentheses represent 90% confidence interval.
* d Two independent evaluations of exposure to formaldehyde, designated assessments A and B

### Summary of risk measures from cohort studies

<table>
<thead>
<tr>
<th>Cohort exposed</th>
<th>Cancer</th>
<th>Risk measure</th>
<th>Reference (comments)</th>
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</thead>
<tbody>
<tr>
<td>Male anatomist</td>
<td>Brain</td>
<td>SMR = 270 (130-500); 10</td>
<td>Stroup et al., 1986 (Likely exposure to other substances; no quantitative data on exposure)</td>
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<tr>
<td></td>
<td>Leukaemia</td>
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<tr>
<td></td>
<td>&quot;Other lymphatic tissues&quot;</td>
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<td></td>
<td>Nasal cavity and sinus</td>
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<td></td>
<td>Larynx</td>
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<td></td>
<td>Lung</td>
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<tr>
<td></td>
<td>Multiple myeloma</td>
<td>SIR = 4 (0.5-14); 2</td>
<td>Edling et al., 1987 (Increases based on only two cases each)</td>
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<td></td>
<td>Lymphoma</td>
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<td>Pancreas</td>
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<td>Lung</td>
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<tr>
<td>Male abrasives production workers</td>
<td>Buccal cavity</td>
<td>SMR = 343 (118-786)(^b); 4</td>
<td>Stayner et al., 1988</td>
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<td></td>
<td>Connective tissue</td>
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<td></td>
<td>Trachea, bronchus, and lung</td>
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<td></td>
<td>Pharynx</td>
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<td></td>
<td>SMR = 111 (20-359)(^b); 2</td>
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<td>Garment manufacturing workers</td>
<td>Alimentary tract</td>
<td>SMR = 134 (P &gt; 0.05); 11</td>
<td>Bertazzi et al., 1989 (Small cohort exposed primarily to low concentrations; few deaths)</td>
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<td></td>
<td>Stomach</td>
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<td>Liver</td>
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<td>Lung</td>
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<tr>
<td>Resin manufacturing workers</td>
<td>Buccal cavity and pharynx</td>
<td>SMR = 52 (28-89); 13</td>
<td>Matanoski, 1989</td>
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<td></td>
<td>Respiratory system</td>
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<td></td>
<td>Hypopharynx</td>
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<td>Pancreas</td>
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<tr>
<td></td>
<td>Leukaemia</td>
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<tr>
<td></td>
<td>SMR = 56 (44-77); 77</td>
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<td>SMR = 470 (97-1340); 3</td>
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<td>SMR = 140 (104-188); 47</td>
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<td>SMR = 168 (114-238); 31</td>
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<tr>
<td>Male pathologists</td>
<td>Buccal cavity and pharynx</td>
<td>SMR = 120 (81-171); 30</td>
<td>Hayes et al., 1990</td>
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<td></td>
<td>Nasopharynx</td>
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<td></td>
<td>Lymphatic and haematopoietic</td>
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<tr>
<td></td>
<td>Colon</td>
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<td></td>
<td>Trachea, bronchus, and lung</td>
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<td></td>
<td>PMR = 216 (59-554); 4</td>
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<td>Male mortuary workers</td>
<td>Buccal cavity and pharynx</td>
<td>PMR = 127 (104-153); 111</td>
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<td>Nasopharynx</td>
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<td>Lymphatic and haematopoietic</td>
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<td></td>
<td>Colon</td>
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<td>Trachea, bronchus, and lung</td>
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<td></td>
<td>PMR = 139 (115-167); 115</td>
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<td>PMR = 94.9; 308</td>
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<td></td>
<td>PMR = 26 (17-40); 3</td>
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<td>Summary of risk measures from cohort studies (cont.)</td>
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<tr>
<td><strong>Male chemical workers employed before 1965</strong></td>
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<tr>
<td>Lung</td>
<td>Buccal cavity</td>
<td>Pharynx</td>
<td>SMR = 123 (110-136): 348</td>
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<td>SMR = 137 (28-141): 3</td>
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<td>SMR = 147 (59-303): 7</td>
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<tr>
<td>Gardner et al., 1993</td>
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<tr>
<td>(35% of cohort exposed to &gt;2 ppm [≥2.4 mg/m³])</td>
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<td><strong>Workers exposed to &gt;2 ppm (&gt;2.4 mg/m³) at one specific plant</strong></td>
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<tr>
<td>Lung</td>
<td>SMR = 126 (107-147): 165</td>
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<tr>
<td>Gardner et al., 1993</td>
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<tr>
<td><strong>Male industrial workers</strong></td>
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<tr>
<td>Nasal cavity</td>
<td>Nasopharynx</td>
<td>Lung</td>
<td>SPIR = 2.3 (1.3-4.0): 13</td>
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<tr>
<td>Larynx</td>
<td>Oral cavity and pharynx</td>
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<tr>
<td>Hansen &amp; Olsen, 1995</td>
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<tr>
<td><strong>Male industrial workers exposed above baseline levels</strong></td>
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<tr>
<td>Nasal cavity</td>
<td>SPIR = 3.0 (1.4-5.7): 9</td>
<td>Hansen &amp; Olsen, 1995</td>
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<tr>
<td><strong>Male automotive foundry workers</strong></td>
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<tr>
<td>Buccal cavity and pharynx</td>
<td>Trachea, bronchus, and lung</td>
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<tr>
<td>SMR = 131 (48-266): 14</td>
<td>SMR = 120 (89-158): 51</td>
<td>Andjelkovich et al., 1995</td>
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<tr>
<td>(25% of cohort exposed to &gt;1.5 ppm [≥1.8 mg/m³])</td>
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<tr>
<td><strong>White male industrial workers exposed to ≥0.1 ppm formaldehyde</strong></td>
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<tr>
<td>Nasopharynx</td>
<td>SMR = 270 (P &lt; 0.05): 6</td>
<td>Blair et al., 1986</td>
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</tr>
<tr>
<td>(4% of cohort exposed to ≥2 ppm [≥2.4 mg/m³])</td>
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<tr>
<td><strong>White male industrial workers with cumulative exposures of:</strong></td>
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<tr>
<td>0 ppm-years</td>
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<tr>
<td>≤0.5 ppm-years</td>
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<tr>
<td>0.5-5.5 ppm-years</td>
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<tr>
<td>&gt;5.5 ppm-years</td>
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<tr>
<td>Nasopharynx</td>
<td>SMR = 530: 1</td>
<td>Blair et al., 1986</td>
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<tr>
<td>(4% of cohort exposed to ≥2 ppm [≥2.4 mg/m³])</td>
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<tr>
<td><strong>White male industrial workers co-exposed to particulates with cumulative formaldehyde exposures of:</strong></td>
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<tr>
<td>0 ppm-years</td>
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<tr>
<td>≤0.5 ppm-years</td>
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<tr>
<td>0.5-5.5 ppm-years</td>
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<tr>
<td>&gt;5.5 ppm-years</td>
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<tr>
<td>Nasopharynx</td>
<td>SMR = 0: 0</td>
<td>Blair et al., 1987</td>
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<td>SMR = 192: 1</td>
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<td>SMR = 403: 2</td>
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<td>SMR = 746: 2</td>
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<td><strong>White male industrial workers exposed for &lt;1 year:</strong></td>
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<tr>
<td>Nasopharynx</td>
<td>SMR = 517 (P &lt; 0.05): 3</td>
<td>Collins et al., 1988</td>
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<td>SMR = 218 (P &lt; 0.05): 3</td>
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<td>SMR = 1031 (P &lt; 0.01): 4</td>
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<tr>
<td><strong>White male workers, hired between 1947 and 1956, employed at one specific plant for:</strong></td>
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<tr>
<td>&lt;1 year</td>
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<tr>
<td>≥1 year</td>
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<tr>
<td>Nasopharynx</td>
<td>SMR = 768 (P &lt; 0.05): 2</td>
<td>Marsh et al., 1996</td>
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<tr>
<td>SMR = 1049 (P &lt; 0.05): 2</td>
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<tr>
<td><strong>White male industrial workers exposed to ≥0.1 ppm formaldehyde</strong></td>
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<tr>
<td>Lung</td>
<td>SMR = 111 (96-127): 270</td>
<td>Blair et al., 1986</td>
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<tr>
<td>(4% of cohort exposed to ≥2 ppm [≥2.4 mg/m³])</td>
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</table>
### Summary of risk measures from cohort studies (cont.)

<table>
<thead>
<tr>
<th>Study Description</th>
<th>Exposure Period</th>
<th>SMR or RR</th>
<th>Confidence Interval</th>
<th>Reference</th>
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<tbody>
<tr>
<td><strong>White male industrial workers with ≥20 years since first exposure</strong></td>
<td>Lung</td>
<td>SMR = 132 (P ≤ 0.05): 151</td>
<td>Blair et al., 1986 (4% of cohort exposed to &gt;2 ppm [≥2.4 mg/m³])</td>
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<tr>
<td><strong>White male industrial workers with cumulative exposures of:</strong></td>
<td>Lung</td>
<td>SMR = 68 (37-113): 14</td>
<td>Blair et al., 1986 (4% of cohort exposed to ≥2 ppm [≥2.4 mg/m³])</td>
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<tr>
<td>0 ppm-years</td>
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<tr>
<td>&lt;0.5 ppm-years</td>
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<td>0.51-5.5 ppm-years</td>
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<td>&gt;5.5 ppm-years</td>
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<tr>
<td><strong>Wage-earning white males in industrial cohort exposed to formaldehyde and other substances</strong></td>
<td>Lung</td>
<td>SMR = 140 (P &lt; 0.05): 124</td>
<td>Blair et al., 1990a</td>
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<tr>
<td><strong>Wage-earning white males in industrial cohort exposed to formaldehyde</strong></td>
<td>Lung</td>
<td>SMR = 100 (P &gt; 0.05): 88</td>
<td>Blair et al., 1990a</td>
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<tr>
<td><strong>Subjects in industrial cohort less than 65 years of age with cumulative exposures of:</strong></td>
<td>Lung</td>
<td>RR = 1.0</td>
<td>Sterling &amp; Weinkam, 1994</td>
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<tr>
<td>&lt;0.1 ppm-years</td>
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<td>0.1-0.5 ppm-years</td>
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<td>0.5-2.0 ppm-years</td>
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<td>&gt;2.0 ppm-years</td>
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<td><strong>Males in industrial cohort less than 65 years of age with cumulative exposures of:</strong></td>
<td>Lung</td>
<td>RR = 1.0</td>
<td>Sterling &amp; Weinkam, 1994</td>
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<td>&lt;0.1 ppm-years</td>
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<td>0.1-0.5 ppm-years</td>
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<td>0.5-2.0 ppm-years</td>
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<td>&gt;2.0 ppm-years</td>
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<td><strong>White wage-earning males in industrial cohort with &gt;2 ppm-years of cumulative exposure and exposure durations of:</strong></td>
<td>Lung</td>
<td>SMR = 0: 0</td>
<td>Blair &amp; Stewart, 1994</td>
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<tr>
<td>&lt;1 year</td>
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<td>5-&lt;10 years</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;10 years</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>White male workers employed at one specific plant for:</strong></td>
<td>Lung</td>
<td>SMR = 134 (P &lt; 0.05): 63</td>
<td>Marsh et al., 1996 (25% exposed to &gt;0.7 ppm [≥0.84 mg/m³])</td>
<td></td>
</tr>
<tr>
<td>&lt;1 year</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥1 year</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>White males in industrial cohort with cumulative exposures of:</strong></td>
<td>Lung</td>
<td>RR = 1.00</td>
<td>Callas et al., 1996</td>
<td></td>
</tr>
<tr>
<td>0 ppm-years</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.05-0.5 ppm-years</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.51-5.5 ppm-years</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;5.5 ppm-years</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Unless otherwise noted, values in parentheses are 95% confidence interval or level of statistical significance. Risk measures are presented in the format reported in the references cited. Values in italics are the number of observed deaths or cases, when specified in the reference cited. Abbreviations are as follows: SMR = standardized mortality ratio; SIR = standardized incidence ratio; PMR = proportionate mortality ratio; SPIR = standardized proportionate incidence ratio; RR = relative risk.*

*b Values in parentheses represent 90% confidence interval.
Appendix 4

GHS Classification

In this report, formaldehyde has been classified against the NOHSC Approved Criteria for Classifying Hazardous Substances (NOHSC, 2004) (see Section 12). The hazard classification of formaldehyde using the GHS classification system is presented in Table A4-1. This system is not mandated in Australia and carries no legal status, but is presented for information purposes. GHS classification and information documentation is available at http://www.unece.org/trans/danger/publi/ghs/ghs_rev01/01files_e.html.

Table A4-1: GHS classification for health and environmental hazards of formaldehyde

<table>
<thead>
<tr>
<th>Hazards</th>
<th>Classification</th>
<th>Hazard communication</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Health hazard</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute toxicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oral</td>
<td>Category 3</td>
<td>Toxic if swallowed</td>
</tr>
<tr>
<td>Dermal</td>
<td>Category 3</td>
<td>Toxic in contact with skin</td>
</tr>
<tr>
<td>Inhalation</td>
<td>Category 2</td>
<td>Fatal if inhaled</td>
</tr>
</tbody>
</table>
| Corrosion/Irritation (Skin & Eye) | Category 1 | Symbol: Corrosion  
|                    |                | Signal word: Danger  
|                    |                | Hazard statements: Causes severe skin burns and eye damage |
| Sensitisation      |                |                      |
| Skin               | Category 1     | May cause an allergic skin reaction |
| Carcinogenicity    | Category 1B    | Symbol: Health hazard  
|                    |                | Signal word: Danger  
|                    |                | Hazard statements: May cause cancer by inhalation |
| **Environmental hazard** |            |                      |
| Acute toxicity     |                |                      |
| Crustaceans        | Category 2     | Symbol: No symbol is used  
|                    |                | Signal word: No signal word is used  
|                    |                | Hazard statements: Toxic to aquatic life |
Appendix 5

Conceptual Framework for Considering Mode-of-Action of Chemical Carcinogenesis of Formaldehyde

For Nasal Tumours

1. Introduction

The available data indicate that prolonged inhalation exposure to formaldehyde induce tumours in the nasal cavity of rats in a highly non-linear pattern. Sharp increases in tumour incidence in the nasal cavity occur at concentrations greater than 6 ppm (7.2 mg/m³) formaldehyde. Exposure to concentrations of 2 ppm (2.4 mg/m³) and lower induced no malignant nasal tumours. Results from several epidemiological studies of occupational exposure to formaldehyde have indicated an increased risk of nasopharyngeal cancers, although the data are not consistent. However, while the evidence is not considered to provide sufficient evidence of a causal association, it cannot be entirely excluded from the available data that exposure to formaldehyde may result in the development of nasopharyngeal cancers. This framework analysis will focus on nasal tumours as a result of formaldehyde exposure by inhalation.

2. Postulated mode of action

The mechanisms by which formaldehyde induces nasal cancers in rats are not fully understood and a specific mechanism to account for this observation has not been identified, especially given that in vivo studies have provided weak or negative evidence of a genotoxic action. However, several lines of evidence suggest a sustained increase in nasal epithelial cell regenerative proliferation resulting from cytotoxicity and mutation marked by DNA-protein crosslinks (DPX), are likely factors contributing to the induction of nasal tumours in rats. Increased cellular proliferation as a consequence of epithelial cell toxicity is the most significant determinant of neoplastic progression. DPX are considered a possible marker of mutagenic potential because they may initiate DNA replication errors that may result in mutation. It is proposed that inhalation of formaldehyde causes inhibition of mucociliary clearance, followed by nasal epithelial cell regenerative proliferation resulting from cytotoxicity and DPX that leads to mutation, and consequent tumour formation. This hypothesis is mainly based on observations of consistent, non-linear dose-response relationships for all three end-points (DPX, sustained increase in proliferation, and tumours) and concordance of the incidence of these effects across regions of the nasal passages.

3. Key events

The key precursor events associated with nasal cancer formation following inhalation exposure to formaldehyde include cytotoxicity and DPX, and nasal epithelial cell regenerative proliferation that are highly non-linear and in concordance with the...
incidence of nasal tumours. These events have been well defined and measured in a number of studies in rat, monkey, and human epithelial cells.

4. Dose-response relationship

Available data show a highly non-linear dose-response pattern for the key events, with no observed effects at 2 ppm (2.4 mg/m³), a minimal response at 6 ppm (7.2 mg/m³) and a sharp increase at 10 ppm (12.0 mg/m³) and 15 ppm (18.0 mg/m³). Additionally, there is good correlation between key events and regional tumour incidence and tumour sites.

There is also evidence that glutathione-mediated detoxification of formaldehyde within nasal tissues becomes saturated in rats at inhalation exposures above 4 ppm (4.8 mg/m³) (Casanova and Heck, 1987), which may also contribute to the non-linearity of the dose-response relationship for formaldehyde-induced DPX formation, epithelial cell proliferation and subsequently nasal tumour at exposures above this level.

5. Temporal association

A number of short-, medium-, and long-term studies on the effect of formaldehyde exposure on cell proliferation within the respiratory epithelium of rats has indicated a sustained increase in proliferation of nasal epithelial cells following exposure to concentrations greater than 2 ppm (2.4 mg/m³), irrespective of the exposure period. Cell proliferation was observed in animals exposed to formaldehyde from as short as 3 days. In a well-conducted 2-year study in rats with interim kills at 3, 6, 12, and 18 months (Monticello et al. 1996), the magnitude of increased cell proliferation generally decreased over time but still remained significantly increased over controls up to and including the 18 months observation period when this effect was last examined.

Data relating to temporal associations for DPX are not of good quality as most available inhalation studies regarding formaldehyde-induced DPX are short-term studies (i.e. exposure duration up to 1 day). Formaldehyde-induced DPX in the nasal epithelium of rats and monkeys were consistently revealed across these studies. However, a well-conducted study investigating acute and cumulative DPX yields in rats exposed to formaldehyde for about 12 weeks (Casanova et al., 1994) found that the acute DPX yield in the lateral meatus was about half that in controls at concentrations greater than 6ppm (7.2 mg/m³). Results of cumulative DPX yields indicated that no significant cumulation of DPX occurred in exposed rats.

Regenerative cell proliferation following formaldehyde-induced cytotoxicity increases the number of DNA replications and, thus, increases the probability of a DPX initiating a DNA replication error, resulting in a mutation. This is supported by the observed inhibition of DNA replication in the rat nose at elevated concentrations (Heck & Casanova, 1995) and increased p53 expression in preneoplastic lesions (Wolf et al., 1995). In 5 of 11 squamous cell carcinomas from rats exposed to 15 ppm (18.0 mg/m³) for up to 2 years, there were point mutations at the GC base pairs in the p53 cDNA sequence (Recio et al., 1992).

6. Strength, consistency and specificity of association of tumour response with key events

There are extensive studies investigating formaldehyde-induced carcinogenicity in both animals and humans. Available data revealed formaldehyde-induced DPX formation and increased epithelial cell proliferation within the upper respiratory tract in a range of
animal species including rats and monkeys and a variety of rat and human cells in vitro. It was found that at similar levels of exposure, concentrations of DPX were approximately an order of magnitude less in monkeys than in rats. Increased human epithelial cell proliferation following in situ exposure to formaldehyde was also reported using a model system in which rat trachea populated with human tracheobronchial epithelial cells were xenotransplanted into athymic mice.

In addition, proliferative response and increased DPX are seen in regions of the nasal cavity similar to those where tumours have been observed. The highly non-linear dose-response relationships for DPX, cytotoxicity, proliferative response and tumours are consistent, with significant increases in all end-points being observed at concentrations of greater than 4 ppm (4.8 mg/m³). This is also in good correlation with the concentration at which mucociliary clearance is inhibited and glutathione-mediated metabolism saturated i.e., 4 ppm (4.8 mg/m³). The study by Morgan et al. (1986) examining effects of inhaled formaldehyde on the nasal mucociliary apparatus in male rats also included 18 hr recovery groups following day 1, 9 and 14 of exposure to concentrations of 2 (2.4 mg/m³), 6 (7.2 mg/m³) and 15 ppm (18.0 mg/m³). Inhibition of mucociliary clearance was progressively more extensive with increasing duration of exposure but showed little or no evidence of recovery 18 hr after cessation of exposure.

7. Biological plausibility and coherence

There is a growing body of evidence supporting the biological plausibility that prolonged regenerative cell proliferation can be a causal mechanism in chemical carcinogenesis (IPCS, 2002). The hypothesised mode of action for formaldehyde–induced nasal tumour in animals exposed by inhalation is consistent with the biological plausibility, although the respective roles of DPX, mutation, and cellular proliferation in the induction of tumours in the rat nose are not fully outlined. DPX are proposed to be able to cause mutations as a result of errors of DNA replication on the damaged template. At low doses of formaldehyde, low frequency of DPX was induced and the DNA replication rate will be the normal rate of cell turnover that lead to a very low to negligible mutation frequency. However, at higher doses of formaldehyde when cytotoxicity is induced, the probability of a DPX resulting in a mutation via DNA replication is much higher. The dose-response curve for mutations will be highly non-linear. Thus, the mode of action for tumour induction at higher doses is different from that at low concentrations because of involvement of regenerative cell proliferation.

Association of the mode of action for nasal tumours with that for other toxicological end points has been demonstrated in repeated dose toxicity. Sustained increased cell proliferation has been observed in the nasal cavity in extensive short- and medium-term toxicity studies in rats and a few studies in other species. Histopathological effects in the nasal cavity (epithelial cell dysplasia and metaplasia) were consistent in a range of subchronic and chronic animal studies.

8. Other mode of action

Based on the available data, including limited evidence for a direct genotoxic action, it is not possible to identify a further mode of action that could potentially account for the observed nasal tumours.
9. Assessment of postulated mode of action

Based on the weight of evidence, the hypothesized mode of action for formaldehyde-induced nasal tumours satisfies several criteria, including consistency, concordance of dose–response relationships across all key events, and biological plausibility and coherence of the database. Given the extensive experimental data that addresses the mechanisms of formaldehyde-induced tumours in the nasal cavity, a moderate degree of confidence may be ascribed to the above hypothesis.

10. Uncertainties, inconsistencies and data gaps

Uncertainties exist for the proposed mode of action for formaldehyde-induced tumours.

In most of the cancer bioassays, data on intermediate end-points, such as proliferative response as a measure of cytotoxicity and DPX, is limited. Consequently, direct comparison of the incidence of intermediate lesions and tumours is restricted. Additionally, information on a direct relationship between DPX and mutation induction and the probability of converting a DPX into a mutation is desirable, while the mode by which regenerative cell proliferation is involved in the production of mutations required for tumour development needs to be determined.

Relevance to humans

Because formaldehyde is highly reactive at the site of contact, it is critical to take dosimetry into consideration when extrapolating across species. Humans and other primates are oronasal breathers whereas rats are obligate nose breathers. Together with significantly different anatomical features of the nasal and respiratory passages and patterns of inhaled airflow, effects associated with the inhalation of formaldehyde in humans are likely to be observed in a larger area, including deeper parts of the respiratory tract. This is supported by the effects (histopathological changes, increased epithelial cell proliferation, and DPX formation) being observed further along the upper respiratory tract in monkeys, compared to similar effects being restricted to the nasal cavity in rats exposed to moderate levels of formaldehyde.

The postulated mode of action on formaldehyde-induced tumours is likely relevant to humans based on the weight of evidence, at least qualitatively. In addition, increased cell proliferation and DPX formation within epithelia of the upper respiratory tract have been observed in monkeys exposed to formaldehyde vapour. Moreover, increased human epithelial cell proliferation following in situ exposure to formaldehyde has also been observed in a model system in which rat trachea populated with human tracheobronchial epithelial cells were xenotransplanted into athymic mice.

Direct evidence on histopathological lesions in the nose of humans exposed primarily to formaldehyde in the occupational environment is consistent with a qualitatively similar response of the upper respiratory tract in experimental animals, although this is not sufficient as a basis for inferring causality in itself. While the epidemiological studies do not provide sufficient evidence for a causal association between formaldehyde exposure and human cancer, the possibility of increased risk in humans of respiratory cancers, particularly those of the upper respiratory tract, cannot be excluded on the basis of available data.
For Leukaemia

1. Introduction

Increased risks for leukaemia, occasionally significant, have been seen in some epidemiology studies in industrial workers. A recent update of a major cohort study reported an association for leukaemia, specifically myeloid leukaemia, and formaldehyde. A reanalysis of the data using additional analysis provided little evidence to support the suggestion of a casual association. Similarly, although increased risks of leukaemia have been observed more consistently in studies of professional workers (e.g. embalmers), it cannot be excluded that observed increases are related to occupational exposures other than formaldehyde. No increased incidence of leukaemia was reported in rodent inhalation studies. An increased incidence of haemolymphoreticular tumours (i.e. leukaemias and lymphomas combined) was reported in a single questionable drinking water study in the rat. This framework analysis will focus on leukaemia as a result of formaldehyde exposure by inhalation in humans and ingestion in rats.

2. Postulated mode of action

A mode of action by which formaldehyde may induce leukaemia has not been identified. Although the possibility of transforming mutations to stem cells has been proposed in the scientific literature as a mechanism for leukaemia (Reya et al., 2001) there is currently no experimental data with formaldehyde to support this proposal. While the detection of cytogenetic abnormalities in circulating lymphocytes of workers exposed to formaldehyde might be regarded as supporting such a possibility, such effects have not been consistently observed and co-exposure to other chemicals means that it cannot be reliably concluded that they were caused by formaldehyde. Furthermore, there is only limited evidence suggesting a weak direct genotoxic action in in vivo studies in rodents. Therefore, presently, there is insufficient evidence to support the postulation that formaldehyde-induced leukaemia occurs from mutations to stem cells.

3. Key events

There is presently no experimental data that addresses the mechanism of formaldehyde-induced leukaemia. Consequently, the key precursor events associated with the induction of leukaemia following exposure to formaldehyde have not been defined in animal or human studies.

4. Dose-response relationship

Although an increased incidence in haemolymphoreticular tumours was reported in a single questionable drinking water study in the rat the increase was not dose-related. Furthermore, the pooling of tumour types reported as leukaemias and lymphomas prevents the dose-response relationship for leukemia to be specifically determined. In humans, an increased risk of leukaemia, occasionally significant, has been inconsistently reported in human epidemiology studies. Nearly all of these studies estimated exposure levels. The available data do not allow construction of a reliable dose-response relationship for formaldehyde exposure and incidence of leukaemia.
5. Temporal association

No key events have been identified in human or animal studies for formaldehyde-induced leukaemia, consequently, an analysis of potential temporal associations cannot be undertaken.

6. Strength, consistency and specificity of association of tumour response with key events

There are extensive studies investigating formaldehyde-induced carcinogenicity in both animals and humans. In human epidemiology studies, an increased risk of leukaemia has not been consistently observed, while in rodent studies a single, questionable oral study reported an increase incidence in haemolymphoreticular tumours. No increased incidence of leukaemia was reported in two further oral studies or inhalation studies in rodents. No key events have been identified in human or animal studies for formaldehyde-induced leukaemia.

7. Biological plausibility and coherence

The available data, such as the toxicokinetic profile for formaldehyde, does not support the biological plausibility of formaldehyde-induced leukaemia. No increase in formaldehyde concentration was seen in blood in humans and rats following exposure to concentrations of 1.9 ppm (2.3 mg/m$^3$) and 14.4 ppm (17.3 mg/m$^3$) formaldehyde, respectively. This has been attributed to the rapid metabolism of formaldehyde. Such rapid metabolism would inhibit systemic distribution of formaldehyde. This is supported by the absence of an effect on the bone marrow in subchronic rodent studies, and the absence of leukaemia in several inhalation bioassays and two drinking water bioassays in rodents. Furthermore, with the exception of a single, questionable, non-standard in vivo study, negative results were seen in several bone marrows cytogenetic and micronuclei studies conducted to validated test methodology. Thus, while inconsistent results of an increased risk of leukaemia have been seen in epidemiology studies and there is limited and questionable evidence from animal studies supporting the possibility of leukaemia, there are numerous negative findings in animal studies that do not support such a possibility. Toxicokinetic information suggests that following absorption formaldehyde would not reach distal sites. Overall, the available data do not support formaldehyde being leukemogenic.

8. Other mode of action

No experimental data that addresses the mechanism of formaldehyde-induced leukaemia are available and, hence, no mode of action has been postulated.

9. Assessment of postulated mode of action

No postulated mode of action has been proposed. However, from the available data formaldehyde-induced leukaemia do not satisfy several criteria, including consistency, and biological plausibility and coherence of the database. Consequently, a low degree of confidence may be ascribed to the hypothesis that formaldehyde induces leukaemia.

10. Uncertainties, inconsistencies and data gaps

No experimental data that addresses the mechanism of formaldehyde-induced leukaemia is available and, hence, no mode of action has been proposed. Furthermore, increased
incidences of leukaemia have been inconsistently observed in epidemiology studies. Similarly, while there is a large database for testing in animals, a non dose-related increased incidence of leukaemia and lymphomas combined has only been reported in a single questionable drinking water study in the rat. However, it should be noted that the absence of clear evidence of bone marrow toxicity in humans and animals indicates that if formaldehyde is a human myeloid leukogen its mode of action is likely to be different from known myeloid leukogens (such as benzene). Consequently, the absence of findings of leukaemia in the animal studies would suggest that a reliable rodent model for formaldehyde-induced myeloid leukaemia is not presently available.

Relevance to humans

No postulated mode of action has been identified. The available human and animal data do not satisfy several criteria, including consistency, and biological plausibility and coherence, for formaldehyde being leukaemogenic.

Reference

Appendix 6

Modelling for Atmospheric Concentrations of Formaldehyde

CSIRO Atmospheric Research Division undertook modelling of atmospheric concentrations of formaldehyde for:

- the impact of industrial sources taking the source configuration and Australian meteorology into account;
- urban levels of formaldehyde away from significant local sources, such as industry or large roads. These are based on a re-analysis of detailed urban airshed modelling undertaken using a comprehensive spatially distributed inventory of emissions (this work was originally carried out for EPA Victoria); and
- the near-road impact of formaldehyde emissions from a large urban freeway.

Details of the modelling techniques to assess the impact of formaldehyde emissions on the air environment at an urban scale (3 km), an industrial neighbourhood scale (100 m) and a near-road scale (0 to 100 m from curb side) are given below.

A1. Modelling methodology

In the calculation procedures described, a number of different approaches have been adopted to calculate PECs, depending on the type of source. In each case, the maximum annual average and maximum 24-hour average concentrations have been computed. The conversion 1 ppb = 1.2 μg/m³ for formaldehyde has been used.

For individual industrial sources, year-long modelling with AUSPLUME version 5.4 (a regulatory model developed by EPA Victoria) (EPA Victoria, 2000) was carried out using a 1997-1998 meteorological data file for Paisley in the western part of Melbourne. This meteorological file was derived using data from The Air Pollution Model (TAPM) modelling of the urban region described in Section A3 below. AUSPLUME is a Gaussian plume dispersion model, which is suitable for predicting ground-level concentrations of pollutants from a variety of sources. In addition to the emission rates (derived from 2001-2002 NPI data), the modelling requires information on the source configuration, for example, whether it is a diffuse area source, a fugitive emission from a building, or a release from a chimney stack. A detailed analysis would require details of the source characteristics for each facility in the NPI database, which is beyond the scope of this modelling. Instead, estimates of the source configurations were based on the source descriptions provided by NICNAS and summarised in Section 8 of the assessment report. Concentrations were calculated at a distance of 100 m from the source, except in cases where the maximum occurs at a greater distance (e.g. for tall stack releases), in which case the maximum PECs are reported. The distance of 100 m is representative of the distance from the source to the boundary of an industrial site. For near-surface sources, concentrations decrease at greater distances from the boundary. AUSPLUME was run for 1997-1998 for each source and so included the full range of meteorological conditions and stabilities. The results were analysed to derive the maximum 24-hour average concentration and the annual average concentration for each source. Separate calculations were made for the
average emitter and the largest emitter. Based on the information available about the sources, in some cases, it was appropriate to use different source configurations for the average and the largest emitters.

For diffuse urban sources, results were obtained from re-analysis of a detailed urban airshed modelling study of Melbourne undertaken for EPA Victoria by CSIRO in 2001 (Hurley et al., 2001). This study used the most up-to-date spatially-distributed emissions inventory for the region for a large number of pollutants. The modelling was carried out using TAPM. This model was developed at CSIRO Atmospheric Research Division (Hurley, 2002) and consists of prognostic meteorological and air pollution modules that can be run for multiple-nested domains. The meteorological module is an incompressible, non-hydrostatic, primitive equation model for three-dimensional simulations. It predicts the three components of the wind, temperature, humidity, cloud and rainwater, turbulent kinetic energy and eddy dissipation rate, and includes a vegetation/soil scheme at the surface and radiation effects. The model is driven by six-hourly analysis fields (on an approximately 100-km spaced grid) of winds, temperature and specific humidity from the Bureau of Meteorology’s Global Assimilation and Prediction system (GASP). These analyses contain the larger-scale synoptic variability, while TAPM is run for much finer grid spacings and predicts the meteorology at smaller scales. The air pollution module solves prognostic equations for pollutant concentration using predicted wind and turbulence fields from the meteorological module. The modelling used in the original report (Hurley et al., 2001) was carried out with 20 · 20 · 20 point nested grids at 30-km, 10-km, 3-km and 1-km horizontal grid spacings for the year July 1997 to June 1998. The re-analysis generated 24-hour averages, which supplemented the results for annual average concentrations for formaldehyde presented in the original report.

For roadway emissions, an idealised large urban freeway was modelled using AUSROADS (EPA Victoria, 2002), with the concentration calculated at distances of 0, 20 and 100 m from the edge of the freeway. The same 1997-1998 meteorological data file was used as for the industrial source modelling. AUSROADS is a line source Gaussian plume dispersion model that predicts the near-road impact of vehicle emissions in relatively uncomplicated terrain (EPA Victoria, 2002).

The models do not include effects of secondary formation and destruction of formaldehyde, which can have an impact on PECs. However, the annual average is probably dominated by poor dispersion in winter when the inversion level is low (i.e. a smaller volume of air in which the formaldehyde is mixed) rather than by secondary production in summer when the mixing height is much greater. Furthermore, it is expected that these effects are small compared to the uncertainties in emission rates and source configurations used in the modelling.

**A2 Industrial source impact modelling**

NICNAS identified seven industrial source categories and used the NPI database to derive emission data for each category (Section 13.1.1). The average and maximum emission rates are summarised in Table A6-1.

Modelling was carried out using the Gaussian plume model AUSPLUME version 5.4. A one-year meteorological file was derived from the TAPM modelling of 1997-1998 (described in Section A3 below) for a site near the Paisley air quality monitoring site in the west of Melbourne.
The following section provides details of the emission rates from each industry category and the source configurations used in the AUSPLUME modelling. The results shown are the predicted maximum annual average and maximum 24-hour average concentrations at a distance of 100 m from the source, except where the maximum occurs at a greater distance (e.g. for tall stack releases), in which case the maximum PECs are reported.

**Table A6-1: Average and maximum annual emissions of formaldehyde for each industry category and source configuration used in the AUSPLUME modelling**

<table>
<thead>
<tr>
<th>Type of industry</th>
<th>Annual release rate (kg/year)</th>
<th>Source details used in modelling for average industry source</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Average</td>
<td>Maximum from an individual facility</td>
</tr>
<tr>
<td>Mining</td>
<td>12 203</td>
<td>401 112</td>
</tr>
<tr>
<td>Wood &amp; paper</td>
<td>8195</td>
<td>27 082</td>
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<td>Electricity supply</td>
<td>4792</td>
<td>85 614</td>
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<td>Petroleum</td>
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<td>Chemical manufacture</td>
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<tr>
<td>Miscellaneous</td>
<td>79</td>
<td>1099</td>
</tr>
<tr>
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</tr>
</tbody>
</table>

**A2.1 Mining operations**

For the average source with an annual release rate of 12 203 kg/year, a representative source configuration was assumed to be a surface source with a diameter of 1000 m and an initial vertical spread of 10 m. Using AUSPLUME modelling, the annual average PEC at 100 m from the edge of the activity was 1.8 ppb and the maximum 24-hour average was 8.1 ppb. These results are approximately inversely proportional to the diameter of the area source (for a given emission rate).
Given that the main sources of emissions from mining operations are distributed surface sources, the area of emissions is likely to be approximately proportional to the emissions rate, so that PECs from the largest emitter (401 112 kg/year) are expected to be similar to those from the average emitter.

A2.2 Wood and paper product manufacturers

For the AUSPLUME modelling it was assumed that the emissions are split between two points: 50% from a 30 m stack (with a diameter of 2 m, efflux velocity of 10 m/s, and temperature of 25°C) and 50% as fugitive emissions at a height of 10 m (represented as a volume source released at a height of 10 m and with an initial vertical and horizontal spread (two times the standard deviation) of 10 m). The annual average PEC 100 m from a facility with an average emission rate was 4.8 ppb and the maximum 24-hour average was 36 ppb. The highest estimated PECs from the largest emitter were 16 ppb (annual average) and 119 ppb (maximum 24-hour average). A sensitivity analysis showed that the PECs are much more sensitive to the configuration of the source of the fugitive emissions than the stack emissions. All of the wood and paper product industries in the NPI database are located outside major urban areas. However, given the high PECs, it would be appropriate to verify these predictions by obtaining more information about the source configurations for these industries.

A2.3 Electricity supply

The source configuration for the average emitter was assumed to be a 50 m stack (2 m diameter, 10 m/s efflux velocity, and a temperature of 25°C). This produced PECs of 0.11 ppb (annual average) and 1.12 ppb (maximum 24-hour average). For the largest emitter, the source was taken to be a 200 m stack (3 m diameter, 20 m/s efflux velocity, 25°C), which produced similar PECs of 0.10 ppb (annual average) and 0.98 ppb (maximum 24-hour average) due to the greater release height. These PEC estimates are conservative because buoyant plume rise was ignored by setting the efflux temperature to 25°C. The largest emitter has slightly lower PECs than the average emitter because of the higher release height.

A2.4 Materials manufacture

The source configuration for the average facility is assumed to split between two points: 50% from a 30 m stack (diameter of 2 m, efflux velocity of 10 m/s, and temperature of 25°C) and 50% as fugitive emissions at a height of 10 m (represented as a volume source released at a height of 10 m and with an initial vertical and horizontal spread (two times the standard deviation) of 10 m). The PECs from the AUSPLUME modelling are 2.1 ppb (annual average) and 16 ppb (maximum 24-hour average).

For the largest emitter (an aluminium refinery) the source is taken to be a 50 m stack (2 m diameter, 10 m/s efflux velocity at 25°C). This produces PECs of 0.78 ppb (annual average) and 8.2 ppb (maximum 24-hour average). These values are lower than for the average emitter because the emission occurs from a taller stack.

A2.5 Petroleum refining, oil and gas extraction

The point source emissions are assumed to occur from a 50 m stack (2 m diameter, 10 m/s efflux velocity at 25°C). For the average emitter, AUSPLUME modelling produces PECs of 0.07 ppb (annual average) and 0.74 ppb (maximum 24-hour average), whereas the
largest emitter (8883 kg/year) produces PECs of 0.20 ppb (annual average) and 2.1 ppb (maximum 24-hour average).

A2.6 Chemical industry

As most emissions occur via stacks, the source configuration used for the AUSPLUME modelling was a 30 m stack (2 m diameter, 10 m/s efflux velocity, 25°C temperature) next to a 20 m high building. Maximum concentrations were found to occur 300 to 500 m from the stack. For the average facility (651 kg/year), the maximum annual average PEC was 1.5 ppb and the maximum 24-hour average was 0.41 ppb. For the largest formaldehyde manufacturing plant (6960 kg/year), the maximum annual average PEC was 0.57 ppb and the maximum 24-hour average was 4.4 ppb.

A2.7 Miscellaneous industries

In the AUSPLUME modelling, the source configuration was taken to be fugitive emissions from a 10 m building (represented as a volume source released at a height of 5 m and with an initial vertical and horizontal spread (two times the standard deviation) of 5 m). For an average emitter, the PECs were 0.14 ppb (annual average) and 1.2 ppb (maximum 24-hour average). For the largest emitter, the PECs were a factor of 14 larger, namely 2.0 ppb (annual average) and 17 ppb (maximum 24-hour average).

A2.8 Summary of point source PECs

Table A6-2 summarises the results predicted environmental concentrations described in the above sections. In each case, the maximum annual average and maximum 24-hour average PECs are listed for the average emitter and for the largest emitter.

The highest PECs occur for the wood and paper industries, which are the second largest emitter after mining operations (Table A6-1). The impact on PECs from the wood and paper industries is greater because the concentrations at the release points is higher than in mining (which is mainly due to vehicles and other surface sources). However, all of the wood and paper product industries in the NPI database are located outside major urban areas, so that they will not impact on formaldehyde concentration in large urban areas. In spite of this, the high PECs indicate that it would be useful to verify these predictions by obtaining more information about the source configurations for these industries to check the modelling assumptions.

A3 Urban impact modelling

Urban levels of formaldehyde due to diffuse urban emissions were determined from a re-analysis of detailed urban airshed modelling of ambient pollutant concentrations in Melbourne undertaken by CSIRO for EPA Victoria (Hurley et al., 2001). The original study, which was part of work for the EPAV Air Quality Improvement Plan, used a comprehensive inventory of emissions from industry, motor vehicles (petrol fuel type), wood heater emissions and biogenic emissions. The modelled year was 1997/98. The re-analysis generated 24-hour averages to supplement the original modelling of annual average concentrations. The results from the modelling with a 3-km grid spacing are listed in Table A6-3. This grid spacing of 3 km was used because it minimises the local impact from some industrial sources and thus provides an estimate of urban concentrations away from significant local sources, such as industry or large roads. The annual average concentration is 1.6 ppb and the maximum 24-hour average is 13 ppb.
Table A6-2: Summary of maximum annual average and maximum 24-hour average predicted environmental concentrations calculated from AUSPLUME modelling for each industry category

<table>
<thead>
<tr>
<th>Type of industry</th>
<th>Maximum Annual Average PECs</th>
<th>Maximum 24-hour Average PECs</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Average emitter</td>
<td>Largest emitter</td>
</tr>
<tr>
<td>Mining</td>
<td>1.8 ppb</td>
<td>H1.8 ppb</td>
</tr>
<tr>
<td>Wood &amp; paper</td>
<td>4.8 ppb</td>
<td>16 ppb</td>
</tr>
<tr>
<td>Electricity supply</td>
<td>0.11 ppb</td>
<td>0.10 ppb</td>
</tr>
<tr>
<td>Materials manufacture</td>
<td>2.1 ppb</td>
<td>0.78 ppb</td>
</tr>
<tr>
<td>Petroleum</td>
<td>0.07 ppb</td>
<td>0.20 ppb</td>
</tr>
<tr>
<td>Chemical manufacture</td>
<td>0.05 ppb</td>
<td>0.57 ppb</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>0.14 ppb</td>
<td>2.0 ppb</td>
</tr>
</tbody>
</table>

Table A6-3: Maximum formaldehyde concentrations in the Melbourne urban region from TAPM modelling for the year July 1997 to June 1998 (3-km grid spacing)

<table>
<thead>
<tr>
<th>Averaging time</th>
<th>Maximum formaldehyde concentration</th>
<th>70th percentile</th>
</tr>
</thead>
<tbody>
<tr>
<td>Annual average</td>
<td>1.6 ppb</td>
<td>-</td>
</tr>
<tr>
<td>24-hr average</td>
<td>13 ppb</td>
<td>2.2 ppb</td>
</tr>
</tbody>
</table>

When determining the impact of an industrial source located in an urban area, it is common practice to add the maximum PEC for the industrial source to a typical urban background concentration, represented by the 70th percentile (EPA Victoria, 1985), rather than the maximum urban background, which is unlikely to occur at the same time as the maximum source impact. Figure A6-1 shows the cumulative probability distribution of the 24-hour averages with the 70th percentile which equals to 2.2 ppb.

A4 Near road modelling

Maximum formaldehyde concentrations due to roadway emissions were determined from modelling of emissions from a 6-lane dual carriageway freeway. Modelling was carried out using AUSROADS, which is a Gaussian dispersion model based on the Caline-4 model, with a user-friendly interface developed by EPA Victoria (EPA Victoria, 2002). The modelled roadway geometry was a straight section 3 km in length with 3 lanes in each direction, representative of a large urban freeway. Each lane was 4 m wide and there was a separation of 8 m between the carriageways. The total daily flow rate was modelled to be 150 000 cars per day, evenly divided between each of the 6 lanes with the diurnal distribution shown in Figure A6-2. This diurnal distribution was based on weekday flows
in the Sydney M5 East tunnel and Melbourne's CityLink tunnel, scaled up to 25 000 vehicles per lane per day, which was considered to be typical of city freeway flows.

**Figure A6-1:** Annual cumulative probability distribution of 24-hour average formaldehyde concentrations in the Melbourne urban region from re-analysis of TAPM modelling for the year July 1997 to June 1998 (3-km grid spacing)

**Figure A6-2:** Assumed diurnal variation in traffic flow on each lane of the modelled roadway for a total daily flow of 25 000 vehicles per lane.
The fleet average emission factor for formaldehyde was taken to be 20 mg/km, as reported in a Melbourne study by EPA Victoria (1999c). This compares with a recent value of 13.7 mg/km reported for measurements on Melbourne’s CityLink (Tran et al., 2003). However, the lower value for CityLink traffic probably reflects the higher proportion of newer cars (with reduced emissions) than would be found in a city-wide average.

The meteorological data file for AUSROADS was the same as that used in the AUSPLUME modelling discussed in Section A2 above, i.e. for 1997-1998 and representative of the western region of Melbourne. To remove any influence of a predominant wind direction from the results, modelling was carried out with the roadway aligned north-south and then east-west. Less than 10% difference was found between the maximum predicted concentrations for these two orientations. The results at three distances from the edge of the roadway, listed in Table A6-4, show a rapid decrease with increasing distance from the roadway.

Table A6-4: PECs for typical large urban freeway (150 000 cars per day) modelled using AUSROADS

<table>
<thead>
<tr>
<th>Location</th>
<th>Maximum annual average PEC</th>
<th>Maximum 24-hour average PEC</th>
</tr>
</thead>
<tbody>
<tr>
<td>At edge of freeway</td>
<td>0.77 ppb</td>
<td>2.3 ppb</td>
</tr>
<tr>
<td>20 m from edge of freeway</td>
<td>0.37 ppb</td>
<td>1.06 ppb</td>
</tr>
<tr>
<td>100 m from edge of freeway</td>
<td>0.15 ppb</td>
<td>0.50 ppb</td>
</tr>
</tbody>
</table>
Appendix 7

Estimates of Point Source Emissions from Industry

Table A7-1: Annual release estimates of formaldehyde from various mining operations (NPI database 2001-2002 & 2002-2003)

<table>
<thead>
<tr>
<th>Mining Type</th>
<th>Number of Facilities</th>
<th>Annual release rate (kg/year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metal ore</td>
<td>22</td>
<td>23</td>
</tr>
<tr>
<td>Construction material</td>
<td>11</td>
<td>11</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>7</td>
<td>8</td>
</tr>
<tr>
<td>Coal</td>
<td>8</td>
<td>9</td>
</tr>
<tr>
<td>Average</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* range of emissions from individual facilities.


<table>
<thead>
<tr>
<th>Type of industry (number of facilities)*</th>
<th>Number of Facilities</th>
<th>Annual release rate (kg/year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wood products (14)</td>
<td>14</td>
<td>14</td>
</tr>
<tr>
<td>Paper &amp; paper products (4)</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Log sawmilling and timber dressing (9)</td>
<td>3</td>
<td>9</td>
</tr>
<tr>
<td>Average</td>
<td>8195</td>
<td></td>
</tr>
</tbody>
</table>

*reporting to NPI.

<table>
<thead>
<tr>
<th>Type of industry</th>
<th>Number of Facilities</th>
<th>Annual release rate (kg/year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basic non-ferrous metal (7)</td>
<td>6</td>
<td>14</td>
</tr>
<tr>
<td>Glass and glass products (2)</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Non-metallic mineral (3)</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Cement, lime, concrete (5)</td>
<td>4</td>
<td>13</td>
</tr>
<tr>
<td>Ceramic Product Manufacturing (12)</td>
<td>7</td>
<td>12</td>
</tr>
<tr>
<td>Fabricated metal (6)</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>Iron and Steel Manufacture (1)</td>
<td>–</td>
<td>1</td>
</tr>
<tr>
<td><strong>Average</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table A7-4: Annual release estimates of formaldehyde from the chemical manufacturing industry (NPI database 2001-2002 & 2002-2003)

<table>
<thead>
<tr>
<th>Manufacturing Category</th>
<th>Number of Facilities</th>
<th>Annual release rate (kg/year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basic chemical</td>
<td>11</td>
<td>14</td>
</tr>
<tr>
<td>Other Chemical</td>
<td>14</td>
<td>13</td>
</tr>
<tr>
<td><strong>Average</strong></td>
<td>14</td>
<td>13</td>
</tr>
</tbody>
</table>
Table A7-5: Release estimates from formaldehyde manufacturing plants (NPI database 2001-2002)

<table>
<thead>
<tr>
<th>Manufacturing Company</th>
<th>Annual release rate (kg/year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Woodchem, Oberon, NSW</td>
<td>157</td>
</tr>
<tr>
<td>Orica, VIC</td>
<td>485</td>
</tr>
<tr>
<td>Deer Park, VIC</td>
<td>6960</td>
</tr>
<tr>
<td>Hexion, Laverton, VIC</td>
<td>93.2</td>
</tr>
<tr>
<td>Hexion, Gibson Island, QLD</td>
<td>5750</td>
</tr>
<tr>
<td>Dynea, Dardanup, WA</td>
<td></td>
</tr>
<tr>
<td>Total (Average)</td>
<td>13,445 (2689)</td>
</tr>
</tbody>
</table>
Appendix 8

EASE Modelling for Film Processing

The EASE (Estimation and Assessment of Substance Exposure) model (UK HSE, 2000) is a knowledge-based electronic data system designed to facilitate the assessment of workplace exposure. It predicts exposure as ranges in the form of conventional 8-hour time weighted average (TWA).

Exposure is determined by the EASE model at the high-end or maximum concentrations (i.e. worst-case estimates) in feasible but not unrealistic situations (i.e. reasonable worst-case situation). The estimates are not intended to be representative of extreme or unusual use scenarios that are unlikely to occur in the workplace. It is acknowledged that the EASE model takes a conservative approach and is likely to overestimate exposure.

EASE model assumes that the operator spends full shift (8 h) working at sites and is exposed to 100% formaldehyde solutions alone. However, the majority of work processes involving potential exposure to formaldehyde solutions do not fit this assumption. Therefore, the uncertainties have been taken into consideration when potential occupational exposure is discussed (see Section 15.6.4).

Three temperatures (10°C, 25°C and 40°C) were modelled to cover the atmospheric changes in different seasons of a year. The results were the same at the three temperatures for inhalation exposure estimation. Also, information obtained from film processing industry indicates that the work is carried out at room temperatures. Therefore, only results estimated at 25°C are presented here.

The input to the EASE model for film processing and results are presented below.

PARAMETERS used in the modelling:

The name of the substance is formaldehyde

The temperature of the process is 25°C

The physical-state is liquid

The exposure-type is gas/vapour/liquid aerosol Aerosol-formed is false

The use-pattern is closed system

Significant-breaching is false

The pattern-of-control is Full containment

The status-vapour-value is measured at process temperature

The vapour pressure is 20 mm Hg

Formaldehyde
Converting vapour pressure to kiloPascals:
The vp-value of the substance is 2.66578947368421
The volatility of the substance is moderate
The ability-airborne-vapour of the substance is moderate

CONCLUSION:

Inhalation exposure to the gas, vapour or liquid aerosol of formaldehyde at a process temperature of 25°C is determined by:

- The pattern of use (Closed system),
- The pattern of control (Full containment), and
- The ability of the substance to become airborne (moderate).

and resulting in an exposure range of very low (0-0.1 parts per million) if the substance is being used within a closed system.
Appendix 9

Biologically Motivated Case-Specific Model for Cancer


Derivation of the dose–response model and selection of various parameters are presented in greater detail in CIIT (1999); only a brief summary is provided here. The clonal growth component is identical to other biologically based, two-stage clonal growth models (Figure A9-1) (also known as MVK models), incorporating information on normal growth, cell cycle time, and cells at risk (in various regions of the respiratory tract).

Formaldehyde is assumed to act as a direct mutagen, with the effect considered proportional to the estimated tissue concentration of DNA–protein crosslinks. The concentration–response curve for DNA–protein crosslink formation is linear at low exposure concentrations and increases in a greater than linear manner at high concentrations, similar to those administered in the rodent carcinogenicity bioassays. For cytotoxicity and subsequent regenerative cellular proliferation associated with exposure to formaldehyde, the non-linear, disproportionate increase in response at higher concentrations is incorporated. Values for parameters related to the effects of formaldehyde exposure upon the mutagenic (i.e., DNA–protein crosslink formation) and proliferative response (i.e., regenerative cell proliferation resulting from formaldehyde-induced cytotoxicity) were derived from a two-stage clonal growth model developed for rats (Figure A9-2), which describes the formation of nasal tumours in animals exposed to formaldehyde.

Species-specific dosimetry within various regions of the respiratory tract in laboratory animals and humans was also incorporated. Regional dose is a function of the amount of formaldehyde delivered by inhaled air and the absorption characteristics of the lining within various regions of the respiratory tract. The amount of formaldehyde delivered by inhaled air depends upon major airflow patterns, air-phase diffusion, and absorption at the air–lining interface. The “dose” (flux) of formaldehyde to cells depends upon the amount absorbed at the air–lining interface, mucus/tissue-phase diffusion, chemical interactions such as reactions and solubility, and clearance rates. Species differences in these factors influence the site-specific distribution of lesions.

The F344 rat and rhesus monkey nasal surface for one side of the nose and the nasal surface for both sides of the human nose were mapped at high resolution to develop three-dimensional, anatomically accurate computational fluid dynamics (CFD) models of rat, primate, and human nasal airflow and inhaled gas uptake (Kimbell et al., 1997; Kepler et al., 1998; Subramaniam et al., 1998). The approximate locations of squamous epithelium and the portion of squamous epithelium coated with mucus were mapped onto the reconstructed nasal geometry of the CFD models. These CFD models provide a means for estimating the amount of inhaled gas reaching any site along the nasal passage walls and allow the direct extrapolation of exposures associated with tissue damage from animals to humans via regional nasal uptake. Although development of the two-stage clonal growth
modelling for rats required analysis of only the nasal cavity, for humans, carcinogenic risks were based on estimates of formaldehyde dose to regions (i.e., regional flux) along the entire respiratory tract.

The human clonal growth modelling (Figure A9-3) predicts the additional risk of formaldehyde-induced cancer within the respiratory tract under various exposure scenarios.

Two of the parameters in the human clonal growth model — the probability of mutation per cell division and the growth advantage for preneoplastic cells, both in the absence of formaldehyde exposure — were estimated statistically by fitting the model to human 5-year age group lung cancer incidence data for non-smokers. The parameter representing the time for a malignant cell to expand clonally into a clinically detectable tumour was set at 3.5 years.

In addition to the human nasal CFD model, a typical path, one-dimensional model (see CIIT, 1999) of formaldehyde uptake was developed for the lower respiratory tract. This latter model consisted of the tracheobronchial and pulmonary regions in which uptake was simulated for four ventilatory states, based on an ICRP (1994) activity pattern for a heavy-working adult male. Nasal uptake in the lower respiratory model was calibrated to match overall nasal uptake predicted by the human CFD model. While rodents are obligate nasal breathers, humans switch to oronasal breathing when the level of activity requires a minute ventilation of about 35 litres/min. Thus, two anatomical models for the upper respiratory tract encompassing oral and nasal breathing were developed, each of which consisted basically of a tubular geometry. For the mouth cavity, the choice of tubular geometry was consistent with Fredberg et al. (1980). The rationale for using the simple tubular geometry for the nasal airway was based primarily upon the need to remove formaldehyde from the inhaled air at the same rate as in a corresponding three-dimensional CFD simulation. However, in calculations of carcinogenic risk, the nasal airway fluxes predicted by the CFD simulations, and not those predicted by the single-path model, were used to determine upper respiratory tract fluxes.

To account for oronasal breathing, there were two simulations. In one simulation, the nasal airway model represented the proximal upper respiratory tract, while in the other simulation, the mouth cavity model was used for this region. In both simulations, the fractional airflow rate in the mouth cavity or in the nasal airway was taken into account. For each segment distal to the proximal upper respiratory tract, the doses (fluxes) of formaldehyde from both simulations were added to obtain the estimated dose for oronasal breathing. The site-specific deposition of formaldehyde along the human respiratory tract coupled with data on effects upon regional DNA–protein crosslinks and cell proliferation (derived from studies in animals) (Casanova et al., 1994; Monticello et al., 1996) were reflected in calculations of carcinogenic risks associated with the inhalation of formaldehyde in humans.

---

1 Data on predicted risks of upper respiratory tract cancers for smokers are also presented in CIIT (1999)
Figure A9-1: Two-stage clonal growth model (reproduced from CIIT, 1999).

Figure A9-2: Roadmap for the rat clonal model (reproduced from CIIT, 1999)

CFD=computational fluid dynamics; DPX=DNA-protein crosslinking; SCC= squamous cell carcinoma
Figure A9-3 – Roadmap for the human clonal growth model (reproduced from CIIT, 1999)
References

Casanova M, Morgan KT; Gross E; Moss OR; & Heck H; (1994) DNA-Protein cross links and cell replication at specific sites in the nose of F344 rats exposed subchronically to formaldehyde. Fundamental and Applied Toxicology, 23: 525-536.


Appendix 10

Worst-case Scenario Cancer Risk Estimation
By Rory B. Conolly at the Chemical Industry Institute of Toxicology (CIIT)
March 2005

Estimates of additional lifetime cancer (respiratory tract) were calculated based on worst-case childhood exposures. The details of the CIIT cancer risk model (Conolly et al., 2004) is summarised in Appendix 9.

**Exposure information**

**Exposure durations**

*Schooling age: 5-17 years old (12 years education)*

*Overall time spent indoors for all age groups: 20 hours per day (based on recent Australian survey data, enHealth Council, 2003)*

*Average duration in schools: 6 hours a day, 5 days a week*

**Indoor air formaldehyde levels**

The indoor air levels of formaldehyde are based on limited Australian monitoring data, as discussed in Section 13.2, and summarised in Table A10-1.

**Table A10-1: Indoor air levels of formaldehyde in mobile homes and relocatable buildings**

<table>
<thead>
<tr>
<th>Type of building</th>
<th>Indoor air level (average)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mobile homes</td>
<td>29 ppb (2000 data)</td>
</tr>
<tr>
<td></td>
<td>346 ppb (1991 data)</td>
</tr>
<tr>
<td>Relocatable buildings</td>
<td>No data in classrooms available</td>
</tr>
<tr>
<td></td>
<td>No recent data in relocatable buildings</td>
</tr>
<tr>
<td></td>
<td>710 ppb (1992 data for relocatable offices)</td>
</tr>
</tbody>
</table>

Although no data are available for relocatable classrooms, it is reasonable to assume a similar level as relocatable offices.

Due to lack of recent data in relocatable buildings, it is assumed that the indoor air formaldehyde levels in this type of building have not changed (worst-case scenario).

Two indoor air exposure scenarios were considered:

- **Scenario A:** Based on recent data - 29 ppb for 14 h/day at home and 710 ppb for 6 h/day at school
- **Scenario B:** Based on earlier data - 346 ppb for 14 h/day at home and 710 ppb for 6 h/day at school (worst-case)

Indoor air exposures for 18-80 year olds were the same for both scenarios (i.e. 30 ppb for 20 hours/day).
**Ambient formaldehyde levels**

Ambient air level of formaldehyde is estimated annual average of 5.5 ppb (Section 13.1).

**Risk estimations and results**

The cancer risk model used (Conolly et al., 2004) specifies how breathing rate changes on an hour-by-hour basis each day and these changes are incorporated into the analysis of cancer risk, as it affects the respiratory tract dosimetry of formaldehyde. Table A10-2 presents a matrix of formaldehyde levels and ventilation rate used for the risk estimation.

Risks were predicted for 80-year lifetimes. The predicted additional risks were $2.9 \times 10^{-7}$ for scenario A (29 ppb) and $4.5 \times 10^{-7}$ for scenario B (346 ppb).

Most of this risk is attributable in the model to the mutagenic pathway mediated by formation of DNA-protein crosslinks (DPX), with only a small fraction of the predicted risk being attributable to effects on the rate of cell division. This is notable, as the risks were predicted using an upper bound estimate of the value of the parameter (KMU) that links DPX with direct mutation. In the statistical development of the model the best estimate of the value of KMU was zero ($0$). Figure A10-1 shows the predicted relationships between duration of exposure and additional cancer risk using the upper bound estimate for KMU.

![Figure A10-1: Predicted additional risks for the two exposure scenarios.](image_url)
Table A10-2: Exposure concentrations – respiratory ventilation rate matrix

<table>
<thead>
<tr>
<th>Respiratory Ventilation rate</th>
<th>0 - 17 Years Old</th>
<th>Weekend day (h)</th>
<th>18 - 80 Years Old</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>School day (h)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Home (Scenario A) 29 ppb</td>
<td>School 710 ppb</td>
<td>Outdoors 5.5 ppb</td>
</tr>
<tr>
<td>Sleeping, 7.5 L/min, 8 h/day</td>
<td>8</td>
<td></td>
<td>8</td>
</tr>
<tr>
<td>Sitting, 9.0 L/min, 8 h/day</td>
<td>4</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>Light activity, 25 L/min, 8 h/day</td>
<td>2</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Total</td>
<td><strong>24 hours</strong></td>
<td><strong>24 hours</strong></td>
<td><strong>24 hours</strong></td>
</tr>
</tbody>
</table>

*The 24 h day is partitioned by exposure concentrations and breathing rate. This table can be used to identify concentration – breathing rate pairs for a full 80-year lifetime.

\(^b\)maximum average in Australian conventional homes (Section 13.2).
Appendix 11

PAA PRODUCT CERTIFICATION BRAND STAMPS

PRODUCT CERTIFIED
Laminated Veneer Lumber
PAA LWOOD
An accredited body under reg. No. Z16880AA0
Laminated Veneer Lumber to AS/NZS4357

PRODUCT CERTIFIED
Structural Plywood to AS/NZS2269

PRODUCT CERTIFIED
A Bond Exterior Plywood to AS2271

PRODUCT CERTIFIED
B Bond Exterior Plywood to AS2271

PRODUCT CERTIFIED
Interior Plywood to AS2270

PRODUCT CERTIFIED
Marine Plywood to AS/NZS2272

JAS-ANZ
Appendix 12

Australian-made Wood Panel Products

(A) Sample Label for Australian-made Wood Panel Products
(B) Emissions from Australia-made wood panel products [Source: AWPA (2005)]

Figure A12-1: Particleboard Formaldehyde Emission Data (Dessicator Method)
Figure A12-2: MDF Formaldehyde Emissions Data (Dessicator Method)
Testing Methodology for Figures A12-1 and A12-2

Product Testing and Compliance

Formaldehyde results (Desiccator method to AS/NZS4266:16) are generated as part of the AWPA certification process which all significant sized plants of AWPA members participate in.

The first stage in the Certification process is for the plant to establish a history of product compliance to the standards used for Certification purposes, as determined by testing at the AWPA Test Center. In general, product compliance sampling will be based on the criteria for External Control stipulated in Section 6 of EN 326:2 2000.

The mandatory tests applying are the AS/NZS 4266 Wood based panels test method series and the specifications are from AS/NZS 1859 and 1860.

For each product category for which it wishes to be certified, the plant must achieve the specification for each thickness range produced based on a minimum of 12 tests. Samples must be from a minimum of 3 different production batches.

There are 12 participating plants in the certification program and with the above requirements, a total of approx. 60 samples a week are supplied to the test centre. Formaldehyde analysis is conducted on each sample. This means that each annual formaldehyde result per product grade shown on the bar chart represents the average of several hundred results. The minimum number which would be supplied by an individual manufacturer for a particular grade in one year is 24.

Individual results can be accessed by the members on AWPA website, while monthly summaries are issued from the AWPA office.

Test Sample Submission

<table>
<thead>
<tr>
<th>Product</th>
<th>Sample Numbers Per Product</th>
<th>Plant annual production capacity (M³)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Each product defined as a combination of grade, line and AS/NZS thickness range</td>
<td>12 per 6 month period</td>
<td>N/A</td>
</tr>
</tbody>
</table>

Sampling Method

1) The test panel size is 1200 x 750mm or equivalent e.g. 1800 x 600mm. For thicknesses over 25mm, send 2 test panels.

2) A consistent sampling routine must be established in your plant to ensure consistent results.

3) Sampling must be carefully managed so that over each 6 months period, a minimum of 12 samples are dispatched for each combination of grade, line and thickness range produced.

4) The Quality Representative or his delegate will draw at random the test panels so that each panel has an equal chance of being selected.
5) Panels are to be selected after sanding and cutting prior to packing. They are to be wrapped in plastic and dispatched within 3 days. Samples are to be dispatched regularly - no more than a fortnight’s test panels are to be sent in one lot.
   a)

6) Test panels are to be marked with the following information:
   □ Company name and plant location
   □ Batch No or Sample No
   □ Product Type e.g. MR MDF, Particleboard flooring
   □ Thickness
   □ Any variations to the normal specification (e.g. trial board, E0, JIS, Low Density) must be clearly identified so that it can be classified accordingly.
   □ Indication of top face where the 2 surfaces are different.

7) Do not send more than 6 panels in one pack, as the AWPA has no mechanical handling equipment.
Appendix 13

MSDS Assessment

Introduction

The National Code of Practice for the Preparation of Material Safety Data Sheets (the MSDS Code) (NOHSC, 2003) provides guidance on the content and format of MSDS. It identifies ‘core’ information that should be present in all MSDS. This assessment focussed on the adequacy of the information provided in relation to the following selected core elements: product identification, health hazard information, precautions for use, safe handling information and company details. Information considered most important in each of these sections was identified and listed in Table A13-1. These items were assessed for both the presence and accuracy of the information.

An MSDS for a product containing a mixture of ingredients must address the hazards posed by the product as a whole, taking into account all of the ingredients. However, as this report only focuses on formaldehyde, some of the elements listed in Table A13-1 were not addressed in the assessment of MSDS for mixtures. With regard to health effects, mixtures were checked for inclusion of at least the health effects associated with formaldehyde. In deciding which of the health effects should apply in each case, the concentrations of formaldehyde in the product, and the cut-off levels associated with the different hazard criteria (for details see the Hazardous Substances Information System [DEWR, 2004]) were taken into account. In some cases MSDS gave a range for the concentration of formaldehyde, for example 1% to 10%. In such cases, it was assumed that the maximum concentration of formaldehyde was present. If an item could not be assessed then its presence or absence was simply noted.

Information on paraformaldehyde (including MSDS and labels) was collected during the assessment, as paraformaldehyde decomposes to formaldehyde under heat and can be a significant formaldehyde source. Paraformaldehyde is not listed in the OASCC’s Hazardous Substances Information System (DEWR, 2004). It is, however, listed in the Australian Code for the Transport of Dangerous Goods by Road and Rail (the ADG Code) (FORS, 1998) as a dangerous good and has a UN number (2213). It is also scheduled (Schedule 6 in concentrations ≥ 5%) by the Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) (NDPSC, 2005). The assessment of health effects in MSDS for paraformaldehyde focused on the presence or absence of health effects information relevant to formaldehyde.

Table A13-2 shows the number of MSDS provided to NICNAS in the course of this assessment, and the number selected and assessed against the MSDS Code. Where possible, NICNAS selected MSDS from different companies and for different concentrations of formaldehyde.
Table A13-1: The key information checked for inclusion in MSDS

<table>
<thead>
<tr>
<th>MSDS Section</th>
<th>Items Checked</th>
</tr>
</thead>
<tbody>
<tr>
<td>Introductory</td>
<td>Presence of statement of hazardous nature i.e. ‘Hazardous according to the criteria of NOHSC’.</td>
</tr>
</tbody>
</table>
| Product Identification| □ Product name  
                         □ UN Number* 
                         □ Dangerous Goods Class*  
                         □ HAZCHEM code*  
                         □ Poisons Schedule*  
                         □ Major recommended uses  
                         □ Disclosure of presence of formaldehyde  
                         □ Disclosure of the exact proportion or a range |
| Health hazards        | □ Acute and chronic health effects\(^1\)  
                         □ Appropriate first aid statements\(^2\) |
| Precautions for use   | □ Exposure standard  
                         □ Advice on PPE |
| Safe handling         | Advice on storage and transport, spills and disposal, fire/explosion hazard |
| Company details and contact point | □ Name, address and telephone number of company  
                          □ Emergency telephone number  
                          □ Title and telephone number of a contact point |

1. Acute effects: Acutely toxic; causing irritation of skin, eyes, nose, throat and respiratory system; skin sensitisation; corrosion of gut lining if swallowed. Chronic effects: nasal tumours in animals but insufficient data for humans. This information is in accordance with the current NOHSC hazard classification for formaldehyde (DEWR, 2004).

2. Inhalation: Remove from exposure. Apply artificial respiration if not breathing; Swallowed: Do NOT induce vomiting; Eyes: Hold eyelids apart and flush the eye continuously with running water. Continue flushing for at least 15 minutes or until advised to stop by a Poisons information Centre or a doctor; Skin: Remove contaminated clothing and wash skin and hair thoroughly.

*Products with concentrations of formaldehyde ≤ 25% are not classified under these items in the Australian Code for the Transport of Dangerous Goods by Road and Rail (FORS, 1998). However, in these circumstances, the MSDS Code requires a statement that no number/class/code has been allocated.

* Products with concentrations of formaldehyde of ≤ 5% are not scheduled under the current Standard for the Uniform Scheduling of Drugs and Poisons (NDPSC, 2005). However, in these circumstances, the MSDS Code requires a statement to that effect.

Table A13-2: Number and type of MSDS received and assessed

<table>
<thead>
<tr>
<th>MSDS Type</th>
<th>Number Received</th>
<th>Number Assessed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Formalin</td>
<td>49</td>
<td>10</td>
</tr>
<tr>
<td>Formaldehyde products</td>
<td>107</td>
<td>10</td>
</tr>
<tr>
<td>Formaldehyde containing resins</td>
<td>185</td>
<td>10</td>
</tr>
<tr>
<td>Paraformaldehyde</td>
<td>14</td>
<td>11</td>
</tr>
<tr>
<td>Paraformaldehyde products</td>
<td>5</td>
<td>Not assessed</td>
</tr>
</tbody>
</table>

Priority Existing Chemical Assessment Report No. 28
Results of assessment of MSDS for formalin

Statement of hazardous nature

Nine out of ten MSDS assessed included a statement of hazardous nature. However, it should be noted that three of these MSDS referred to “Worksafe”, which is the former business name of the National Occupational Health and Safety Commission.

Product identification

All MSDS had the product name, disclosed the presence and proportion of formaldehyde, and provided the correct UN Number and Dangerous Goods Class. Nine gave correct HAZCHEM Code and Poisons Schedule Number. Two MSDS gave no details on major recommended uses.

Health hazard information

Formaldehyde was present in concentrations greater than 25% in all 10 formalin MSDS, hence, all the health hazards associated with the chemical should be referred to. Acute health effects were fully covered in all MSDS. Information on chronic toxicity was not provided in two. First aid was addressed in all MSDS, however, in one MSDS the information was presented in highly technical language more applicable to medically trained personnel.

Precautions for use

Five MSDS quoted the correct Australian exposure standard. Of the remainder, two did not provide units though the actual numbers given were correct, and three quoted overseas standards. All MSDS included information on eye protection and respirator type and nine addressed glove type and protective clothing.

Safe handling information

All MSDS addressed clean up of spills/leaks, special equipment for clean up and disposal of spilled material. Six gave advice on precautions for clean-up crews. Regarding storage and transport, all indicated the preferred location for storage of the chemical. Nine MSDS provided advice on storage temperatures. Incompatibilities with other agents were addressed in six MSDS. Fire fighting agents and special precautions were given in nine MSDS whilst information on fire fighting protective clothing was given in six MSDS. Only five gave details of dangerous decomposition products that could result from fire.

Company details

No MSDS provided all required details. One MSDS had no Australian contact details, two had no company address, one had no emergency phone number, two gave no company phone number, and two provided the switchboard number for the emergency phone number. The MSDS Code states that the contact point should not be a general switchboard number and should always be in Australia.

Results of assessment of MSDS for formaldehyde products

Of the ten MSDS examined, one was in the format of an overseas country, and did not fully comply with the format recommended by the MSDS Code of Practice, although it did presented some data which is required by the MSDS Code of Practice. Concentration range of formaldehyde in mixtures as stated in the MSDS was between 0.5% and 30%.
Statement of hazardous nature

Eight MSDS included the statement that the product was hazardous, the overseas one and one other did not include any statement.

Product identification

All MSDS gave the product name. The UN Number, Dangerous Goods Class and Hazchem Code were provided in most MSDS, however, this assessment could not determine whether the data were correct. Only one product had a formaldehyde concentration greater than 25% and this MSDS did not provide the required information. Poisons Schedule was either correct or correctly stated as not having been allocated in most MSDS. Ten MSDS gave the correct chemical name and CAS Number and the proportion of formaldehyde in the product.

Health hazard information

Acute eye and respiratory effects were correctly covered in all MSDS. Acute skin effects were fully addressed in nine MSDS with the remaining one omitting contact dermatitis. Acute oral toxicity was correctly covered in eight MSDS, but two MSDS for products containing >10% formaldehyde omitted to state the product was corrosive. Chronic toxicity was given in nine MSDS and not covered in one.

First aid was generally well covered. One MSDS simply advised contacting a doctor following swallowing. First aid facilities were not addressed in five MSDS.

Precautions for use

No exposure standards were provided in three MSDS and one MSDS gave overseas standards. Personal protective equipment was addressed in most MSDS.

Safe handling information

Storage and transport was not well covered. Six MSDS addressed location for storage and ventilation requirements, five MSDS stated storage temperature ranges and four MSDS stated protection from sunlight and storage incompatibilities. Spills and disposal were covered by most MSDS. Most addressed precautions for the clean-up crew and disposal of recovered material. Fire and hazards of storage were covered by most MSDS, however, decomposition products were addressed in only three MSDS.

Company details

Nine MSDS had all relevant details but one gave overseas contact details.

Results of assessment of MSDS for formaldehyde containing resins

Statement of hazardous nature

All ten MSDS included the correct statement.

Product identification

All MSDS gave the product name. UN Number, Dangerous Goods Class and Hazchem Code were not required as formaldehyde concentrations were less than 25% in all MSDS assessed. As with MSDS for formaldehyde products, NICNAS could not determine whether the data given (which was generic) were correct. Poisons Schedule was given correctly in all MSDS. Nine MSDS gave the correct chemical name, the proportion of chemical in the product and
the CAS Number for formaldehyde. The remaining one contained a melamine/formaldehyde resin and only gave the concentration range for this combined resin and did not give CAS numbers. Concentration ranges for formaldehyde in the resin ranged from < 0.3% to 10%.

**Health hazard information**

Acute toxic effects were covered by most MSDS. One did not provide oral toxicity and two MSDS did not cover chronic toxicity. One MSDS gave health effect information for other components of the resin but not for formaldehyde. One MSDS only gave acute skin toxicity and another omitted skin sensitisation. One MSDS for resin containing up to 10% formaldehyde did not mention corrosive effects.

First aid was covered by all MSDS, however one MSDS incorrectly advised the induction of vomiting following oral ingestion. A discussion of first aid facilities was given in five MSDS.

**Precautions for use**

The correct exposure standard was given in seven MSDS. One provided no exposure standard, another only provided the TWA and a third did not give the exposure standard for formaldehyde. All MSDS included advice on PPE, however, one simply stated that “suitable” protective equipment be used.

**Safe handling information**

Advice on storage location, ventilation and storage temperatures were given in nine MSDS. Storage incompatibilities were given in seven MSDS. Precautions to be taken during clean up and disposal of recovered material were addressed in most MSDS. Six MSDS covered hazards of storage and dangerous decomposition products and most provided information on fire fighting precautions, protective clothing and extinguisher types. Reactions with other agents were addressed in eight MSDS.

**Company details**

All required details were given in all MSDS.

**Results of assessment of MSDS for paraformaldehyde**

Of the 11 MSDS examined, four were in the format of overseas countries and hence did not fully comply with the MSDS Code, although they presented some data which is required by the MSDS Code.

**Product identification**

All MSDS gave the product name and correct UN number. Dangerous Goods Class was correctly given in nine MSDS and Hazchem Code was correct in eight, but was not provided in the other MSDS. Poisons Schedule was correct in six MSDS, not given in four and incorrect in one. Ten MSDS gave the correct chemical name and CAS Number and one did not provide this data. Eight had the proportion of formaldehyde in the product.

**Health hazard information**

Eight MSDS provided the health effects for formaldehyde, however, only two stated that the health effects of paraformaldehyde were due to the decomposition product, formaldehyde. One stated that formaldehyde was a decomposition product, but did not advise that the health effects of paraformaldehyde are due to formaldehyde. One MSDS did not give any details of health effects. One stated the chemical was harmful by various routes of exposure, but did not
detail the toxic effects that would be observed. Chronic health effects were given in three MSDS and not mentioned in eight.

Advice on inducing vomiting following swallowing was the most inconsistent item for the MSDS evaluated. The advice should be ‘Do not to induce vomiting’, and was reported in five MSDS. However, three advised that vomiting should be induced. The other three addressed first aid after swallowing, but did not mention whether to induce vomiting or not. First aid statements following eye, skin and inhalation exposure were correctly given in all MSDS. First aid facilities were described in eight MSDS.

**Precautions for use**

Paraformaldehyde is not listed in the *Exposure Standards for Atmospheric Contaminants in the Occupational Environment* (DEWR, 2004). However, as paraformaldehyde gives off formaldehyde gas, it is considered prudent to provide the exposure standard for formaldehyde. Two MSDS quoted overseas standards stating they applied to formaldehyde and one MSDS gave overseas standards, which were stated as applying to paraformaldehyde. Three MSDS gave the correct Australian standard for formaldehyde, one gave a wrong figure for TWA (8h) and did not quote STEL. Four gave no exposure limits. Most MSDS gave full details of PPE.

**Safe handling information**

The majority of MSDS provided sufficient information on fire/explosion hazard, storage, transport, spills and disposal. Only a few MSDS mentioned state/territory regulations/requirements.

**Company details**

All required details were given in eight MSDS. No Australian company details were in two of the overseas MSDS. One listed all except an emergency telephone number.
Appendix 14

Sample Material Safety Data Sheet for Formaldehyde Solution, 37%

It is understood that stabilisers, such as methanol and various amine derivatives, are added to formaldehyde solutions to reduce the intrinsic polymerisation of formaldehyde. To avoid complications introduced by the additive, this sample MSDS only presents information on a solution containing 37% formaldehyde. The information used in this sample MSDS is from the findings of this assessment and reliable sources. Also, where relevant, only guidance information (text in italic) is provided. Industry is required to create accurate text accordingly in consultation with relevant documents and/or organisations.

### Section 1 - Identification of the Material and Supplier

<table>
<thead>
<tr>
<th><strong>Product name</strong></th>
<th>Formalin</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Other names</strong></td>
<td>Formaldehyde solution, 37%</td>
</tr>
<tr>
<td><strong>Recommended use</strong></td>
<td>Used in resin manufacture, photographic film processing, embalming, leather tanning, lubricant manufacture, fumigation, sanitation treatment, personal care products, and consumer products. Used as a preservative in a wide range of products and in chemical and biological analysis.</td>
</tr>
</tbody>
</table>

| **Company name** | |
| **Address** | |
| **State** | **Postcode** |
| **Telephone number** | **Emergency telephone number** |
Section 2 - Hazard Identification

HAZARDOUS SUBSTANCE - DANGEROUS GOODS.

- Risk phrases
  R23/24/25 – Toxic by inhalation, in contact with skin and if swallowed
  R34 – Causes burns
  R43 – May cause sensitisation by skin contact
  R49 – May cause cancer by inhalation

- Safety phrases
  S1/2 – Keep locked up and out of the reach of children
  S9 – Keep container in a well-ventilated place
  S13 – Keep away from food, drink and animal feeding stuffs
  S24/25 – Avoid contact with skin and eyes
  S26 – In case of contact with eyes rinse immediately with plenty of water
  S27 – Take off immediately all contaminated clothing
  S35 – This material and its container must be disposed of in a safe way
  S36/37 – Wear suitable protective clothing and gloves
  S45 – In case of accident or if you feel unwell, seek medical advice immediately (show label whenever possible)
  S51 – Use only in well ventilated areas
  S52 – Not recommended for interior use on large surface areas
  S53 – Avoid exposure - obtain special instructions before use

Poison Schedule
S6 – Poison

Section 3 - Composition/Information on Ingredients

<table>
<thead>
<tr>
<th>Chemical entity</th>
<th>Proportion</th>
<th>CAS Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water</td>
<td>**%</td>
<td>7732-18-5</td>
</tr>
<tr>
<td>Formaldehyde</td>
<td>37%</td>
<td>50-00-0</td>
</tr>
</tbody>
</table>
Section 4 - First Aid Measures

Inhalation: If inhaled remove from contaminated area. Apply artificial respiration if not breathing.
Skin: If skin or hair contact occurs, remove contaminated clothing and flush skin and hair with running water.
Eyes: If in eyes, hold eyelids apart and flush the eye continuously with running water. Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes.
Swallowed: If swallowed do NOT induce vomiting.

First aid facilities: Ensure eye bath and safety showers are available and ready for use. For advice, contact a Poisons Information Centre (131 126) or a doctor at once.

Advice to doctor
There is no specific antidote for formaldehyde solution poisoning. Treat symptomatically and supportively.

Section 5 - Fire Fighting Measures

Suitable extinguishing media
Water fog. In the absence of fog, a fine spray may be used.

Hazard from combustion products
In a fire, aqueous solutions will evolve irritating gaseous formaldehyde. Formaldehyde solution emits acrid fumes and smoke when heated to decomposition. Decomposition products include carbon dioxide and carbon monoxide.

Precautions for fire fighters and special protective equipment
May be ignited by heat, spark or flames. Vapour may travel to source of ignition and flash back. Containers may explode on heating. If safe to do so, remove undamaged containers from the path of fire. Keep containers cool with water spray.
Fire fighters wear positive pressure self-contained breathing apparatus and chemical protective clothing.

Section 6 - Accidental Release Measures

Emergency procedures
- Fully encapsulating, vapour protective clothing should be worn for spills and leaks with no fire.
- Eliminate all ignition sources (no smoking, flares, sparks or flames in immediate area).
- All equipment used when handling the product must be grounded.
- Do not touch or walk through spilled material.
- Stop leak if you can do it without risk.
- Prevent entry into waterways, sewers, basements or confined areas.
Methods and materials for containment and clean up
Absorb with earth, sand or other non-combustible material and transfer to containers.
Use clean non-sparking tools to collect absorbed material. Small spill may be absorbed
with rags and other absorbing materials. The rags can be then allowed to evaporate
within a fume cupboard before disposal. A vapour suppressing foam may be used to
reduce vapours.
Large spills/leaks
Dike ahead of the liquid spill. Water spray may reduce vapour, but may not prevent
ignition in closed spaces. Contain using absorbent (soil, sand, vermiculite or other inert
material). Prevent run off into drains and waterways.
If contamination of sewers or waterways has occurred, advise local emergency services.
Refer to Section 8 for personal protection equipment.

Section 7 - Handling and Storage

Precautions for safe handling
Broken containers should only be handled by persons wearing appropriate protective
equipment. To reduce electrical hazard, storage tanks should be adequately grounded.
Protect containers from physical damage.
This material is a S6 Poison and must be stored, maintained and used in accordance with
relevant regulations.
Always wash hands before smoking, eating, drinking, or using the toilet. Wash
contaminated clothing and other protective equipment before storing or reuse.

Conditions for safe storage, including any incompatibilities
Formaldehyde meets the criteria for a dangerous good so national
storage and handling regulations for dangerous goods are
applicable. Storage and handling requirements are described in
the NOHSC National Standard for the Storage and Handling of
Workplace Dangerous Goods and NOHSC National Code of Practice for
the Storage and Handling of Workplace Dangerous Goods

Section 6 - Exposure Controls/Personal Protection

National exposure standards*
TWA: 1 ppm (1.2 mg/m³)
STEL: 2 ppm (2.5 mg/m³)
Carcinogen Category: 2
Notice: Skin sensitizer

Engineering controls
Ensure adequate ventilation and air concentration of formaldehyde is controlled below
the occupational exposure standards. Use with local exhaust ventilation.
**Personal protective equipment**

*Please refer to NICNAS Safety Information Sheet on formaldehyde, relevant Australian Standards and consult with manufacturers of personal protective equipment for accurate information.*

Note: Contact lenses use is not recommended when working with this chemical. If contact lenses are worn, appropriate eye and face protection must be used.

### Section 9 - Physical Description and Properties

<table>
<thead>
<tr>
<th>Property</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Appearance</strong></td>
<td>Clear, colourless liquid with pungent, irritating odour.</td>
</tr>
<tr>
<td><strong>PH</strong></td>
<td>2.8 - 4.0</td>
</tr>
<tr>
<td><strong>Boiling point</strong></td>
<td>96 °C (water)</td>
</tr>
<tr>
<td><strong>Melting point</strong></td>
<td>NA</td>
</tr>
<tr>
<td><strong>Vapour pressure</strong></td>
<td>2.26 - 2.66 kPa (at 25 °C)</td>
</tr>
<tr>
<td><strong>Specific gravity (H₂O=1)</strong></td>
<td>1.08 - 1.1 (25 °C)</td>
</tr>
<tr>
<td><strong>Flash point</strong></td>
<td>83 - 85 °C (closed cup)</td>
</tr>
<tr>
<td><strong>Flammability limits</strong></td>
<td>7 - 73%</td>
</tr>
<tr>
<td><strong>Solubility in water</strong></td>
<td>≥100 mg/mL at 20.5 °C</td>
</tr>
</tbody>
</table>

### Section 10 - Stability and Reactivity

**Chemical stability**

Formalin may become cloudy on standing, especially at cool temperatures, and form paraformaldehyde at very low temperatures. Formaldehyde slowly oxidizes in air to formic acid and is sensitive to light. It is easily hydrated and polymerised if not stabilised.

**Conditions to avoid**

Forms paraformaldehyde at very low temperature. Slowly oxidises to formic acid in air and is sensitive to exposure to light.

**Incompatible materials**

Incompatible with ammonia, alkalis, bisulphites, iron preparations, iodine, phenols, potassium permanganate, tannin and salts of copper, iron, and silver, iron.

**Hazardous decomposition products**

When heated to decomposition emits acid smoke and fumes.
Hazardous reactions
Reacts violently with hydrogen peroxide, magnesium carbonate, nitromethane, perchloric acid and aniline, and performic acid and also reacts with strong oxidizers and acids.
Reactions with nitrous oxides (nitrogen dioxide) become explosive at 180 °C. Corrosive to carbon steel as well as copper and its alloys.

Section 11 - Toxicological Information

Acute effects:
LD50 (oral, rats): 800 mg/kg bw
LD50 (dermal, rabbits): 270 mg/kg bw
4-h LC50 (inhalation, rats): 578 mg/m³ (480 ppm)
Gaseous formaldehyde causes sensory irritation (eyes, nose and respiratory tract).
Aqueous formaldehyde is an eye and skin irritant as well as a strong skin sensitizer.

Acute effects of vapour exposure include sensory irritation, such as watery eyes and nose.
The lowest observed-effect level (LOEL) for sensory irritation is 0.5 ppm.
When formaldehyde vapour or liquid comes in contact with the skin, it can produce irritant contact dermatitis. Repeated or prolonged skin contact may lead to allergic contact dermatitis.
When swallowed, formaldehyde solution can cause ulceration and damage along the aero-digestive tract.

Chronic effects:
Based on the available human data, formaldehyde should be regarded as if it may cause cancer in the nose following inhalation exposure.

Section 12 - Ecological Information

Overall:
Harmful to aquatic organisms. Readily biodegradable and does not bioaccumulate. Do NOT allow to enter water, waste water or the soil!

Ecotoxicity
Formaldehyde (as 100%):
Formaldehyde is harmful to fish, LC50 96hr values generally 10-100 mg/L
Acute Fish Aquatic Toxicity LC50 (Atlantic Salmon, 96hrs): 70 mg/L
Acute Fish Aquatic Toxicity LC50 (Rainbow Trout, 96hrs): 60 mg/L
Invertebrates, Algae, Bacteria are more susceptible to formaldehyde, however responses differ widely.
Acute Invertebrate Aquatic Toxicity LC50 (Crustaceans): 14-58 mg/L
Acute Algae Aquatic Toxicity IC50 (Scenedesmus): 0.3-2.5 mg/L
Acute Bacterial Aquatic Toxicity EC50 (E.Coli): 1 mg/L
### Persistence
Readily biodegradable.

### Mobility
Mobile in soil

### Bioaccumulative potential
Does not bioaccumulate.

#### Section 13 - Disposal Considerations

**Disposal methods and containers**
Dispose of container and unused contents in accordance with Federal, State and Local waste regulations.
Advise its corrosive, toxic, sensitising and combustible liquid nature. Dispose by reaction or incineration in an approved waste treatment facility.
Empty containers must be decontaminated.

**Special precautions for landfill or incineration**
Refer to State/Territory waste management authority.

#### Section 14 - Transport Information

<table>
<thead>
<tr>
<th>UN Number</th>
<th>2209</th>
</tr>
</thead>
<tbody>
<tr>
<td>UN proper shipping name</td>
<td>Formaldehyde solution</td>
</tr>
<tr>
<td>Class and subsidiary risk</td>
<td>Class 8</td>
</tr>
<tr>
<td>Packing group</td>
<td>III</td>
</tr>
<tr>
<td>Special precautions for user</td>
<td>Combustible liquid</td>
</tr>
<tr>
<td>Hazchem code</td>
<td>2Z</td>
</tr>
</tbody>
</table>

#### Section 15 - Regulatory Information

**Australian regulatory information**
Formaldehyde is listed on the Australian Inventory of Chemicals Substances (AICS).
**Section 16 - Other Information**

<table>
<thead>
<tr>
<th>Date of preparation</th>
</tr>
</thead>
</table>

**Abbreviations/Acronyms**
- NOHSC - National Occupational Health and Safety Commission
- TWA - Time Weighted Average
- STEL - Short Term Exposure Limit

**Literature references**

*Note: In this report, NICNAS has made a recommendation to the Office of Australian Safety and Compensation Council (OASCC) to lower the occupational exposure standard.*
Appendix 15

Label Assessment

Introduction

Under the National Code of Practice for the Labelling of Workplace Substances (NOHSC, 1994a), hazardous substances in containers of greater than 500 mL(g) capacity require the presence of a list of items. Smaller containers require less detail, but need to draw the attention of persons handling or using the substance to the significant hazards involved.

This assessment examined the following core elements:

- Signal word(s);
- Product name;
- The recognised chemical name of the hazardous ingredient and details of the amount present in the product;
- Risk and safety phrases (refer to the Hazardous Substances Information System (DEWR, 2004));
- First aid procedures;
- Emergency procedures;
- Name and address of the Australian supplier and a telephone number where advice can be obtained; and
- A reference to the MSDS.

Formaldehyde is defined as a dangerous good within the ADG Code and is scheduled (Schedule 6) by the SUSDP. The appropriate signal word for formaldehyde is “POISON” for products containing > 5% formaldehyde.

The appropriate risk and safety phrases for labelling mixtures containing formaldehyde are determined by the concentration cut-off levels of the hazardous substance (DEWR, 2004). In some cases labels gave a range for the concentration of formaldehyde, for example 1% to 5%. It was assumed that the concentration of formaldehyde was the highest in the range specified.

As for MSDS, information supplied on a label for mixtures should be relevant to the mixture as a whole, not its individual constituents, and the information may differ depending on what ingredients are present and in what proportions. A full assessment of labels for mixtures cannot be carried out because an assessment of each ingredient has not been made. If any items of the core elements could not be assessed, its presence or absence was simply noted.

As for MSDS, labels were randomly selected for assessment. However, due to smaller number of labels available, more than one label from the same company were selected. Therefore, the label assessment was not as representative as it could have been as companies tended to repeat the same errors in all their labels.

Table A15-1 shows the number of labels provided to NICNAS in the course of this assessment, and the number selected and assessed against the Labelling Code.
Table A15-1: Number and type of labels received and assessed

<table>
<thead>
<tr>
<th>Label Type</th>
<th>Number Received</th>
<th>Number Assessed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Formalin</td>
<td>21</td>
<td>8</td>
</tr>
<tr>
<td>Formaldehyde products</td>
<td>68</td>
<td>10</td>
</tr>
<tr>
<td>Formaldehyde containing resins</td>
<td>76</td>
<td>10</td>
</tr>
<tr>
<td>Paraformaldehyde</td>
<td>10</td>
<td>Not assessed</td>
</tr>
<tr>
<td>Paraformaldehyde products</td>
<td>nil</td>
<td>Not assessed</td>
</tr>
</tbody>
</table>

Assessment of labels for paraformaldehyde and paraformaldehyde products were not undertaken as the chemical is not listed in the *Hazardous Substances Information System* (DEWR, 2004).

**Results of assessment of labels for formalin**

All labels (8) examined were for containers of greater than 500 ml.

One label contained no signal word. All included chemical identification, proportion of formaldehyde, UN Number and company contact details. Five labels included all risk phrases. One label had R10 which was not necessary and another omitted R40 and R43.

Five labels provided all relevant safety phrases. One label only gave safety phrases S1/2 and another provided the wrong safety phrases apart from S1/2.

First aid statements were completely addressed in three and partially in four labels. One label did not include any information on first aid. Emergency procedures were not addressed in two labels and another two labels advised to dial 000. A reference to MSDS was not given in two labels.

**Results of assessment of labels for formaldehyde products**

Pack size was provided on six out of ten labels assessed. Two were for approximately 20 litres and the remainder were for packs in excess of 25 kg.

The signal word “POISON” was given in only three labels. The Labelling Code requires that where a hazardous substance is not defined as a dangerous good and is not scheduled by the SUSDP, the word “HAZARDOUS” should be used. Of the labels giving “POISON”, only one had a potential concentration of formaldehyde of greater than 5%. The other two gave concentrations of formaldehyde of less than 0.8% and less than 0.25%. These would require the signal word “HAZARDOUS”. The labels which did not give a signal word had formaldehyde concentrations ranging from less than 1% to up to 48%, hence, would have required a signal word, either “HAZARDOUS” or “POISON”.

The UN Number was not required on nine labels and was not provided on the one which required it. Most labels gave full company contact details.

Safety phrases were provided on nine labels, but only six addressed all risk phrases. Three gave no risk phrases and one omitted irritation to the respiratory system.
First aid procedures for swallowing were provided in only two labels, but most addressed first aid following inhalation, skin and eye contact. A reference to an MSDS was not given in three labels. Two labels covered emergency procedures, with the remainder giving no information.

**Results of assessment of labels for formaldehyde containing resins**

Pack size was in excess of 25 kg in seven labels and not provided in other three of the ten labels examined.

The correct signal word “HAZARDOUS” was given on eight of the labels examined.

All labels either provided a statement that no UN Number was allocated or gave a UN Number which may have applied to entities other than formaldehyde or been a generic UN Number.

Product name and proportion of chemicals were provided on all labels. All labels gave full company contact details.

Safety phrases were provided on nine labels, and all labels fully addressed the required risk phrases.

First aid procedures were not well covered. Four labels gave advice on procedures following inhalation, three following swallowing, seven after eye contact and six after skin contact. Two labels advised that vomiting be induced after oral ingestion. A reference to an MSDS was given in all labels. Emergency procedures were detailed on six labels, with the remainder giving no information.
Appendix 16

Proposed Occupational Exposure Standard

The following documents will serve as attachments to the Regulatory Impact Statement (RIS) when the proposed national occupational exposure standard is released for public comment by OASCC.

ATTACHMENT 1

Proposed National Occupational Exposure Standard Released for Public Comment

<table>
<thead>
<tr>
<th>OASCC current exposure standard</th>
<th>Proposed exposure standard</th>
</tr>
</thead>
<tbody>
<tr>
<td>TWA 1 ppm</td>
<td>TWA 0.3 ppm</td>
</tr>
<tr>
<td>STEL 2 ppm</td>
<td>STEL 0.6 ppm</td>
</tr>
<tr>
<td>TWA 1.2 mg/m³</td>
<td>TWA 0.36 mg/m³</td>
</tr>
<tr>
<td>STEL 2.4 mg/m³</td>
<td>STEL 0.72 mg/m³</td>
</tr>
</tbody>
</table>

TWA = the average airborne concentration of a particular substance when calculated over a normal eight-hour working day, for a five-day working week.

STEL = a 15 minute TWA exposure which should not be exceeded at any time during a working day even if the 8-hour TWA average is within the TWA exposure standard. Exposures at the STEL should not be longer than 15 minutes and should not be repeated more than 4 times per day. There should be at least 60 minutes between successive exposures at the STEL.

According to the measured data summarised in the NICNAS Priority Existing Chemical assessment report on formaldehyde, the average levels of formaldehyde around workers’ breathing zone in a number of major use scenarios (long-term and short-term personal sampling) are:

<table>
<thead>
<tr>
<th>Major Use Scenario</th>
<th>Long-term</th>
<th>Exposure (ppm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Formaldehyde manufacture</td>
<td>Most δ 0.2</td>
<td>0.5 ppm (one sample only)</td>
</tr>
<tr>
<td>Resin manufacture</td>
<td>Most δ 0.2</td>
<td>Most δ 0.5</td>
</tr>
<tr>
<td>Product formulation (limited data)</td>
<td>Most δ 0.2</td>
<td>Up to 2</td>
</tr>
<tr>
<td>Pressed wood product manufacture</td>
<td>Most δ 0.3</td>
<td>No data</td>
</tr>
<tr>
<td>Wood working industry using particleboard and MDF</td>
<td>Most &lt; 0.2</td>
<td>No data</td>
</tr>
<tr>
<td>Forensic/hospital mortuaries &amp; pathology laboratories</td>
<td>Most δ 0.3</td>
<td>No data</td>
</tr>
<tr>
<td>Embalming</td>
<td>Most &gt; 0.5</td>
<td>Up to 1.4 (limited data)</td>
</tr>
</tbody>
</table>

MDF, medium density fibreboard.
ATTACHMENT 2

Summary Information to Support the Proposed Occupational Exposure Standard
[Source: NICNAS Priority Existing Chemical Assessment Report on Formaldehyde]

Proposed exposure standard
8-hour TWA: 0.3 ppm (0.36 mg/m3)
STEL: 0.6 ppm (0.72 mg/m3)
Skin sensitiser

The information below has been taken from the National Industrial Chemicals Notification and Assessment Scheme’s (NICNAS) Priority Existing Chemical Assessment Report Number 28 on Formaldehyde.

Basis for setting the limit
Section 11, 16, 18.3 of the Priority Existing Chemical report

Based on the review of the available human and animal data, the critical health effect for setting the occupational exposure standard is sensory irritation. Although formaldehyde is a known eye and upper respiratory tract irritant in humans, the limitations of the available data and subjective nature of sensory irritation do not allow identification of a definitive no-observed-effect level (NOEL). However, the data from chamber studies demonstrates that the sensory irritation responses at levels of 1 ppm (1.2 mg/m3) can definitely be attributed to formaldehyde. Some individuals begin to sense irritation from 0.5 ppm (0.6 mg/m3), although the response rate is often similar to that reported in controls. Although there is limited evidence that some individuals report sensory irritation as low as 0.25 ppm (0.3 mg/m3), the data is very unreliable. Therefore, the lowest-observed-effect level (LOEL) is considered to be 0.5 ppm. Data for asthmatics, who are generally thought to be sensitive to irritants, indicate that they are likely to be no more sensitive than non-asthmatics. This is supported by the absence of direct effects of formaldehyde on pulmonary function in asthmatics in these studies.

In order to protect the majority of workers from sensory irritation, the recommended exposure standard should be a concentration that is a lower than the LOEL identified. As this is a reversible effect and is generally mild at 0.5 ppm, the standard should be slightly lower than the LOEL. For these reasons the recommended exposure standard is 0.3 ppm TWA and 0.6 ppm STEL. At this level, the nasal cancer risk can be also managed. Furthermore, the recommended exposure standards are consistent with best practice overseas and appear technically achievable in most Australian workplaces (based on industry information submitted for this report).

Identity and properties of gaseous formaldehyde
Section 4 and 5 of the Priority Existing Chemical report

| CAS number: | 50-00-0 |
| EINECS number: | 200-001-8 |
| Formula: | CH₂O |
| Synonyms: | Formalin |
| Vapour pressure: | 516 kPa at 25°C |
| Melting point: | -118 to -92 °C |
| Solubility: | 400 to 550 g/L at 25°C |

Formaldehyde
Conversion factor: 1 ppm = 1.2 mg/m³
Formaldehyde is a colourless gas with a pungent, irritating odour at room temperature. The odour threshold of formaldehyde varies widely ranging from 0.05 to 1 ppm. However, for most people, the odour threshold is in the 0.5 to 1 ppm range. Formaldehyde is readily soluble in water, alcohol, and other polar solvents. Formaldehyde is generally available as a 37% to 54% (by weight) aqueous solution, known as formalin.

**Occurrence and uses**

*Section 7 of the Priority Existing Chemical report*

Formaldehyde occurs naturally in the environment, with numerous sources of emission, primarily due to the combustion of organic materials and a variety of natural and human activities including bush fires, animal wastes, plant emissions and both direct and indirect combustion processes. Formaldehyde is also naturally present in the human body at very low concentrations, as a result of various metabolic processes.

Based on 2000-2002 data, approximately 55 000 tonnes formaldehyde per year (calculated as 100% formaldehyde) is manufactured in Australia as formalin solutions. It is also imported as formalin and products/mixtures containing formaldehyde at approximately 90 tonnes a year. Paraformaldehyde, a significant source of formaldehyde, is imported at around 700 tonnes a year as either pure material or mixtures containing paraformaldehyde.

The main industrial use of formalin is for the manufacture of formaldehyde-based resins. These resins are widely used in a variety of industries, predominantly pressed wood manufacture. The majority of the formaldehyde-based resins contain < 0.2% free formaldehyde, but some can contain up to 13%. Formalin is also used directly or in blends in a number of industries including hospitals, mortuaries, medicine-related laboratories, embalming in funeral homes, film processing, leather tanning, and a wide range of personal care and consumer products. The concentrations of formaldehyde in these products range from 40%, such as embalming and film processing solutions, to < 0.2%, for example, the majority of cosmetics and consumer products. Formaldehyde also has agricultural and pharmaceutical uses.

**Occupational exposure**

*Section 15 of the Priority Existing Chemical report*

It is estimated that there are approximately 120 potentially exposed workers in formaldehyde and formaldehyde-based resin manufacturing industry at the four manufacturing sites in Australia. Occupational exposure during formaldehyde manufacturing is generally low due to full containment in enclosed systems. There is no information available on the total number of workers who are potentially exposed to formaldehyde during use of products containing formaldehyde in a wide range of industry categories.

The levels of formaldehyde exposure based on measured data, although limited for most industries, are summarised as follows:

**Long-term (TWA) exposures (personal monitoring data) are:**
- Formaldehyde manufacture: Most $\leq 0.2$ ppm
- Formaldehyde resin manufacture: Most $\leq 0.2$ ppm
- Product formulation (limited data): Most $\leq 0.2$ ppm
- Pressed wood product manufacture: Most $\leq 0.3$ ppm
- Wood working using particleboard and MDF: Most $< 0.2$ ppm
- Forensic/hospital mortuaries & pathology laboratories: Most $0.3$ ppm, up to 3 ppm
- Embalming: most $> 0.5$ ppm, up to 4 ppm

**Short-term exposures (personal monitoring data) are:**

*Formaldehyde*
Formaldehyde manufacture (limited data): 0.5 ppm
Formaldehyde resin manufacture: Most δ 0.5 ppm
Product formulation (limited data): Up to 2 ppm
Pressed wood product manufacture: No data
Wood working using particleboard and MDF: No data
Forensic/hospital mortuaries & pathology laboratories: No data
Embalming (limited data): Up to 1.4 ppm

**Workplace air monitoring methods**

*Section 6 of the Priority Existing Chemical report*

A number of sampling and analytical methods are available for measuring formaldehyde in air at the workplace. An air sample can be obtained by a filter and impinges, solid sorbent tube or cartridge and quantified by spectrometry, high performance liquid chromatography (HPLC) or gas chromatography. Other methods, such as passive sampler/monitor followed by chromatropic acid test and gas tube detector with infrared analysers, are also used in Australia.

Instantaneous measurement of the concentration of airborne formaldehyde by direct read, hand-held electronic formaldehyde devices is commonly used in Australia, for example, formaldehyde meters and Interscan machines.

**Toxicokinetics**

*Section 9 of the Priority Existing Chemical report*

Formaldehyde is readily absorbed at the site of contact by all exposure routes due to its high reactivity with biological macromolecules, high water solubility and low molecular weight.

It is rapidly metabolised after absorption, with a half-life of about 1 to 1.5 minutes in blood circulation following intravenous administration in animals. Formaldehyde is metabolised to formate by a number of widely distributed cellular enzymes in which formaldehyde dehydrogenase is the most important one. A minor pathway for formaldehyde metabolism is oxidisation to formic acid by the enzyme catalase.

Due to the rapid metabolism of formaldehyde, much of the material is expired in air shortly after exposure, and as formate in urine.

**Health effects**

**Animal studies**

*Section 10 of the Priority Existing Chemical report*

Following acute exposure via inhalation, dermal and oral routes, formaldehyde is moderately toxic in animals. Formaldehyde solution is known to be a skin and eye irritant and strong sensitisers.

Following repeated inhalation exposure, the target organ is the nasal tract where the observed effects include alterations in mucociliary clearance, cell proliferation and histopathological changes (cytotoxicity and hyperplasia) to the nasal epithelium at doses ε 2 ppm. The principal non-neoplastic effect observed in animals after repeated oral dosing is irritation at the site of contact (i.e. fore- and glandular-stomach). The limited data available on the repeated dermal toxicity of formaldehyde solution indicate skin irritation and no evidence of systemic toxicity.

Formaldehyde is genotoxic in vitro, and it appears that the chemical may be genotoxic at the site of contact in vivo.
A significantly increased incidence of nasal squamous cell carcinomas was observed in rats exposed by long-term inhalation at concentrations > 6 ppm formaldehyde. However, these were not observed in mice and hamsters at equivalent or greater exposure concentrations. The available data in animals do not support formaldehyde being carcinogenic by the dermal or oral routes.

Limited data indicated that formaldehyde does not produce reproductive or developmental effects in animals.

**Human data**

*Section 11 of the Priority Existing Chemical report*

There are old reports of human deaths following ingestion of formaldehyde. Recent cases reported ulceration and damage along the aero-digestive tract that needed surgical operations following ingestion of approximately 700 mg/kg of formaldehyde solution.

Sensory irritation has been reported in human epidemiological and chamber studies following inhalation exposure to formaldehyde. However, the limitations of the available data and subjective nature of sensory irritation do not allow identification of a definitive no-observed-effect level (NOEL). The chamber studies suggest that sensory irritation definitely occurs at ≥1 ppm (≥1.2 mg/m³) with some individuals beginning to sense irritation from 0.5 ppm (0.6 mg/m³) (Bender, 2002). Although asthmatics are thought to be more sensitive to irritants, studies by Green et al. (1987), Sauder et al. (1986; 1987) and Witek et al. (1987) have demonstrated that at concentrations of 2 to 3 ppm (2.4 to 3.6 mg/m³) for up to 3 hours, asthmatics were not particularly sensitive to formaldehyde.

Skin sensitisation by formaldehyde solution is clearly observed in numerous clinical trials and case reports in humans.

Epidemiology data from occupational studies investigating cytogenetic effects in nasal and buccal cells are suggestive of formaldehyde having a weak localised genotoxic activity, while the evidence for a systemic activity, including peripheral lymphocytes, is equivocal.

Many epidemiology studies have investigated formaldehyde exposure and cancers of the respiratory tract. The strongest evidence of an association has been observed for nasopharyngeal cancers. The most recent meta-analysis (Collins et al., 1997) concluded that although there was an increased, non-significant risk of nasopharyngeal cancers, overall, the data did not provide sufficient evidence to establish a causal relationship between nasopharyngeal cancers and formaldehyde exposure. Studies published since the meta-analysis provide mixed results for both case-control studies and cohort studies. Three large industrial cohort studies with a long follow-up have been recently published (Hauptman et al. 2004, Pinkerton et al., 2004 and Coggon et al., 2003). The study by Hauptman et al. (2004) found that compared to the national population, there was a significantly increase risk of nasopharyngeal cancer. In addition, the relative risk increased with average exposure intensity, cumulative exposure, highest peak exposure and duration of exposure to formaldehyde. However, no such cancers were seen in the study by Pinkerton et al. (2004), while no increased risk was seen by Coggon et al. (2003). Similarly, mixed results have been observed in recent case-control studies of formaldehyde exposure and nasopharyngeal cancer.

Overall, although it cannot be definitely concluded that occupational formaldehyde exposure results in the development of nasopharyngeal cancer, there is some evidence to suggest a causal association between formaldehyde exposure and nasopharyngeal cancer. In addition, the postulated mode of action is considered likely to be relevant to humans and is biological plausible. Therefore, based on the available nasopharyngeal cancer data, formaldehyde should be regarded as if it may be carcinogenic to humans following inhalation exposure.
There are several case-control studies that indicate an increased risk for sinonasal cancer and formaldehyde exposure, but this has not been observed in cohort studies. The most recent meta-analysis (Collins et al., 1997) concluded that the data did not suggest an association between formaldehyde and sinonasal cancer. There is limited and inconsistent evidence with respect to laryngeal and lung cancers. Overall, the available data do not support an association between sinonasal, laryngeal and lung cancers and formaldehyde exposure.

An increased risk of leukaemia, occasionally significant, has been inconsistently reported in human epidemiology studies. The available data do not allow construction of a dose-response relationship for formaldehyde exposure and incidence of leukaemia. Additionally, there is currently no biologically plausible mode of action to explain why formaldehyde would be leukaemogenic. Overall, the available human and animal data are insufficient to establish an association between formaldehyde exposure and leukaemia.

Based on animal and limited epidemiology data, formaldehyde is unlikely to cause reproductive and developmental effects at exposures relevant to humans.

References


Appendix 17

AUSTRALIAN WOOD PANELS ASSOCIATION

MDF PLANT
FORMALDEHYDE AIR DISPERSION

Report No 79365
May 2006

EML AIR PTY LTD
ABN 98 006 678 342
417-431 Canterbury Road Surrey Hills VIC 3127
Telephone (03) 9836 1999 Facsimile (03) 9836 0517

EML GROUP OF LABORATORIES
Consulting Chemists and Microbiologists
MELBOURNE • SYDNEY • BRISBANE
EXECUTIVE SUMMARY

Australian Wood Panels Association (AWPA) requested that the dispersion of formaldehyde from an MDF Plant emitting 27,000 kilograms per year be compared for two (2) scenarios.

The first air dispersion modelling scenario attempts to replicate the model used by CSIRO Atmospheric Research “Formaldehyde Air Quality Assessment” Report C/0928 for NICNAS May 2004. This scenario used the annual national pollutant inventory data for a large wood products plant from year 2000. The formaldehyde emissions to air were assumed by CSIRO to be split, 50 percent each between stack and fugitive sources, for an assumed flat ground site location.

The second scenario, described in the following report, considers the same quantity of annual formaldehyde emissions being emitted from both stack and fugitive sources but based on a more representative site source emission profile. The emission profile used in the model has been distributed to the various sources as follows:

i. a layout that is more representative of an MDF products plant;
ii. proportioned to reflect different stack source emission test ratios; and
iii. assumes that the estimated levels of fugitive emissions are the difference between those accounted for from stack source tests and the total emission data.

The peak predicted 24-hour average and annual average ground level concentrations from both air dispersion model scenarios are compared and discussed. Using a typical plant layout and test emission profile for the stack sources has predicted formaldehyde ground level concentrations, at the same locations, which are an order of magnitude lower than the CSIRO predicted concentrations.
1. INTRODUCTION

Australian Wood Panels Association requested that the dispersion of formaldehyde from an MDF Plant emitting 27,000 kgs p.a. be compared in two (2) scenarios. The first would review the CSIRO Atmospheric Research “Formaldehyde Air Quality Assessment” Report C/0928 for National Industrial Chemicals Notification and Assessment Scheme (NICNAS) May 2004 and the second as typical plant layout and source emission profile.

The Ausplume Gaussian plume dispersion model (EPAV Version 6) has been used for these predictions of ground level concentration (g.l.c.) 24-hour average and annual averages.

2. BACKGROUND

The draft report by NICNAS “Formaldehyde: Priority Existing Chemical Assessment” in Appendix 6 “Modelling of Atmospheric Concentrations of Formaldehyde” Section A1, Modelling Methodology, acknowledges a potential significant source for error in the predicted environmental concentrations (PEC’s):

- “…. details of the source characteristics for each facility in the NPI database, which is beyond the scope of this modelling”.

Furthermore the National Pollutant Inventory (NPI) Timber and Wood Product Manufacturing Emission Estimation Factors for the particle board dryers and press emissions are coded as D (Below average) and E (Poor). Where Australian facilities have not had available extensive emission tests conducted then considerable variances can arise from actual emissions should the modelling be conducted on data from a facility that has been mostly reliant on using the NPI factors.

The Appendix 6 Section A.2.2, ‘Wood and Paper Product Manufacturers’ has further commented that:

- “…. A sensitivity analysis showed that the PEC’s are much more sensitive to the configuration of the source of the fugitive emissions than the stack emissions. …. Given the high PEC’s, it would be appropriate to verify these predictions by obtaining more information about source configurations for these industries”.

Table A.6-2 Summary of predicted maximum annual and 24-hour average PEC’s concludes that for Wood and Paper Industries that average emitters may have concentrations 100 metres from the plant of 4.8 ppb and 36 ppb respectively.

These average PEC’s are consistent with the requirement of EPA’s in Australia that new or altered facilities had to meet dispersed formaldehyde ground level concentrations of 50 ppb (3-minute average) up until the late 1990’s and since then, 33 ppb (3-minute average).

However the predicted maximum annual and 24-hour average PEC’s concludes that for Wood and Paper Industries largest emitters the concentrations at 100 metres could be 16 ppb to 119 ppb respectively.
The highest predicted 24-hour average is significantly at variance with the following:

- Typical Australian State EPA dispersed design criteria for formaldehyde as 3-minute average \( \leq 33 \text{ ppb} \) or 1-hour average \( \leq 18 \text{ ppb} \) which would be substantially lower if modelled at the longer averaging periods of 24-hours and annual.
- Fugitive emissions concentrations are predominantly reflected by measurement of workplace exposures. The wood panels industry has data indicating that most workplaces are \( < 20 \text{ ppb} \) (8-hour average). At the perimeter, 100 metres from the source of these fugitive concentrations, the ambient concentrations on a 24-hour and annual average will be much lower.
- Ambient measurements at the boundary of two (2) wood panel facilities in Australia have reported \( < 10 \text{ ppb} \) 24-hour average.

These three aspects are indicative that the modelling by CSIRO for NICNAS has generally over-predicted ground level concentration of formaldehyde at the boundary of wood product industries, particularly in the case of largest emitters. The actual annual and 24-hour average concentrations are more likely to be similar to the PEC’s predicted for the other 6 categories of large emitter industries listed in the CSIRO report Table A6-2.

3. METHODOLOGY

The AWPA requested that the model used by CSIRO Atmospheric Research “Formaldehyde Air Quality Assessment” Report C/0928 for NICNAS May 2004 be replicated. The CSIRO scenario used the annual national pollutant inventory data for a large wood product plant from year 2000. The formaldehyde emissions to air were assumed by CSIRO to be split, 50 percent each between stack and fugitive sources, for an assumed flat ground site location.

The AWPA further requested a second modelling scenario as described in the following report. This model considers the same quantity of annual formaldehyde emissions being emitted from both stack and fugitive sources as the CSIRO model. However a more representative site source emission profile has been used in the model by distributing the various source emissions as follows:

- a site layout that is more typical of an MDF products plant;
- proportioned to reflect typical stack source emission test ratios; and
- assumes that the estimated levels of fugitive emissions are the difference between those accounted for from stack source tests and the total emission data.

Attachment 1 Air Dispersion Model Inputs compares the CSIRO Atmospheric Research “Formaldehyde Air Quality Assessment” Report C/0928 for National Industrial Chemicals Notification and Assessment Scheme (NICNAS) May 2004 inputs with the EML Air typical MDF plant scenario input data.

Total annual emissions from stack and fugitive sources of 27,000 kg p.a. are identical in both cases. However the distribution is dissimilar because the CSIRO report splits the emission 50 percent each between the two (2) sources whereas the EML plant scenario for stack emissions is proportioned across the typical MDF plant (heat plant; dryers; press hoods & baghouses) based on actual stack tests. These stack sources represent 95.7 percent of the total emissions.

For the EML volume sources the emission is the difference between total annual emission and stack source total emission; i.e. only 4.3 percent of the total emission.

The CSIRO modelling assumed the stack and fugitive sources to be adjacent whereas the EML scenario assumed a spread of the sources as shown in a site layout (not to scale) Attachment 2. Stack heights; diameters; discharge temperatures and discharge
velocities vary markedly as shown in Attachment 1. Again the EML scenario is based on test results for these sources.

Similarly the EML scenario volume sources are assumed to be emitting from doors and roof ridge rather than a single source in the CSIRO model.

The EML model assumes a building present whereas the CSIRO model has not considered the specific influence of wake effects on the dispersion from and around a typical large plant building.

The receptor system is the same in both cases with a discrete receptor placed 100 metres from the sources in an easterly direction. The EML Air scenario places this receptor 100 metres from the eastern end of the building where fugitive and baghouse emissions are prominent and press hood emissions will be entrained in the building wake.

Both models use the meteorological file used by CSIRO (CSIRO Atmospheric Research personal communication).

To assist interpretation of the modelling predictions the EML Air scenario includes three (3) source groups i.e. all sources, stack sources only, and the fugitive sources only.

4. RESULTS

The predicted ground level concentrations (or PEC’s as referred to in the CSIRO report) are summarised in Table A17-1 below.

Table A17-1: Predicted ground level concentration

<table>
<thead>
<tr>
<th>Source Group</th>
<th>Annual average Predicted concentration ppbv (mg/m³)*</th>
<th>24-hour average Predicted concentration ppbv (mg/m³)*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CSIRO at receptor</td>
<td>Plant scenario at receptor</td>
</tr>
<tr>
<td>All</td>
<td>15 (0.018)</td>
<td>2 (0.0028)</td>
</tr>
<tr>
<td>Stacks only</td>
<td>Not reported</td>
<td>1 (0.0016)</td>
</tr>
<tr>
<td>Fugitive only</td>
<td>Not reported</td>
<td>1 (0.0012)</td>
</tr>
</tbody>
</table>

Note * ppbv = (mg/m3 ÷ 1.2) x 1000 @ 30°C same as CSIRO report.

In particular the results in Table A17-1 can be summarised as follows:

i. The CSIRO reported predictions of 16 ppb (annual average) and 119 ppb (24-hour average) are almost identical to the 15 & 122 re-modelled by EML Air for all sources. The small difference has probably arisen in selection of model factors to take account of roughness factors for location.

ii. The EML Air typical plant scenario predicted ground concentrations are an order of magnitude lower than the CSIRO concentrations.

iii. The EML Air typical plant scenario illustrates that stack and fugitive sources are predicted to be contributing almost equally to the predicted concentration.
5. SUMMARY

The re-modelling of the CSIRO scenario for a large MDF plant emitting 27,000 kilograms per annum of formaldehyde has verified the predicted environmental concentrations reported to NICNAS for a large source.

Using emission test results from the stack sources typical of a large MDF plant and:

i. Distributing these stack emissions to a plant layout scenario; and
ii. Assigning emissions to the fugitive sources (roof ridge and doorways) as the difference between the annual sum of stack emissions and 27,000 kg p.a.; then

- the modelling predicted environmental concentrations from a scenario plant configuration are an order of magnitude lower when compared to the CSIRO model (as shown in Table A17-1 above).

Regardless of modelling using the typical test and plant configuration these predicted concentrations are 3 times higher than measurements at the boundary of two (2) wood panel facilities in Australia who reported < 10 ppb 24-hour average.

Geoff White
Senior Environmental Consultant

References

ATTACHMENT 1

AUSTRALIAN WOOD PANELS ASSOCIATION

MDF PLANTS
FORMALDEHYDE AIR DISPERSION

AIR DISPERSION MODEL INPUTS
<table>
<thead>
<tr>
<th>Model Input</th>
<th>CSIRO</th>
<th>Plant scenario</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Formaldehyde</strong></td>
<td>27000 kg x 1000 grams (÷ 365 days; 24 hours; 60 minutes; 60 seconds) = 0.86 g/sec</td>
<td>27000 kg x 1000 grams (÷ 365 days; 24 hours; 60 minutes; 60 seconds) = 0.86 g/sec</td>
</tr>
<tr>
<td><strong>Stack sources</strong></td>
<td>1 only</td>
<td>16 total</td>
</tr>
<tr>
<td></td>
<td>30 metre</td>
<td>2 Heat plant</td>
</tr>
<tr>
<td></td>
<td></td>
<td>15 metres</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2 Dryers</td>
</tr>
<tr>
<td></td>
<td></td>
<td>15 metres</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2 Press lines 10 stacks 15 m.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2 Baghouse</td>
</tr>
<tr>
<td></td>
<td></td>
<td>6 metres</td>
</tr>
<tr>
<td><strong>Stack temperature</strong></td>
<td>25°C</td>
<td>Heat plants</td>
</tr>
<tr>
<td></td>
<td></td>
<td>200°C</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dryers</td>
</tr>
<tr>
<td></td>
<td></td>
<td>50°C</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Press hoods</td>
</tr>
<tr>
<td></td>
<td></td>
<td>30°C</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Baghouse</td>
</tr>
<tr>
<td></td>
<td></td>
<td>20°C</td>
</tr>
<tr>
<td><strong>Stack exit velocity</strong></td>
<td>10 m/sec (2 m diameter)</td>
<td>Heat plants 20 m/sec (1.5m dia.)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dryers 15 m/sec (1m dia.)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Press hoods 10 m/sec(1m dia.)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Baghouse 8 m/sec (1m dia.)</td>
</tr>
<tr>
<td><strong>Stack emissions</strong></td>
<td>50 percent</td>
<td>Heat plants 0.007 g/sec</td>
</tr>
<tr>
<td></td>
<td>0.43 g/sec</td>
<td>Dryers 0.635 g/sec</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Press hoods 0.175 g/sec</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Baghouse 0.006 g/sec</td>
</tr>
<tr>
<td><strong>Volume sources</strong></td>
<td>1 only</td>
<td>3 Doors</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Roof ridge</td>
</tr>
<tr>
<td><strong>Initial spread factors</strong></td>
<td>Horizontal &amp; vertical 5 metres</td>
<td>Doors</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Roof ridge 50 &amp; 0.2 metres</td>
</tr>
<tr>
<td><strong>Source height</strong></td>
<td>10 metres</td>
<td>Doors 3 metres</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Roof ridge 10 metres</td>
</tr>
<tr>
<td>Model Input (cont.)</td>
<td>CSIRO</td>
<td>Plant scenario</td>
</tr>
<tr>
<td>---------------------</td>
<td>-------</td>
<td>----------------</td>
</tr>
<tr>
<td>Volume emissions (fugitive)</td>
<td>50 percent 0.43 g/sec</td>
<td>Doors¹ 0.0148 g/sec&lt;br&gt;Roof ridge² 0.0222 g/sec</td>
</tr>
<tr>
<td>Source locations</td>
<td>Stack and Volume sources adjacent</td>
<td>Dryers &amp; heat plants outside west end of building&lt;br&gt;Press hoods inside west end of building: 2 lines parallel&lt;br&gt;Baghouses south side &amp; mid and west end of building&lt;br&gt;Doors south side &amp; mid and west end of building: and 1 door east end of building&lt;br&gt;Roof ridge entire length of building</td>
</tr>
<tr>
<td>Building wake effects</td>
<td>Nil</td>
<td>Building 100 x 25 x 12 metres&lt;br&gt;Orientated east-west</td>
</tr>
<tr>
<td>Receptor system</td>
<td>Polar 100 metre radii to 1000 metres and 5 degree steps</td>
<td>Polar 100 metre radii to 1000 metres and 5 degree steps</td>
</tr>
<tr>
<td>Discrete receptor</td>
<td>100 metres due east of sources</td>
<td>100 metres due east of building at 1 metre height</td>
</tr>
<tr>
<td>Receptors</td>
<td>Polar 100 metres radii @100 metre intervals and 5 degree steps</td>
<td>Polar 100 metres radii @100 metre intervals and 5 degree steps</td>
</tr>
</tbody>
</table>

Notes: Plant scenario volume source emission based the following –
- Total formaldehyde emission 0.860 g/sec.
- Total stack source test emission 0.823 g/sec
- Difference as fugitive 0.037 g/sec
- 40% assigned to doors 0.015 g/sec
- 60% assigned to roof ridge 0.022 g/sec
ATTACHMENT 2
AUSTRALIAN WOOD PANELS ASSOCIATION

MDF PLANTS
FORMALDEHYDE AIR DISPERSION

PLANT SCENARIO: SOURCE LAYOUT

(DATA NOT INCLUDED IN NICNAS PEC REPORT)
Plan scenarios schematic

Emissions Heat plant, Dryers, Presses and Baghouses based on MDF plant 2005 tests
Building 100 x 25 x 12
Press hood vents 10 off 3 metre above roof height
Appendix 18

REVIEW OF MDF PLANT - FORMALDEHYDE AIR DISPERSION, FOR AUSTRALIAN WOOD PANELS ASSOCIATION, EML REPORT 79365, MAY 2006

prepared for

NICNAS
(National Industrial Chemicals Notification and Assessment Scheme)

by

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Summary

The modelling in the EML report appears to provide a good description of the configuration of the sources of formaldehyde emissions at a large MDF plant. The use of stack test measurements to determine stack emissions is appropriate. However the method of estimating fugitive emissions is subject to large uncertainties. This is a critical issue because the 24-hour average predicted environmental concentrations (PECs) are significant compared to EPA design criteria and are the largest identified in the NICNAS report. The large uncertainty in the fugitive emissions is due the large uncertainties inherent in the NPI total emissions estimates and to the fact that the fugitive emissions are only a small fraction of the total formaldehyde emissions. For example, allowing for an uncertainty of 5% in the NPI total could reduce the fugitive emission estimate to zero or more than double it to 2,500 kg p.a. The latter case would produce a maximum 24-hour average PEC of 79 ppb, compared to 37 ppb estimated in the EML report. Reducing the uncertainty in the fugitive emission estimate is not straightforward. A possible method would be to measure/estimate the volume flows and formaldehyde concentrations of the fugitive emissions from the building ridge vent and doors.

The review comments on and provides suggestions on a range of issues in the EML report relating to the estimates of formaldehyde PECs for a large MDF plant.

1. Introduction

This is a review for NICNAS of the EML report for the Australian Wood Panels Association titled MDF Plant, Formaldehyde Air Dispersion, May 2006, (Report No 79365).

The EML report addresses the issue of refining PEC results for the largest formaldehyde emitter identified in the NICNAS report for wood and paper manufacturing industries. The impacts of formaldehyde emissions from such a plant are modelled to determine the predicted environmental concentrations (PECs). These PECs are presented as an update to those presented in an earlier CSIRO report.

The CSIRO report (Formaldehyde Air Quality Assessment, May 2004) had assumed that 50% of the emissions from such a plant were released from a 30 m stack and 50% were fugitive emissions which were treated as a volume source centred at a height of 10 m. This simple configuration was “based on the source descriptions in the NICNAS report”. The CSIRO report noted that “A detailed analysis would require details of the source characteristics for each facility in the NPI database, which is beyond the scope of this study.” The results obtained for wood and paper product manufacturers concluded that “Given the high PECs, it would be appropriate to verify these predictions by obtaining more information about source configurations for these [Wood and Paper] industries.” This is the issue addressed by the EML report.

The results from the EML modelling are reproduced in Table A18-1.
2. Modelling approach in EML report

The improved modelling approach in the EML report is to use:
   i. a site layout that is more typical of an MDF products plant;
   ii. emissions proportioned to reflect typical stack source emission test results; and
   iii. estimated levels of fugitive emissions that are the difference between those accounted for from stack source tests and the total NPI emission estimates.

We consider that the first two points are basically sound. However, there is a major problem in the modelling because of uncertainty in the estimate of fugitive emissions derived using the method given in point (iii).

3. Specify the plant being modelled

The issue addressed in the EML report is the PEC results for the largest emitter in the wood and paper manufacturing industries. A more detailed study could consider the configurations of all large formaldehyde emitters in the wood and paper manufacturing industries, but the methodology adopted in the NICNAS report is to consider the largest emitter. Because NPI estimates and stack test results are used to determine the fugitive emissions, it is essential that both be for the same plant. Although it is inferred that this was done in the EML report, it is not explicitly stated that this was the case. This needs to be clarified.

The NPI database indicates that the largest emitter referred to in the NICNAS report reported formaldehyde emissions of 27,082 kg p.a. in the 2001 – 2002 reporting year. This facility reported similar emissions in the following two years: 27,134 kg in 2002-2003; and 26,718 kg in 2003-2004. This indicates that the value used in the model was indicative.
4. Uncertainties in the fugitive emission estimate

Because the fugitive emission estimate (≈1000 kg p.a.) is determined as the difference between two large numbers (27,000 and 25,950), it is critical that the impact of uncertainty in these large numbers be taken into account in the calculation. The EML report does not include any uncertainty estimates. [Note that the EML report uses both kg p.a. and g/sec emission estimates; rounding errors in the conversion produce small differences when comparing the numerical values estimated in different units.]

Uncertainties in the estimate of the magnitude of fugitive emissions arise from (in approximate order of importance):

i. uncertainty in the NPI estimate of total formaldehyde emissions;

ii. uncertainty in the total stack emission rates derived from the stack testing on up to 16 separate stacks;

iii. uncertainty due to differences in the production rate assumed for the NPI estimates and that prevailing at the time the stack testing was undertaken.

i. *Uncertainty in NPI estimates*

As mentioned in the EML report, the degree of certainty of the emission factors used in the NPI estimate of 27,000 kg p.a. has been identified as D (below average) or E (poor), see *NPI Emission Estimation Technique Manual for Timber and Wood Product Manufacturing, 11 January 2002, Version 1.1*. Although uncertainty estimates are not given in the manual, it would appear reasonable to conclude that these descriptors correspond to an uncertainty of at least 20% in the NPI estimates (i.e. 27,000 ± 5,400 kg p.a.). In the estimate below, we consider the impact of uncertainties of 20%, 10% and 5% in the NPI estimate.

ii. *Uncertainty in total stack emission rate*

The uncertainty in the total stack emissions testing involving measurements on up to 16 separate stacks is likely to be much smaller than that in the NPI estimate; a guess estimate is 5%. However, in the absence of more detailed information and the large uncertainty in the NPI estimate, we ignore this uncertainty in estimating the uncertainty in the fugitive emission rate.

iii. *Uncertainty due to differences in the production rate*

No information is available to make an estimate of the uncertainty due to differences in the production rate assumed for the NPI estimates and that prevailing at the time the stack testing was undertaken. Thus we also ignore this uncertainty in estimating the uncertainty in the fugitive emission rate.

The EML report determined the fugitive emission rate to be 0.037 g/sec. This is equal to 1170 kg p.a. Taking into account possible uncertainties of 20%, 10% and 5% in the NPI total emission rate would give the following averages and range of values for the fugitive emissions:

- 1170 kg p.a. (range 0 – 6600 kg p.a.) for 20% uncertainty in NPI estimate
- 1170 kg p.a. (range 0 – 3900 kg p.a.) for 10% uncertainty in NPI estimate
- 1170 kg p.a. (range 0 – 2500 kg p.a.) for 5% uncertainty in NPI estimate.

These indicate that even a 5% uncertainty in the NPI estimate could more than double the fugitive emissions. Scaling the PEC results in Table 18A-1 produces the following range of maximum 24-hour average PECs:

- 0 kg p.a. fugitives: 33 ppb (only stack impact)
- 2,500 kg p.a. fugitives: 79 ppb
That is, the model results are very sensitive to uncertainties in the NPI estimate. Because the 24-hour average PECs are significant compared to NEPM Air Toxics formaldehyde investigation level (40 ppb, 24-hour average) and are the largest identified in the NICNAS report, further work is needed to reduce the uncertainty in the fugitive emission estimate.

5. Possible method for reducing uncertainty in fugitive estimate

It is very difficult to see how to reduce the uncertainty in the fugitive emission estimate computed as the difference between the NPI estimated total emissions and the measured stack total. The alternative is to use some other method for estimating fugitive emissions, for example to measure/estimate volume flows (air exchange rates) and 24-hour average formaldehyde concentrations in the fugitive emissions from the roof ridge and doors.

6. Justification for 40:60 split of fugitives between door:roof ridge

No justification is provided for using a 40%:60% split of the fugitive emissions between the door and roof ridge. Is this important? This could be determined by looking at the sensitivity of the PEC estimates to changes in this split?

7. Ausplume Modelling

We consider that the set-up of the model, the inclusion of buildings, stacks and fugitive sources is appropriate. The 24-hour average PECs were calculated on a radial grid at 5° increments and at spacings from 100 m to 1000 m at 100 m increments. This is considered to be suitable for determining the maximum 24-hour average PEC. However, the annual average concentrations were only determined at a single discrete receptor, which was located 100 m east of the main plant building. This is indicated by the data listed in Attachment 3 (Air Dispersion Model Outputs). Although this may be the location of the highest annual average PEC for the current fugitive/stack emission characteristics it is possible that this point of maximum concentration will change for other configurations. The annual average PECs should be calculated on the same grid as that used for the 24-hour averages.

The EML Ausplume modelling used a 1997/1998 meteorological file for Paisley in the west of Melbourne. This is the same as that used by CSIRO. Although meteorology from this site was appropriate for the CSIRO assessment of the impact of emissions from a wide range of industries as well as urban and roadway sources, it is recommended that for modelling the impact of the largest formaldehyde emitter (in the wood and paper manufacturing industries), it would be more appropriate to use a local meteorological file (either from measurements or generated by TAPM) for the site of the largest emitter.

8. Other comments

The end of Section 2 of the EML report lists a number of observations, which it is suggested indicate “that the modelling by CSIRO for NICNAS has generally over-predicted ground-level concentrations of formaldehyde at the boundary of wood product industries, particularly in the case of the largest emitters”. We list these observations and provide our response to each.

- EML report: Typical Australian State EPA dispersed design criteria for formaldehyde as 3-minute average < 33 ppb or 1-hour average ≤ 18 ppb, which would be substantially lower if modelled at the longer averaging period of 24-hours and annual.
CSIRO response: The implication of including this statement is that all plants meet current EPA design criteria. However, even the modelling in the EML report indicates that emissions from the stacks alone (ignoring the contribution from fugitives) produce 24-hour average PECs up to 33 ppb at a distance of 100 m from the source. This is substantially larger than the 18 ppb 1-hour average listed as an EPA criterion. It is modelling and measurements that are relevant for determining PECs, not current standards.

- **EML report:** Fugitive emissions concentrations are predominantly reflected by measurement of workplace exposure. The wood panels industry has data indicating that most workplaces are < 20 ppb (8-hour average). At the perimeter, 100 m from the sources of these fugitive concentrations, the ambient concentration on a 24-hour and annual average will be much lower.

CSIRO response: The NICNAS analysis is concerned with maximum values, not typical values. What would be relevant here is the maximum workplace exposure, not readings from “most workplaces”. Furthermore, the relevance of workplace exposure measurements to the concentration in the fugitive emissions depends on the ventilation arrangements in the facility. Ventilation systems are generally designed to minimise workplace exposure, for example by drawing fresh air in around the workspace and exhausting it through roof vents. Thus measurements of workplace concentration could be far lower than concentrations in fugitive exhaust air.

- **EML report:** Ambient measurements at the boundary of two wood panel facilities in Australia have reported <10 ppb 24-hour averages."

CSIRO response: The NICNAS analysis is concerned with maximum values, not typical values. Were the measurements made downwind of the largest emitter? Were the measurements made under conditions when the maximum 24-hour averages could be expected to occur? Without further information, such data are not relevant to maximum 24-hour average PECs.
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