



Workplace Exposure Standards

Consideration Paper on Lowering the Workplace Exposure Standard for Formaldehyde

June 2010

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EXECUTIVE SUMMARY

This paper discusses a proposed addition to the Workplace Exposure Standard (WES) for formaldehyde and the basis for that proposal. The paper also discusses setting WES under the Hazardous Substances and New Organisms (HSNO) Act 1996, health effects of exposure to formaldehyde, and lists formaldehyde exposure standards from other countries.

The Department of Labour is proposing the following WES for formaldehyde:

- 1. An 8 hour WES-TWA (Time Weighted Average) of 0.3ppm; and**
- 2. Retaining the WES-Ceiling of 1ppm.**

The formaldehyde occupational exposure limits from 27 other countries or standard setting organisations are listed in this paper. The proposed eight hour WES-TWA of 0.3ppm is equivalent to three of 15 TWAs listed, more stringent than ten of the 15, and less stringent than two of the 15. The current WES-Ceiling limit is equivalent to four of the 17 Ceiling limits listed, more stringent than four of the 17, and less stringent than nine of the 17.

SUBMISSIONS ON THE REPORT

In order to assist the Department of Labour make its final decision on the Workplace Exposure Standards for formaldehyde, submissions on this report are invited. For further details, please refer to: <http://www.dol.govt.nz/consultation/index.asp>. The closing date for feedback is Friday, 30th July 2010.

GLOSSARY OF TERMS

Cilia: Fine hair-like projections from cells in the respiratory tract that "sweep" in unison to remove fluids and particles. Ciliated cells and mucus secreting cells in the respiratory tract have been termed the "muco-ciliary escalator".

Cytotoxic: Toxic to cells.

DNA adducts: A complex formed by the binding of a chemical substance to the DNA. Such complexes will activate DNA repair, and unless repaired prior to DNA replication, may result in changes (mutations) in the DNA.

DNA cross-links: Occur when a compound reacts with two different positions in the DNA; either in the same strand or in opposite strands. Cross-links can also occur between DNA and proteins. DNA replication is blocked by cross-links, which causes replication arrest and cell death if the cross-link is not repaired.

Dyspnoea: Difficult or laboured breathing; shortness of breath.

Genotoxic effect: Alterations to the structure, information content, or segregation of DNA including: DNA damage caused by interference with its normal replication processes, and temporary non-physiological alterations to its replication.

Histopathological: Abnormal changes in the minute structure of cells and tissues (assessed in terms of their functional consequences). For example, with formaldehyde, damage to the epithelial tissues, including reddening, sloughing of cells, leakage of plasma, bleeding, hyperplasia, metaplasia, and a breakdown of cellular components, leading to cellular death (WorkSafe, 2009).

Hyperplasia: An increase in the number of cells.

Hyperproliferation: An abnormally high rate of cell division leading to rapid hyperplasia.

Lachrymation: The production, secretion and shedding of tears.

Metaplasia: Abnormal transformation of differentiated tissue from one type of cell to another type of cell.

Mucosa: Relating to the mucous membranes lining various tubular structures and tracts.

Nasopharynx: The area of the upper throat that lies behind the nose.

Necrosis: Pathological, premature death of cells and living tissue.

Respiratory Epithelium: The outside layer of cells of the respiratory tract.

Rhinitis: Inflammation and swelling of the mucous membrane of the nose, characterized by a runny nose and stuffiness.

Squamous cells: Scale-like cells that make up most of the outer layers of the skin, and line parts of the respiratory tract and digestive tract.

SECTION 1: WORKPLACE EXPOSURE STANDARDS

Workplace Exposure Standards (WES) are health-based guidelines for airborne contaminants and are endorsed by the Department of Labour ("the Department"). The current WES publication (Workplace Exposure Standards and Biological Exposure Indices from 2002) is available at: <http://www.osh.dol.govt.nz/order/catalogue/329.shtml>.

WES are intended to be used as guidelines for those involved in occupational health practice. In assigning the standards, defining a level that will achieve freedom from adverse health effects is the major consideration. Compliance with the designated value does not, however, guarantee protection from discomfort or possible ill-health outcomes for all workers. The range of individual susceptibility is wide and it is possible that workers will experience discomfort or develop occupational illness from exposure to substances at levels below the exposure standards.

In all instances the WES relate to exposure that has been measured by personal monitoring using methods that gather air samples in the worker's breathing zone.

The WES publication defines the following categories of WES:

- **Time Weighted Average (WES-TWA)** - The time-weighted average exposure standard designed to protect the worker from the effects of long-term exposure. This is based on an eight hour working day and a 40 hour working week.
- **Ceiling (WES-Ceiling)** - A concentration that should not be exceeded during any part of the working day.
- **Short-Term Exposure Limit (WES-STEL)** - The 15 minute average exposure standard. Applies to any 15-minute period in the working day and is designed to protect the worker against adverse effects of irritation, chronic or irreversible tissue change, or narcosis that may increase the likelihood of accidents. The WES-STEL is not an alternative to the WES-TWA; both the short-term and time-weighted average exposures apply.
- **General Excursion Limit (WES-GEL)** - Often there is insufficient toxicological data available for the establishment of a Short Term Exposure Limit. Peak exposure should, however, still be controlled even in situations where the Time-Weighted Average level is not exceeded. A 15-minute exposure limit of three times the TWA is recommended. Where a STEL has been assigned, the STEL value takes precedence over the general excursion limit regardless of whether or not it is a stricter standard.

There is some confusion as to whether WES apply to air inside or outside respiratory protective equipment. The intention of the WES is to establish a concentration that can be inhaled without causing adverse health effects. If a respirator is worn, the WES applies to the concentration inside the respirator

as this is the air that the worker will be breathing. As inward leakage of air occurs with respiratory protection (due to poor face seals and valve leakage etc), an important consideration when selecting a respirator is the reduction in exposure the respirator can be expected to provide. This reduction, termed 'protection factor', is defined as the ratio of the concentration of the contaminant outside the respirator to the concentration inside the respirator i.e. breathed by the wearer. Suppliers of respirators provide information on the protection factors afforded by their masks.

The protection factor can also be expressed by the following equation (AS/NZS 1715:2009):

$$\text{Protection factor} = \frac{\text{Ambient air concentration}}{\text{Concentration inside respirator}}$$

The protection factor required of a respirator to reduce exposure to an accepted level is called the 'required minimum protection factor'. It can be expressed by the following equation:

$$\text{Required minimum protection factor} = \frac{\text{Ambient air concentration}}{\text{Exposure Standard}}$$

For example, if the air concentration (in the worker's breathing zone) is 10ppm and the exposure standard is 1ppm, the required minimum protection factor of a mask is (at least) 10.

However, in such situations where the air concentration in the worker's breathing zone exceeds the exposure standard, all practicable steps must be taken as a priority to eliminate or isolate exposure (providing suitable respiratory protection only until this is achieved). However, if elimination or isolation are not practicable, and respiratory protection is used in the longer term to minimise exposure, again a respirator with an appropriate protection factor should be worn. In the above example, the required minimum protection factor of a mask is at least 10. However, as formaldehyde exposure should be reduced as much as practicable, then a protection factor greater than 10 should be the aim in this case.

SECTION 2: WORKPLACE EXPOSURE STANDARDS UNDER HSNO

2.1 Setting WES under HSNO

Formaldehyde and certain formulations containing formaldehyde are approved substances under the HSNO Act and are subject to controls established by that legislation.

Section 77B(1)(b) of the HSNO Act allows the Environmental Risk Management Authority to provide for the setting of exposure limits for approved substances. Such exposure limits may comprise workplace exposure standards. Section 77B(3)(b) requires the setting of the exposure limits to be according to a methodology.

On this basis, the Authority has provided for the setting of a formaldehyde WES by the Department of Labour. The methodology by which the exposure limit (WES in this case) is set is defined in an operational agreement between the Department and ERMA.

(Extracted from Section 77B(5)): Before setting exposure limits, the Authority must:

- a) consider the best international practices and standards for the safe management of substances with toxic or ecotoxic properties; **and**
- b) be satisfied that, against other specified exposure limits that apply to the substance,—
 - (i) the proposed exposure limit is more effective in terms of its effect on the management, use, and risks of the substance; **or**
 - (iii) the proposed exposure limit is more likely to achieve its purpose; **and**
- c) do everything reasonably practicable on its part to advise all people who in its opinion may be affected by the proposed exposure limit; **and**
- d) give those people a reasonable opportunity to make submissions and comments to the Authority on the proposed exposure limit; **and**
- e) consider all submissions and comments received.

In considering an alteration of the formaldehyde WES, the Department:

- considered the best international practices and standards for the safe management of formaldehyde (S77B(5)(a)); and,
- is satisfied that, against other specified exposure limits that apply to formaldehyde, that the proposed exposure limit is more effective in terms of its effect on the management, use, and risks (S77B(5)(b)(i)); and,
- is satisfied the proposed exposure limit is more likely to achieve its purpose (S77B(5)(b)(iii)).

In complying with sections 77B(5)(c), the Department has done everything reasonably practicable to advise all people who in its opinion may be affected by the proposed exposure limit, and to give those people a reasonable opportunity to make submissions and comments on the proposed exposure limit. This has been achieved by:

- a mail-out on 29th November 2008 alerting affected people to the proposed change and requesting submissions by 31st January 2009 (Appendix A), and
- a web posting on the Department of Labour Health and Safety site on 1st December 2008 alerting to the proposed change and requesting submissions by 31st January 2009, and
- a mail-out on the 19th December 2008 informing of an extension to the submission period (Appendix A) to 31st March 2009, and,
- a review of the submissions received, prompting a decision to hold a public meeting to discuss the issues raised in the submission process, and,
- a mail-out on the 19th August 2009, to people affected by the proposed exposure limit to inform them of a public meeting to discuss the proposed change to the WES (Appendix A). This meeting was held on 25th November 2009, and
- a web posting on the Department of Labour Health and Safety site on 26th August 2009 alerting to the public meeting on 25th November 2009, and
- a web posting on the Department of Labour Health and Safety site on 5th November 2009 with a position paper, and
- an email sent on 9th November 2009 to the confirmed attendees to the public meeting with a Department position paper on the proposed WES reduction (Appendix A).

2.2 WES as Controls under HSNO

Regulation 29(4) of the Hazardous Substances (Classes 6, 8 and 9 Controls) Regulations 2001 requires that "The person in charge of a place of work must ensure that a person is not exposed to a concentration of the substance that exceeds the workplace exposure standard for that substance". Therefore, workers' exposure to formaldehyde must be controlled to levels below the WES.

HSNO legislation does not provide guidance on achieving control, however the Health and Safety in Employment Act (1992) requires that where a significant hazard exists, employers must take all practicable steps to eliminate it. If it is not practicable to eliminate the significant hazard, it must be isolated, and if it is not practicable to isolate, it must be minimised. The steps of elimination, isolation and minimisation (called the 'hierarchy of controls') should be applied to formaldehyde exposures that exceed the WES.

SECTION 3: HEALTH EFFECTS OF FORMALDEHYDE EXPOSURE

The target organs affected by formaldehyde vapour are the eyes, nose and throat (DECOS, 2003).

Inhalation is the primary route of exposure with no indication that formaldehyde is absorbed to any extent through the skin (WorkSafe BC, 2009). When inhaled, it reacts rapidly at the site of contact and is quickly metabolised in the respiratory tissue (NICNAS, 2006).

3.1 Acute health effects

The predominant effect of short-term exposure in humans is sensory irritation experienced firstly in the eyes, followed by perception of the odour and then irritation of the nose and throat accompanied by discomfort, lachrymation, sneezing, coughing, nausea and dyspnoea (DECOS, 2003).

Acute health effects relate to formaldehyde's irritative and inflammatory properties. It readily reacts with biological tissues, particularly the mucous tissues lining the respiratory tract and the eyes.

WorkSafe, BC (2009) states "As a result of its reactivity, inhaled formaldehyde is rapidly and almost entirely absorbed by the mucous tissues lining the upper respiratory tract, penetrating no farther than the major bronchi of the respiratory tract at low or medium concentrations. The mucous tissues of the eyes are also a susceptible target... Localized irritation of mucous epithelial tissues by formaldehyde, if prolonged, can lead to localized tissue damage; basically the cytotoxic effects of formaldehyde may result in histopathological changes".

DECOS (2003) report that histopathological changes range from slight hyperplasia and squamous-cell metaplasia of the ciliated and non-ciliated respiratory epithelium in specific areas, to severe rhinitis, necrosis and extensive hyperplasia and metaplasia of major portions of the nasal respiratory epithelium.

3.2 Chronic health effects

Data on the adverse effects in humans based on long term (chronic) exposures to formaldehyde is minimal and is based almost entirely on animal studies (WorkSafe, 2009).

DECOS (2003) reports that "*effects after long-term inhalation exposure to formaldehyde in experimental animals include inflammatory, degenerative and regenerative changes of the nasal mucosa and squamous cell carcinomas of the nasal epithelium*". The non-cancer nasal changes range from a minimal degree of hyperplasia and squamous-cell metaplasia of the

nasal respiratory epithelium to severe rhinitis, necrosis and extensive restorative hyperplasia and metaplasia of the nasal respiratory epithelium.

3.3 Sensitisation

Evidence clearly indicates that formaldehyde solution is a skin irritant and a strong skin sensitiser.

As to whether formaldehyde is a respiratory sensitiser, the following views are reported:

- *“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” (NICNAS, 2006).*
- *“There is no convincing evidence of formaldehyde being able to sensitize the respiratory tract” (DECOS, 2006).*
- *“Most studies suggested that exposure to formaldehyde in air at concentrations up to 3ppm, with or without moderate exercise, does not cause significant broncho-constriction in subjects with mild asthma when the formaldehyde was inhaled through a mouthpiece. Formaldehyde seems to act more commonly as a direct airway irritant in persons who have bronchial asthma from other causes” (ACGIH, 2001).*

Taken in conjunction, these reviews suggest that formaldehyde is not a proven cause (inducer) of asthma via allergic mechanisms, and furthermore, is not a powerful aggravator of pre-existing asthma (despite the potential of airway irritants to exacerbate this condition). However, such an aggravating effect cannot be completely excluded, especially in those with moderate to severe asthma exposed to relatively high concentrations. There also remains the theoretical possibility that high concentrations might induce ('de novo') a form of asthma described as 'reactive airways dysfunction syndrome' (RADS), as this asthmatic condition is produced by marked irritation and inflammation of the airways, rather than via allergic mechanisms.

3.4 Reproductive toxicity

Based on animal and limited epidemiology data, formaldehyde is unlikely to cause reproductive and developmental effects at exposures relevant to humans (NICNAS, 2006).

This relates at least in part to the very low levels of formaldehyde distributed to distant sites via the blood, due to its high reactivity with tissues of first contact, and its generally modest respiratory intake (limited by the intolerability of inhaling highly irritant air levels for lengthy periods of time).

3.5 Carcinogenicity and mutagenicity

The International Agency for Research on Cancer (IARC) concluded in 2004 that there is sufficient evidence in humans and experimental animals for the carcinogenicity of formaldehyde. In their updated 2009 summary of evaluations IARC concluded that:

- Formaldehyde causes cancer of the nasopharynx
- There is sufficient evidence in humans for a causal association of formaldehyde with leukaemia
- There is limited evidence in humans for a causal association of formaldehyde with sinonasal cancer
- Formaldehyde causes nasal cavity tumours in rats (IARC, 2009).

However, these views in their entirety were not shared by all the IARC expert panel members. Furthermore, the view that formaldehyde is a confirmed human carcinogen is not shared by all organisations. Some of these are summarised as follows:

- The American Conference of Governmental Industrial Hygienists (ACGIH) classifies formaldehyde as A2 - suspected human carcinogen (ACGIH, 2001). A2 is used mainly where there is limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in animals with relevance to humans (ACGIH, 2009)
- The Dutch Expert Committee on Occupational Standards in co-operation with the Nordic Expert Group (DECOS/NEG) concluded that "*the currently available epidemiological database on formaldehyde does not provide evidence for a respiratory tract cancer risk at exposure levels lower than 0.3 mg/m³ (LOAEL for sensory irritation)*" (DECOS, 2006).
- The Working Group on Action to Control Chemicals (WATCH) is the British government's scientific and technical subcommittee of the UK Health and Safety Executives Advisory Committee of Toxic Substances. In a 2005 paper on the carcinogenicity of formaldehyde it reports:

"There have been over 50 epidemiological investigations of cancer in formaldehyde-exposed workers that have been periodically reviewed by various authorities, covering data available up until 2000. Consistently the conclusion has been that although some individual studies are suggestive of formaldehyde having caused nasopharyngeal or nasal cancer in exposed populations, the overall strength of evidence had fallen short of showing a clear and causal association with formaldehyde. However, a number of new epidemiology studies have been reported since 2000. Some of these provide further evidence in relation to formaldehyde exposure and cancer of the upper respiratory tract and also there are some concerns raised for leukaemia... Given the importance of formaldehyde as a chemical and the significance and

consequences of pronouncing a substance as a “human carcinogen”, WATCH was asked to consider the strength of evidence that formaldehyde has caused cancer in humans. WATCH concluded that formaldehyde has probably caused naso-pharyngeal cancer in humans via a mechanism to which it can be predicted that both chronic inflammation (provoked by irritancy) and genotoxicity contributed. In relation to the apparent association seen in some studies between formaldehyde exposure and leukaemia, based on recent reviews of the evidence, and also considering biological plausibility, WATCH concluded that there is no basis for any significant concern for this cancer” (WATCH, 2005).

- In 2006 the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) which is part of the Australian Government Department of Health and Ageing assessed the current state of knowledge on formaldehyde. In the report NICNAS concluded that:

“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”. They went on to classify formaldehyde as a Category 2 carcinogen with risk phrase R49 ‘May cause cancer by inhalation’. They stated: “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” (NICNAS, 2006).

A significant part of the debate is the risk of cancer as a function of exposure levels. As noted in the DECOS report (2006), human and animal studies demonstrate a distinctly positive dose–response relationship for formaldehyde, with statistically significant increased risk not demonstrated with low exposures. This may be because as noted above (WATCH, 2005), a number of experts believe that, with respect to the respiratory tract, a carcinogenic effect from formaldehyde depends on a combination of it’s genotoxicity and its ability to produce chronic tissue inflammation causing

enhanced cellular turnover, the inflammation effect being concentration-dependant.

Others argue, at least on animal study data, that very low risks at low levels cannot be excluded because of the unavoidable limitations in sample sizes, and most theoretical modelling does not totally exclude all risk at very low concentrations e.g. the CIIT model (Conolly et al, 2004).

Studies of the human response to formaldehyde exposure indicate that due to its pungent and unpleasant odour, it is difficult to distinguish between irritation (localized changes to the cells and tissue) from 'chemosensory stimulation' (stimulation of local nerve endings). It should be noted that chemosensory stimulation will not necessarily lead to cellular irritation and any resultant histopathological changes (WorkSafe BC, 2009). Thus subjective responses to exposure can occur at levels too low to cause actual tissue cytotoxicity.

Formaldehyde is considered comprehensively genotoxic *in vivo* in a variety of experimental systems, ranging from bacteria to rodents (DECOS, 2003). Genotoxic effects include the formation of DNA-protein cross-links ('DPX') and other indicators of genetic damage (WorkSafe, 2009). Incomplete repair of such DPX can lead to the formation of mutations, in particular chromosome mutations and micronuclei in proliferating cells. However such findings per se give little idea about the dose-effect relationship *in vivo*. However, *in vitro* tests suggest such effects are related to the concentrations tested (Speit et al, 2007).

Note: Mutagenicity is the ability of chemicals to cause changes in the genetic material (DNA) in the nucleus of cells in ways that allow changes to be transmitted during cell division. The effects of this can be quite different, depending on whether the affected cells are germinal cells (ova or sperm) or other (somatic) cell types. Mutations in somatic cells are not inheritable, but may result in cell death or transmission of a genetic defect to other cells in the same tissue through mitotic division. Because the initiating event of chemical carcinogenesis is thought to be a mutagenic one, mutagenicity tests are often used to screen for potential carcinogens. (However, not all mutagens appear to be carcinogens, and not all carcinogens are mutagens). In contrast, genotoxicity covers a broader spectrum of endpoints than mutagenicity, that is, some genotoxic effects are not mutagenic effects as they are not transmissible either from cell to cell or generation to generation. Such effects include unscheduled DNA synthesis, sister chromatid exchanges, and DNA strand breaks (Klaassen, 2008). Formaldehyde has both mutagenic and other genotoxic properties.

SECTION 4: PROPOSED WES FOR FORMALDEHYDE

4.1 Proposed Department of Labour Workplace Exposure Standard for Formaldehyde

The Department proposes to retain the current WES-Ceiling Limit of 1ppm and to introduce an 8-hour WES-TWA of 0.3ppm. Sources given in Sections 4.2 to 4.9 informed this decision.

Previously, the Department proposed introducing a WES-STEL of 0.6ppm (Appendix A). This was reconsidered, as a STEL (15 minute exposure standard) could result in very high short exposures. For example: Personal samples are taken over a 15 minute period. The average exposure for 3 minutes is 2ppm, the exposure for the rest of the 15 minute period is 0.2ppm, then the time weighted average exposure for the 15 minute exposure period is:

$$\frac{(2\text{ppm} \times 3 \text{ minutes}) + (0.2\text{ppm} \times 12 \text{ minutes})}{15 \text{ minutes}} = 0.56\text{ppm}$$

In this example, although a WES-STEL of 0.6ppm is not exceeded, the exposure over a very short period is higher (less stringent) than the current WES.

Formaldehyde and formulations containing >1% formaldehyde are classified as category 6.7A under the HSNO classification scheme. 6.7A is a substance that is a known or presumed human carcinogen. ERMA's website provides more detail on this classification:

www.ermanz.govt.nz/hs/t&c/HSNOUGTC.pdf

Formaldehyde and formulations containing >1% formaldehyde are also classified as 6.8B i.e. a substance that is a suspected human mutagen. ERMA's website provides more detail on this classification:

www.ermanz.govt.nz/hs/t&c/HSNOUGTC.pdf

Formaldehyde is listed as a sensitizer in the Department of Labour's Workplace Exposure Standards book. Refer to the Department of Labour's website for more detail: <http://www.osh.govt.nz/order/catalogue/329.shtml>

4.2 ACGIH (2001)

The American Conference of Governmental Industrial Hygienists (ACGIH) reports that one of the difficulties in deriving a formaldehyde Threshold Limit Value (TLV) is that *"it would appear that an individual's tolerance of formaldehyde in the air is determined by the subjective perception of the inconvenience or discomfort caused by eye and upper airway irritation. Sensory irritation is not easily measured by a quantitative clinical test"*. ACGIH went on to report studies showing mucous membrane irritation occurred at concentrations as low as 0.05ppm. The ACGIH did not present an NOAEL, but referred to the US National Research Council (NRC) Committee on Toxicology in 1980 which concluded that *"there was no population*

threshold for the irritant effects of formaldehyde in humans and that, at concentrations below 0.25ppm, it expects less than 20% of an exposed human population would react and experience slight irritation of the eyes, nose and throat and a possible decrease in nasal mucus flow".

The ACGIH set a TLV-Ceiling limit of 0.3ppm based on irritation but concluded that the ceiling limit "may not eliminate all worker complaints of sensory irritation". The ACGIH went on to say: "In view of the reported dose-dependant carcinogenic activity for rat and mouse inhalation of formaldehyde, the report of macromolecular adducts in the upper and lower respiratory tracts of nonhuman primates following inhalation of formaldehyde, the human case reports of upper respiratory tract malignant melanoma associated with formaldehyde inhalation and the suggestive epidemiologic data on human cancer risk, ACGIH recommends that workplace formaldehyde air concentrations be reduced to the lowest levels that can be achieved using engineering controls. While the epidemiologic studies and case reports [referenced in the document] do not confirm that occupational exposure to formaldehyde is carcinogenic for human beings, several groups [referenced in the document] have reported that occupational formaldehyde exposure may be a factor in the potentiation or predisposition of upper respiratory tract cancer when a worker is also exposed to other known or suspected occupational carcinogens". The ACGIH also stated, in its 1991 Documentation of the Threshold Limit Values and Biological Exposure Limits, that "interpretation of the biologically based animal cancer risk assessment data indicate that the oncogenic potential of formaldehyde is a threshold phenomenon and that prevention of upper respiratory irritation and the associated regenerative hyperplasia should eliminate, for all practical purposes, any excess risk posed by occupational formaldehyde exposure alone" (Paustenbach, 1997).

The ACGIH has assigned an A2 classification to formaldehyde. A2 is a suspected human carcinogen and is assigned where the epidemiologic studies are conflicting or are insufficient to confirm an increased risk of cancer in workers (ACGIH, 2001).

4.3 German MAK (2002)

The German Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area (the MAK Commission) assigns occupational maximum concentration values (MAK values). In 2002 an eight hour time weighted average of 0.3ppm, a short term exposure limit of 0.6ppm and a ceiling limit of 1ppm was assigned. The 0.3ppm level was set on the basis of protection from eye irritation. As the MAK Commission considers that the cause of cell proliferation is the irritancy effect on the upper respiratory tract, the avoidance of cell proliferation is a consideration in establishing the MAK value (WorkSafe, 2009).

The 2006 MAK Commission reportedly made the following observations:

“Evaluations of the data now available for the carcinogenic effects confirm that the occurrence of tumours in the nasal mucosa of rats and mice are the result of chronic proliferative processes caused by the cytotoxic effects of formaldehyde. The dose-response relationship for all the parameters investigated, such as damage to the nasal epithelium, cell proliferation, and tumour incidence and also the formation of DNA cross-links is very flat for low level exposures and become much steeper at higher levels of exposure. As long as the cellular proliferation rate is not increased – which requires exposure to concentrations in excess of 2ppm – the probability that DNA-protein crosslinks are transformed into mutations is considered low” (WorkSafe, 2009).

The MAK commission 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) (NICNAS, 2006).

4.4 DECOS and NEG (2003)

In 2003 the Dutch Expert Committee on Occupational Standards (DECOS) in co-operation with the Nordic Expert Group for Criteria Documentation of Health Risks from Chemicals (NEG) published the document *Formaldehyde - Health-Based Recommended Occupational Exposure Limit*.

Reviewing the data at the time, the committees estimated that 0.25ppm is the lowest observed adverse effect level (LOAEL) at which sensory irritation may occur “in a low but significant percentage of exposed workers”.

The report recommended a health-based occupational exposure limit of 0.12ppm (eight hour TWA). The report stated this was low enough to protect workers against nasal tissue damage, and as a consequence, also against the potential risk of nasal cancer. Based on the LOAEL the committees applied a safety margin of two, which they considered large enough to prevent significant sensory irritation in workers, “taking into account that:

- (I) the critical effect (sensory irritation) is a local effect,
- (II) the incidence of the effect at 0.25ppm is low (19%) and may not be different from the background incidence in controls and,
- (III) minimal sensory irritation may rapidly subside due to accommodation”.

Note 1: *The term accommodation comes from the Paustenbach paper (1997). Although the term is not explained fully, it appears to refer to a tolerance or acclimatisation that was observed in one study, whereby subjects reported increasing discomfort as formaldehyde exposure increased, but only up to a point when irritation stabilised thus indicating acclimatisation had occurred.*

The DECOS report further considered whether an exposure limit of 0.12ppm would be low enough to protect workers against cytotoxic-induced

hyperproliferation of the nasal respiratory epithelium and against the potential risk of nasal cancer.

The report stated that "Nasal carcinomas in rats have only been found after exposure to high, cytotoxic concentrations causing rhinitis, necrosis and regenerative hyperplasia and squamous metaplasia of the nasal respiratory epithelium. The crucial role of tissue damage followed by hyperplasia and metaplasia of the nasal respiratory epithelium in formaldehyde carcinogenesis has been demonstrated in a convincing way, has meanwhile been widely recognized, and has been included in human cancer risk assessment of formaldehyde. The committees found it reasonable to conclude that the response of the respiratory tract to formaldehyde will be qualitatively similar in rats and humans. If in humans exposure of formaldehyde is accompanied by recurrent tissue damage at the site of contact, formaldehyde may be assumed to have carcinogenic potential in man via mechanisms of cytotoxicity.

Correspondingly, if the respiratory tract tissue is not recurrently injured, exposure of humans to relatively low, non-cytotoxic levels of formaldehyde can be assumed to be associated with a negligible cancer risk. Both committees [DECOS and NEG] observed that the majority of short- and long-term inhalation studies with formaldehyde in experimental animals reveals a NOAEL of 1.2 or 2.4mg/m³ (1 or 2ppm). However, in a few studies slight histopathological changes of the nasal respiratory epithelium were observed at levels ranging from 0.36 to 2.4mg/m³ (0.3 to 2ppm) formaldehyde. Three meta-analyses of human epidemiological studies have shown inconsistent results. In two of them a significant relation between exposure to formaldehyde and nasopharyngeal cancer risk was observed. The association between formaldehyde exposure and nasal cancer was ambiguous. However, according to the committees, in these meta-analyses the authors did not correct for the unreported studies in which no cases of nasal cancers were found. This must have led to an overestimation of the overall relative risk of nasopharyngeal cancer. In the third, more recent, published meta-analysis, a correction was made for underreporting, and the authors concluded that there was no support for a causal relation between formaldehyde exposure and nasopharyngeal cancer".

The DECOS and NEG committees endorsed this conclusion and further concluded that the currently available epidemiological data on formaldehyde does not provide evidence for a respiratory tract cancer risk at exposure levels lower than 0.25ppm (LOAEL for sensory irritation) (DECOS, 2003).

4.5 WATCH (2005)

The Working Group on Action to Control Chemicals (WATCH) is the British government's scientific and technical subcommittee of the UK Health and

Safety Executives Advisory Committee of Toxic Substances. The WATCH paper on the carcinogenicity of formaldehyde reported: *"In humans, sensory irritation of the eyes and respiratory tract occurs at levels above 0.3 to 0.5ppm, with eye irritation being the more sensitive endpoint. Moderate irritation of the eyes, nose and throat occurs at 2 - 3ppm"*.

4.6 NICNAS (2006)

In 2006 the National Industrial Chemicals Notification and Assessment Scheme (NICNAS), which is part of the Australian Government Department of Health and Ageing, assessed the current state of knowledge on formaldehyde in the document Priority Existing Chemical Assessment Report No. 28 Formaldehyde (NICNAS, 2006). In the report NICNAS stated:

"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)". It went on to report studies that showed sensory irritation at concentrations as low as 0.25ppm. NICNAS considered that the Lowest-Observed-Adverse-Effects-Level (LOAEL) for irritation is 0.5ppm and recommended a workplace exposure standard of 0.3ppm TWA and 0.6ppm STEL. The critical effect that was used as the basis for their proposed exposure standards was sensory irritation. In setting these standards NICNAS stated; *"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 0.5ppm, the standard should be slightly lower than the LOEL. At this level, the nasal cancer risk* can be also managed"*. The current occupational exposure limit for formaldehyde in Australia remains at an eight hour TWA of 1ppm and a STEL of 2ppm.

*NICNAS states: *"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"*.

4.7 IRRST (2006)

In 2006 IRSST (Institute de Reserche Robert Sauvé en Santé et en Sécurité du Travail) published a report *Impact of Lowering the Permissible Exposure Value for Formaldehyde – Health Impact of an Occupational Exposure to Formaldehyde*. The report was based on current studies on the dose-response relationship between exposure to formaldehyde and the appearance of health effects. The report concluded:

"Our analysis indicates that, for concentrations less than 0.75ppm, the frequency of irritation in workers exposed to formaldehyde was about the same as the one observed in individuals without occupational exposure. This means that appearance of irritation at such concentrations can hardly be associated with occupational exposure to formaldehyde. For concentrations between 0.75 and 3 ppm, the estimated proportion of workers who may experience moderate irritating effects to the eyes, nose, and throat, attributed to formaldehyde is between 1.6 and 14.9%. It was estimated that at most 2% of workers could have severe eye irritation. In the case of occupational exposure, we cannot exclude the fact that factors other than formaldehyde, such as wood dust, could be the cause of irritating effects or they could increase the probability that these effects would occur through synergy with formaldehyde" (IRRST, 2006).

4.8 SCOEL (2008)

The European Union Scientific Committee on Occupational Exposure Limit Values (SCOEL) is mandated to advise the European Commission on occupational exposure limits for chemicals in the workplace.

In 2008 SCOEL published a report *Recommendation from the Scientific Committee in Occupational Exposure Limits for Formaldehyde*. SCOEL regards formaldehyde as a genotoxic carcinogen. To derive an OEL, taking into account the carcinogenic risk, SCOEL considered the avoidance of cell proliferation is critical. SCOEL considered the cause of cell proliferation is the irritant effect on the upper respiratory tract. As the data is insufficient to establish a clear NOAEL value for upper respiratory tract irritation, the NOAEL for eye irritation can be used and is a more sensitive parameter (SCOEL, 2008). SCOEL considers that the onset of eye irritation provides a safety margin to the onset of irritation-induced cytotoxicity and cell proliferation. Based on this reasoning, SCOEL recommends an occupational exposure limit be set at, or below the NOAEL for sensory irritancy of the eye. SCOEL proposed an eight hour exposure limit of 0.2ppm and stated that this value *"especially considers possible inter individual differences in susceptibility to irritation by formaldehyde, which may be expected based on the entire body of data"*. SCOEL proposed that short term irritation may be prevented by a 15 minute STEL of 0.4ppm.

4.9 WorkSafe British Columbia (2009)

WorkSafe British Columbia has an occupational exposure limit of 0.3ppm (8 hour TWA) and a Ceiling Limit of 1ppm for formaldehyde. In 2009 WorkSafe BC reviewed occupational exposure limits for formaldehyde, and retained current limits.

In the review, WorkSafe reported that the weight of evidence suggests that 1ppm is the irritant threshold, i.e. the dividing line between adverse and non-adverse health effects. WorkSafe considered these health effects to be nasal injury* in humans and concluded that below 1ppm, adverse health

effects are not observed; the odour is detectable but not considered annoying or irritating, and concluded that the No Observed Adverse Effects Level (NOAEL) is 1ppm (Worksafe, 2009).

*Although not specifically defining nasal injury, the following statements from the report give an indication what Worksafe consider nasal injury:

“Localized irritation of mucous epithelial tissues by formaldehyde, if prolonged, can lead to localized tissue damage; basically the cytotoxic effects of formaldehyde may result in histopathological changes. Evidence indicates that tissue damage in experimental animals occurs at airborne formaldehyde concentrations above 4 – 6ppm. ... Recent studies, involving testing of human response to airborne formaldehyde, indicate that due to the pungent and unpleasant odour of formaldehyde, it has been difficult to distinguish: a) mucous tissue irritation (localized changes to the cells and tissue) from b) chemosensory stimulation which involves stimulation of local nerve endings. Chemosensory stimulation will not necessarily lead to cellular irritation and any histopathological changes. Earlier studies conducted on establishing levels of irritation experienced by test subjects failed to distinguish actual mucous tissue irritation from chemosensory irritation. Several expert panels have recently reviewed and researched this apparent dichotomy, and have concluded that cytotoxic effects of formaldehyde do not appear to occur at levels below 0.75 to 1.0ppm formaldehyde. A level of 1.0ppm is considered the no-observed-adverse-effect-level (NOAEL) for nasal injury in humans. This is further supported by the fact that at concentrations below 1.0ppm, symptoms disappear very quickly, underlying the non-adversity of the irritating effects at low concentrations”.

SECTION 5: FORMALDEHYDE OCCUPATIONAL EXPOSURE LIMITS FROM OTHER COUNTRIES

Country	Eight hour exposure limit (ppm)	Short term limit (ppm)	Ceiling limit (ppm)
Australia	1	2	-
Argentina	-	-	0.3
Belgium	-	-	0.3
Brazil	-	-	1.6
Canada - Alberta	0.75	-	2
Canada - British Columbia	0.3	-	1
Canada - Manitoba	-	-	0.3
Canada - Newfoundland	-	-	0.3
Canada - Nova Scotia	-	-	0.3
Canada - Ontario	-	1	1.5
Canada - Quebec	-	-	2
Denmark	-	-	0.3
Finland	0.3	-	1
France	0.5	1	-
Germany (MAK)	0.3	0.6	1
Italy	-	-	0.3
Japan	0.1	-	0.2
Netherlands	1	1.5	-
Norway	0.5	1.5	-
South Africa	2	2	-
Spain	-	0.3	-
Sweden	0.5	-	1
Switzerland	0.5	1	-
United Kingdom (HSE)	2	2	-
USA (ACGIH)	-	-	0.3

Country	Eight hour exposure limit (ppm)	Short term limit (ppm)	Ceiling limit (ppm)
USA - NIOSH	0.016	0.1	-
USA - OSHA	0.75	2	-

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APPENDIX A: CORRESPONDENCE ADVISING OF A REVIEW OF THE WES

29th November 2008

x

x

Dear x,

NOTICE OF INTENDED CHANGES TO WORKPLACE EXPOSURE STANDARDS

Prior to the publication of the third edition of the New Zealand Workplace Exposure Standards (WES), stakeholder consultation is sought in regard to proposed changes for some substances. This document summarises the objectives behind the proposed changes, refers to the status of the WES under the Hazardous Substances and New Organisms Act 1996 (HSNO), and explains the process for making submissions.

1. The proposed changes are listed in Table 1 overleaf.
2. Some of the WES have been revised in order to bring them more into line with current international best practice, notably, USA and the United Kingdom. As New Zealand does not have prevalent epidemiological studies of the working population, New Zealand's standards reflect international experience. The standards chosen for review are currently inconsistent with international comparators and require moderation.
3. Regardless of the WES, it is expected that employee exposure to hazardous substances will be controlled to a level as far below the WES as practicable by applying the hierarchy of control required by the Health and Safety in Employment Act 1992 (HSE).
4. Some WES set by the Department of Labour are now enforceable controls under HSNO legislation via the Hazardous Substances (Chemicals) Transfer Notice 2006. Therefore, all places of work that use approved hazardous substances must also comply with the HSNO Act as well as the HSE Act. The Department of Labour enforces both laws and their associated regulations in the place of work.
5. The Department now seeks your views on the proposed changes. Submissions close at **5pm on 31st January 2009**. No specific format for submissions is provided, however a letter or email outlining your submission is requested please.

Submissions can be sent to:

Department of Labour
Workplace Services
PO Box 105 146
Auckland
New Zealand
Attention: Philippa Gibson

Appendix A Table 1: Proposed changes to WES

Substance	Current WES	Proposed change
Benzene	5 ppm (8 hour TWA)	0.5 ppm (8 hour TWA) 2.5 ppm (STEL)
Lead (in whole blood)	Suspension level of $\geq 3.2 \mu\text{mol/L}$, or 3 consecutive monthly estimations of $\geq 2.6 \mu\text{mol/L}$	BEI of $1.5 \mu\text{mol/L}$ Suspension level of $2.4 \mu\text{mol/L}$ Return to work level of $1.93 \mu\text{mol/L}$
Formaldehyde	1 ppm (Ceiling)	0.3 ppm (8 hour TWA) 0.6 ppm (STEL)
Methyl bromide	5 ppm (8 hour TWA)	1 ppm (8 hour TWA)
Crystalline silica	0.2 mg/m^3 for respirable quartz 0.1 mg/m^3 for respirable cristobalite	0.1 mg/m^3 for respirable quartz 0.1 mg/m^3 for respirable cristobalite
Synthetic vitreous fibres (<i>currently listed under synthetic mineral fibres</i>)	1 respirable fibre/mL 5 mg/m^3 inspirable dust (8 hour TWA's)	0.2 respirable fibre/mL (for refractory ceramic fibres) 1 respirable fibre/mL (for special purpose glass (micro) fibres) 1 respirable fibre/mL (all other SVF's)
Wood dust	1 mg/m^3 for certain hard wood dusts 5 mg/m^3 for soft wood dusts	1 mg/m^3 for hard wood dusts 1 mg/m^3 for soft wood dusts

19th December 2008

xx

Att:

Dear X,

**NOTICE OF INTENDED CHANGES TO WORKPLACE EXPOSURE
STANDARDS**

Change of date for submissions

Due to overwhelming interest, the closing date for submissions on proposed changes to the New Zealand Workplace Exposure Standards (WES) has been changed to **31st March 2009**.

The proposed changes are listed in Table 1 overleaf.

PLEASE NOTE:

1. The purpose of the consultation process is to provide interested parties with the opportunity to provide evidence to further assist in the decision making process surrounding a particular WES level.
2. These WES have been revised in order to bring them more into line with current international best practice. As New Zealand does not have prevalent epidemiological studies of the working population, New Zealand's standards reflect international experience. The standards chosen for review are currently inconsistent with international comparators and require moderation.
3. In the absence of opposing evidence the department has no option but to accept international best practice as the standard for New Zealand.
4. Some WES set by the Department of Labour are now enforceable controls under HSNO legislation via the Hazardous Substances (Chemicals) Transfer Notice 2006. Therefore, all places of work that use approved hazardous substances must also comply with the HSNO Act as well as the HSE Act. The department enforces both laws and their associated regulations in the place of work.
5. We welcome any input you may have that would inform the decision making process.

Send submissions to:
Department of Labour
Workplace Services
PO Box 105 146
Auckland
New Zealand
Attention: Philippa Gibson

or email:
philippa.gibson@dol.govt.nz

Appendix A Table 2: Proposed changes to WES

Substance	Current WES	Proposed change
Benzene	5 ppm (8 hour TWA)	0.5 ppm (8 hour TWA) 2.5 ppm (STEL)
Lead (in whole blood)	Suspension level of $\geq 3.2 \mu\text{mol/L}$, or 3 consecutive monthly estimations of $\geq 2.6 \mu\text{mol/L}$	BEI of $1.5 \mu\text{mol/L}$ Suspension level of $2.4 \mu\text{mol/L}$ Return to work level of $1.93 \mu\text{mol/L}$
Formaldehyde	1 ppm (Ceiling)	0.3 ppm (8 hour TWA) 0.6 ppm (STEL)
Methyl bromide	5 ppm (8 hour TWA)	1 ppm (8 hour TWA)
Crystalline silica	0.2 mg/m^3 for respirable quartz 0.1 mg/m^3 for respirable cristobalite	0.1 mg/m^3 for respirable quartz 0.1 mg/m^3 for respirable cristobalite
Synthetic vitreous fibres (<i>currently listed under synthetic mineral fibres</i>)	1 respirable fibre/mL 5 mg/m^3 inspirable dust (8 hour TWA's)	0.2 respirable fibre/mL (for refractory ceramic fibres) 1 respirable fibre/mL (for special purpose glass (micro) fibres) 1 respirable fibre/mL (all other SVF's)
Wood dust	1 mg/m^3 for certain hard wood dusts 5 mg/m^3 for soft wood dusts	1 mg/m^3 for hard wood dusts 1 mg/m^3 for soft wood dusts

19th August 2009

x

Dear x

Consultation meeting - proposed changes to Workplace Exposure Standards for Wood dust and formaldehyde

Prior to the publication of the third edition of the New Zealand Workplace Exposure Standards (WES), stakeholder consultation was sought in regard to proposed changes for some substances. Some of the WES are in need of revision in order to bring them more into line with current international best practice, notably, USA and the United Kingdom. As New Zealand does not have prevalent epidemiological studies of the working population, New Zealand's standards reflect international experience. The standards chosen for review are currently inconsistent with international comparators and require moderation.

The proposed changes to the wood dust and formaldehyde standards are given below.

Substance	Current WES	Proposed changes
Formaldehyde	1ppm (ceiling)	0.3 ppm (8 hour TWA) 0.6 ppm (STEL)
Wood dust (8 hour TWA)	1 mg/m ³ for certain hard wood dusts 5 mg/m ³ for soft wood dusts	1 mg/m ³ for certain hard wood dusts 1 mg/m ³ for soft wood dusts

The technical committee has reviewed the feedback received so far for the wood dust and formaldehyde WES and is of the opinion further consultation is required.

This letter is to invite you as an interested party to attend a meeting on Wednesday 25th November 2009 in Wellington to discuss submissions received on the proposed changes to the WES for wood dust and formaldehyde and to produce an agreed timetable for the drafting of the exposure standards. The Department of Labour's perspectives on the proposed changes will also be discussed.

The meeting is to be held in the Sonja Davies Room, Level 3 Greenock House, 39 The Terrace, Wellington from 10am to 1pm. Please note that due to constraints of available venues we are only able to welcome one representative from each organization.

Yours sincerely

Richard Steel
Manager Technical Services

POSITION PAPER: EMAILED 9TH NOVEMBER 2009

The Department of Labour Workplace Exposure Standards

The Department of Labour is the primary agency responsible for setting Workplace Exposure Standards (WES) as part of the administration of the Health and Safety in Employment Act (HSE), 1992.

WES relate to worker exposure to airborne substances in workplaces.

WES are health-based values. In assigning the WES value, defining a level that will achieve freedom from adverse health effects is the major consideration.

Employee exposure to hazardous substances should be controlled to a level as far below the relevant WES as practicable by applying the hierarchy of control required by the HSE Act. Section 10(2)(c) of the Act requires that where significant hazards are minimised, the employer must monitor the employees exposure to the hazard. WES can be used by those involved in occupational health practice as guidelines for evaluating exposure to airborne hazards. Compliance with the designated value does not, however, guarantee protection from discomfort or possible ill-health outcomes for all workers.

As New Zealand does not have prevalent epidemiological studies of the working population, New Zealand's standards reflect international experience. The formaldehyde and soft wood dust WES were chosen for review as they are inconsistent with some international workplace exposure guidelines.

Formaldehyde WES

Currently a WES-Ceiling limit of 1ppm is set for formaldehyde.

The following reasons prompt a review of this WES:

- Human studies on acute inhalation of formaldehyde indicate adverse health effects including: mucous membrane irritation, shortness of breath, upper airway irritation and sore throats, at concentrations below 1ppm (source ACGIH Formaldehyde TLV Chemical Substances Documentation 7th Edition).
- Formaldehyde is classified as a 6.7A substance (known or presumed human carcinogen) under the HSNO Act.
- Monitoring against a Ceiling limit can be problematic in terms of sampling methodology and detection limits.

- An 8-hour WES-TWA of 0.3ppm and a WES-STEL of 0.6ppm are being considered. These levels are intended to minimise the potential for sensory irritation, chiefly of the eye and upper respiratory tract. However, even at these levels, some workers may be responsive to the irritant effects of formaldehyde due to increased sensitivity.

Soft wood dust WES

Currently an 8-hour Time Weighted Average WES of 5mg/m³ is set for soft wood dust.

The following reasons prompt a review of this WES:

- Human studies on the health effects from chronic inhalation of soft and mixed wood dusts indicate adverse health effects including: impaired lung function and lower and upper respiratory symptoms, at concentrations below 5mg/m³ (source ACGIH Formaldehyde TLV Chemical Substances Documentation 7th Edition).
- In industries where a variety of wood types are commonly used or multi-tasking with different wood types occurs, a different WES-TWA for hard and soft wood dusts can be problematic in terms of exposure monitoring.
- An 8-hour WES-TWA of 1mg/m³ is being considered. This level is intended to prevent decreases in pulmonary function.