Occupational Health Clinics for Ontario Workers | Centre de Santé des Travailleurs(ses) de l'Ontario

OCCUPATIONAL HEALTH CLINICS FOR ONTARIO WORKERS

SUBMISSION TO THE MINISTRY OF LABOUR REGARDING:

2013 NOTICE OF PROPOSAL TO ADOPT NEW OR REVISED OCCUPATIONAL EXPOSURE LIMITS OR LISTINGS FOR HAZARDOUS CHEMICAL SUBSTANCES MARCH 2013

SUBMITTED TO: ***ONTARIO MINISTRY OF LABOUR***
PREPARED BY: ***OCCUPATIONAL HEALTH CLINICS FOR ONTARIO WORKERS***

1. OEL's, while perhaps necessary as a bare minimum standard, are not the most effective method for reducing workplace exposures. OEL's should be secondary to an **explicit regulatory requirement to identify and control workplace hazards**, so that even if compliance with an OEL is demonstrated, action can be taken to protect workers experiencing symptoms/diseases at exposures below the OEL.

2. Without any regulatory requirement for workplaces to **assess exposures**, nor, any requirement to use **statistically valid sampling strategies** to assess compliance, OEL's may not even be effective in preventing harmful exposure, even in non– compliance situations (i.e. excessive exposures undetected because sampling either wasn't done or not done using an appropriate sampling strategy).

3. Ontario should follow BC's lead (similar as in the EU) in requiring employers using a **carcinogen** (or **sensitizer** or **reproductive hazard**) to demonstrate that there are no practicable substitutes for using the carcinogen in the work process. This would be consistent with the current Ministry of the Environment initiative for toxic use reduction. It would also position Ontario for the closer trade relations with the European Union which will likely result from the upcoming free trade negotiations.

4. We would also repeat our previous endorsements of the **silica** and **wood dust** proposals. Based on our work at OHCOW we perceive a significant need to reduce the OELs for **ozone, manganese, PNOC's, metalworking fluids** and **diesel exhaust** (we have raised this issue in previous submissions). There is also a need to update the Code for Medical Surveillance in the lead designation substance regulation to reduce the **blood lead** biological exposure indices used.

OHCOW Background and Exposure Assessment Experience:

The Occupational Health Clinics for Ontario Workers Inc. is a team of health professionals committed to promoting the highest degree of physical, mental and social well being for workers and their communities. At six clinics in Ontario a team of nurses, hygienists, ergonomists and physicians see patients and identify work-related illness and injuries, promote awareness of health and safety issues, and develop prevention strategies. First established in 1989, the clinics have seen thousands of individual patients and visited hundreds of workplaces helping to identify unhealthy and unsafe conditions, and providing advice to workplace parties on the prevention of occupational diseases.

With respect to occupational exposure limits, OHCOW deals directly with Joint Health and Safety Committees (JHSC's), unions, employers, individual workers and others, helping them to interpret exposure assessments, developing assessment strategies, directly assessing exposures, dealing with issues underlying the requests for assessments (e.g. worker symptoms and health conditions), questions of toxicology and assessment elimination, substitution and/or control measures. OHCOW has a number of trained occupational hygienists throughout the province servicing client workplaces.

OHCOW also has extensive clinical experience with workers who have suffered illness or injury due to exposures in the workplace and have seen the role the OEL's play in prevention (or the lack of prevention when illnesses occur even when exposures comply with the OEL).

Concerns Regarding the ACGIH TLV Committee:

Serious allegations have been leveled in the scientific literature in the past concerning the integrity of the ACGIH TLV's particularly with the role that industry plays in influencing the Committee[1-5]. Reviews have shown that often the level set for the TLV's is more closely related to what industry sees as practically achievable levels, as opposed to health based levels. The ACGIH TLV Committee responded to these criticisms by tightening up its process and documentation of the TLV's. However, a different challenge has been launched against the TLV's in the last few years which also threaten to effect the manner in which they are set. A number of lawsuits were launched against the TLV's from both industry and industrial disease victims. These legal challenges have had a "chilling" effect on the organization and seem to have introduced hesitancy in reacting to situations where there is limited evidence. Recent history has shown that the ACGIH seems to be retreating from certain standards for which they now feel the evidence is not sufficient. This retreat appears to be associated with the recent legal actions against the ACGIH and therefore may be more out of concern to avoid liability than ensuring workers exposed are protected. The removal of the TLV for particulates not otherwise classified (PNOC) is an example. In this effort to become more scientifically exact, protection for exposed workers is lapsing for the sake of scientific precision and avoidance of lawsuits.

Against this trend we would suggest that the precautionary principle (as it was discussed in the context of workplace health and safety in the Campbell Commission http://www.health.gov.on.ca/english/public/pub/ministry_reports/campbell06/campbell06.html) needs to be included in Regulation 833 to address situations where the OEL has been eliminated, or no OEL has been established or the OEL is insufficiently protective. The current provisions in Regulation 833 only provide remedies in such situations if a worker can get medical corroboration for their health concerns, however, a large majority of OEL's are based on preventing irritation which would not be clinically objectively verifiable, thus there is need to address worker health effects which are not clinically measureable.

A Lack of a Legal Requirement to Measure Exposures:

Setting lower OEL's will not necessarily lead to reductions in exposure in Ontario workplaces. (Removing some OEL's will obviously not have such an effect either and may even increase exposures – a very counter-prevention trend!) In order for an OEL to effectively lower workplace exposures, measurements must take place in workplaces particularly where exposures may exceed the new OEL. The proposed changes to the regulation do not require employers to take measurements, so naturally if no measurements are taken, no over-exposures will be detected and there will effectively be no regulatory inducement to reduce or eliminate exposures.

There is thus a need for a regulatory requirement to perform sampling for the purpose of exposure assessments if the changes in OEL's are to impact Ontario workplaces. Without such a legal requirement, employers fearing being found out of compliance may merely decide not to measure at all.

A Lack of a Legal Requirement to Employ Unbiased Sampling Strategies:

Even if measurements are taken, the conditions under which they are taken and the number of measurements taken can be manipulated as to minimize the chances of detecting an over-exposure. This concern is often brought to the attention of OHCOW staff by workers asking for reviews of occupational hygiene reports (e.g. "they should have sampled when …"). In fact it has been shown[6] that mathematically modeling exposures[7] is more accurate than a sampling campaign that covers three or fewer workdays (most sampling campaigns cover only a single day). The Joint Steering Committee on Hazardous Substance Regulations (JSC, 1987-1995) recognized this situation and brought forward a draft regulation on exposure assessment strategies which would require employers to assess exposure using prescribed methods and sampling strategies which would ensure objective assessments. Stephen Rappaport has also written extensively[8,9] on statistically valid sampling strategies and was used as a consultant for the JSC's draft regulation on sampling strategy. The AIHA Exposure Assessment Strategy Committee has produced a manual[10] on procedures and strategies for managing exposure assessments. This manual has become the standard for properly designing exposure assessments strategies. For regulatory purposes, a regulation could simply refer to this monograph and require that sampling strategies would be devised following the procedures outlined in this manual. This would ensure that appropriate exposure assessment strategies are used addressing the common criticisms of biased sampling strategies.

Concerns Regarding the Effectiveness of the OEL as a Means for Improving Workplace Conditions:

If these changes in the OEL's were accompanied with legal requirements to perform exposure assessments and be required to follow recognized sampling strategies, would workplace exposures be reduced? This question has been addressed by the author Eileen Senn[11] who reviewed the US OSHA experience with measurements taken by OSHA representatives in response to workplace exposure complaints. Her findings based on the OSHA database of workplace measurements showed that over 90% of measurements taken in response to complaints were in compliance. What this means is that quantitative exposure assessment essentially had the effect of reinforcing the status quo (i.e. no regulatory onus to reduce

exposures) in situations where workers had lodged complaints regarding exposures. While delivering our services, OHCOW has encountered the general frustration workers have with respect to occupational hygiene exposure assessments. Invariably, exposures are in compliance with current standards, in spite of significant symptoms and concerns experienced by workers. Note as well that most sampling strategies do not follow accepted guidelines as laid out in the AIHA exposure assessment manual. These assessments/reports then become an extra obstacle in the struggle to alleviate symptoms and reduce/eliminate exposures.

Ms. Senn also investigated the effect of updating the US OEL's from 1968 to 1989 would have on the percentage of compliance. Her findings were that such a drastic updating (almost 30 years) would generally only lower the compliance rate by less than 10% (from above 90% compliance to above 80% compliance). Thus the updating of the OEL's would generally have little impact on the level of exposure experienced by most workers. There were some exceptions however, for instance the proposed lowering of the silica OEL's in Ontario would significantly impact those workers working with these chemicals since exposures are often at or over the current exposure limit. But outside a few specific exceptions, it is generally expected that if employers would be obliged to measure exposures and if they used appropriate sampling strategies, the number of workplaces found out of compliance would not change significantly.

Limitations in OEL's in Preventing Occupational Disease:

Even though most workplaces are in compliance with current OEL's and would be expected to be in compliance with the proposed changes (with a few notable exceptions), this does not mean there are little or no hazards due to the exposures among Ontario workers. First of all, the ACGIH in its preamble to the TLV specifically state that not all workers will be protected by complying with these OEL's. In fact if one follows the history of OEL's one will notice a gradual decline in most OEL's over the years as more evidence of workers experience symptoms and diseases are established. What is to say that an exposure which may be legal now, may in the future be considered to be associated with an occupational disease once the evidence (i.e. affected workers) has been collected and assessed. This has been the pattern in the past and there is little reason to suspect it will not continue. This is one of the reasons for the ALARA (as low as reasonably achievable) principle or the precautionary principle, which both suggest that exposures be kept as low as reasonably possible in light of the scientific uncertainty associated with the evidence (or lack of evidence) regarding the association of exposure with disease. Rather than a chemical being assumed to be non-toxic until proven otherwise (thus the absence of evidence supporting non-toxicity), we would adhere to the assumption of a chemical's toxicity until valid evidence is produced to the contrary.

The MOL has instituted a policy which recognizes that just because exposure assessments demonstrate compliance is no reason to ignore workers symptoms and health problems associated with such exposures. The fact that there are relatively few reported investigations assessing worker health in relation to exposures in consideration of the number of workers actually exposed. The standard of evidence for the basis of many OEL's is extremely poor by general scientific standards; some merely suggesting limits by analogy or based on animal toxicity experiments despite that fact that thousands of workers are exposed daily to such chemicals.

For other OEL's where there is sufficient human evidence, a conscious decision has been made by the committee to tolerate a specified amount of occupational disease in setting the limit. An example of this calculated risk is the noise TLV, where the documentation of the TLV

recognizes that up to 10% of workers exposed to 85 dBA in a working life will suffer noise induced hearing loss. Furthermore, it is well known that workers exposed to sensitizers such as isocyanates are not adequately protected by compliance with the OEL (a certain percentage of exposed workers will go on to develop asthma in spite of maintaining exposures below the OEL). Carcinogens often do not have a threshold and thus OEL's are set at an "acceptable" rate of occupational disease (usually 1 worker in 1000 exposed, despite the environmental standard of risk being 1 citizen in 100,000 to 1,000,000). Taking all these limitations into consideration, it is very clear that compliance with OEL's is in no way a guarantee that no significant health effects may occur among workers exposed!

New Paradigms in Exposure Criteria:

The dose-response relationship is more of a continuum than a straight line with a sudden discontinuity at the OEL. The heat stress OEL is graduated response as the WBGT rises. New paradigms in exposure assessment criteria have surpassed the single digit representation of the dose-response relationship which the OEL represents. In indoor air quality investigations, sampling strategies focus on source identification and measurements are interpreted in terms of ranges instead of a single digit threshold. For example, carbon dioxide is used as a surrogate for ventilation performance and is interpreted in terms of ranges[12]:

< 600 ppm	no problem with the quantity of outdoor air supply
600-800 ppm	possible problem particularly if there are other parameters indicating possible problems (select parameter best suited to intervention)
800-1000 ppm	probable problem with inadequate quantity of outdoor air supply
1000 ppm	definite problem with inadequate quantity of outdoor air supply

Similar graduated ranges have been established for volatile organic compounds (VOC's)[13], although the main goal of measuring VOC's is more to find the source and eliminate or control it to prevent exposure in the first place. Thus in the overall scheme of prevention, the single digit threshold concept is a gross reduction of a much more complex dose-response relationship and as such the graduated exposure criteria, such as for VOC's, are a more realistic approach.

Sensitizers, Carcinogens and Reproductive Hazards in the Workplace:

Workers' health in Ontario would benefit if exposures to sensitizers and carcinogens was avoided though methods including substitution, engineering controls, isolation, local ventilation and protective equipment to prevent exposure by any route. The ideal place to prevent exposures is at the source[19]. Any workplace where sensitizers or carcinogens are used should be required to demonstrate, on a regular basis, that it is actively involved in an ongoing process to identify alternative non-toxic chemicals and/or processes, so that these materials are no longer used in the workplace. Until such time that a substitute chemical and/or process replaces the sensitizer or carcinogen, the workplace must demonstrate, using a valid occupational hygiene sampling strategy[10], that exposures are "as low as possible" and that there is a continuing process of

improvement in engineering and occupational hygiene that will result in a further reduction in exposure and that workers are not experiencing symptoms of exposure or are having to leave due to health effects caused by the product.

Innovative Qualitative Exposure Techniques to Address Small & Medium Sized Business Enterprises:

It has also been recognized that most small or medium sized enterprises (SME's) do not have the resources to conduct the amount of quantitative sampling required by an appropriate quantitative exposure assessment strategy consistent with the procedures outlined in the AIHA exposure assessment manual (not to mention the concern that those resources would be more productively allocated to control once workers have identified an exposure of concern). In response, the AIHA manual and various European organizations have developed qualitative exposure techniques to help SME identify the needs for exposure control without using significant resources to measure exposures. One of the most recognized techniques is the control banding method espoused by the British HSE (http://www.coshh-essentials.org.uk/). Other schemes have also been developed in the Netherlands, Germany, Italy and Spain. All these methods attempt to "automate" the decision logic exposure assessors would used to categorize exposures and recommend controls. The Ontario Ministry of Labour had a preliminary meeting with stakeholders a few years ago (1999-2000) introducing the idea, however, nothing appears to have materialized from these efforts.

Other countries, Italy and Brazil in particular, have established mandatory risk mapping exercises, where workers are asked to identify exposure concerns in a diagram format and these become the basis of an exposure control program[15,16]. Also, Malchaire[18] in Belgium has developed an approach to risk assessment and control which recognizes four level of assessment and problem solving (screening (shop floor), observation (JH&SC), analysis (OH&S professional) and expert) which goes by the acronym of SOBANE. The screening and observation risk assessment and problem solving tools are ideal for the SME and the analysis protocols ensure that the work done by hygienists co-ordinates with the preliminary risk assessments done on the shop floor and JH&SC levels.

OEL Update Process (substances not acted upon):

Having participated in a number of rounds of update consultations, we have some concerns about the process, particularly with substances not adopted and seem to have "fallen through the cracks". The appears to be no communication process that allows those who submit comments to get any feed-back from the Ministry other than the regulation stipulating which OEL's have been adopted. No official communications dealt with the substances which were not adopted. On contacting the branch of the MOL which deals with the OEL updates, one is generally told that matter is still "under consideration". One particular substance which has been "under consideration" for more than five years, is silica.

Silica: In 2004 the MOL proposed lowering the silica OEL (or TWAEL as it is called in the designated substance reg for silica) to 0.05 mg/m^3, however it was not adopted. In 2006 (after the ACGIH lowered its TLV again), the MOL again listed silica in its annual OEL update

but this time proposing to lower the TWAEL to 0.025 mg/m^3, but again, it has not been adopted and is said to be "under consideration". The ACGIH has provided extensive documentation for their TLV of 0.025 mg/m^3. As silicosis is still a disease that affects Ontario workers (http://www.cbc.ca/sunday/2009/02/022209_1.html) despite the fact that the knowledge and technology have long been available to prevent silicosis, it speaks to the urgency needed to implement this change. It is also noted that in the current silica designated substance regulation that the ALARA is incorporated:

"Every employer shall take all necessary measures and procedures by means of engineering controls, work practices and hygiene practices and facilities to ensure that the time-weighted average exposure of a worker to silica is reduced to the lowest practical level and in any event shall not exceed" (Section 4(1))

Furthermore as a carcinogen, we would also invoke the comments made above pertaining to the need to substitute workplace carcinogens out of the workplace where practicable.

We realize that quartz is ubiquitous in the Ontario environment (particularly in the Canadian Shield) however, workers are still vulnerable to silicosis if exposed. We do not agree that simply because our environment has more quartz in it than other jurisdictions that therefore Ontario workers should endure inferior protections. Other jurisdictions have other exposures more frequently than northern Ontario (e.g. heat stress and ozone). Thus while Ontario businesses may have to devote funds to control a particular hazard of environmental origin more often than similar enterprises in other jurisdictions, other environmental hazards may be less prevalent in the natural Ontario environment. Asbestos is another natural hazard which needs to be regulated despite the fact that it occurs naturally in the environment.

We can see no environmental justification to deprive Ontario workers from adequate protection against silicosis.

As noted earlier and as reported by the CBC (http://www.cbc.ca/sunday/2009/02/022209_1.html) silicosis is still an issue in Ontario and it behooves the Ministry of Labour to bring their "considerations" to a conclusion after 5 years of "consideration".

Blood Lead: Another issue that appears to have "dropped through the cracks" is the blood lead criteria for review and removal found in the Code for Medical Surveillance associated with the Lead designated substance regulation. When the OEL for lead was reduced, no changes were made to the Code for Medical Surveillance, thus it would appear that Ontario's blood lead removal concentration is 70 µg/dL (or 0.70 mg/L or 3.4 µmol/L). The ACGIH Biological Exposure Index (BEI) for lead is 30 µg/dL with a caveat that women of child bearing potential should not exceed 10 µg/dL. The Ontario Designated Substance Lead Regulation Code for Medical Surveillance specifies the following responses to blood lead levels:

Blood Lead		Action
40 µg/dL	(1.95 µmol/L)	pregnant workers to be removed
50 µg/dL	(2.4 µmol/L)	return to exposure after medical removal
60 µg/dL	(2.9 µmol/L)	exposure review (alert level)
70 µg/dL	(3.4 µmol/L)	medical removal

Philip Landrigan was asked by the Ministry of Labour to compile evidence for a list of health effects and their corresponding threshold blood-lead levels for the Joint Steering Committee's Biomedical Task Force. He came up with the following list:

Toxic Effect	Lowest Blood-Lead Levels Associated with Toxic Effect	
inhibition of heme biosynthesis (ALA-d inhibition)	10 to 20 μg/dL	(0.48-0.97 μmol/L)
renal impairment (inhibition of vitamin D hydroxylation)	25 μg/dL	(1.21 μmol/L)
hypertension	10 to 20 μg/dL	(0.48-0.97 μmol/L)
peripheral neuropathy	30 to 40 μg/dL	(1.45-1.93 μmol/L)
central neuropathy (sub-clinical encephalopathy)	40 to 70 μg/dL	(1.93-3.38 μmol/L)
male reproductive dysfunction	50 to 60 μg/dL	(2.41-2.89 μmol/L)
fetal neurological impairment	10 to 20 μg/dL	(0.48-1.21 μmol/L)

* this table was copied from "Medical Surveillance of Workers Exposed to Lead: A Report to the Biomedical Surveillance Task Force, Ontario Ministry of Labour", by P.J. Landrigan (January 1992).

Based on this review, Landrigan recommended that the blood-lead removal concentration be 20 μg/dL (1.0 μmol/L) and the re-entry concentration be 10 μg/dL (0.5μmol/L). Therefore, for a lead assessment where blood-lead concentrations have been determined, if the levels exceed 10 μg/dL (0.5 μmol/L), it can be argued that the health of the worker could be affected. It should be noted that the Ministry of Labour received this recommendation in 1992 and no changes have been made to the Code for Medical Surveillance.

Substances Under Review

Nitrogen Dioxide

The ACGIH (2012) has lowered the TLV TWA for nitrogen dioxide from 3 ppm to 0.2 ppm and eliminated the previous STEL of 5 ppm. The basis of the TLV is lower respiratory tract irritation and is intended to be protective for workers with asthma.

In other jurisdictions, the Dutch OEL (Netherlands, 2004) has been a TWA of 0.2 ppm with a short tem exposure limit of 0.5 ppm since 2004 and the 2012 recommendation of the European Scientific Committee on Occupational Exposure Limits (SCOEL, 2012) is for a TWA of 0.2 ppm with a short term exposure limit of 1 ppm (SCOEL, 2012); the current NIOSH REL nitrogen dioxide is a STEL of 1 ppm (NIOSH, 2013).

In addition to the ACGIH documentation (2012), three other recent reviews are available that have been prepared for the purpose of setting exposure standards (Netherlands, 2004; SCOEL,

2012; US EPA 2008). Of these three, only the US EPA has considered the effect of nitrogen dioxide on asthmatics.

The Dutch (Netherlands, 2004) have based their short term exposure level on the human NOAEL of 0.5 ppm and extrapolated the 8-hour TWA of 0.2 ppm from the NOAEL derived from long-term animal data, using an overall uncertainty factor of 1. The SCOEL (2012) has relied on recent inhalation studies in rats to determine the NOAEC of 2.15 ppm and then has used an uncertainty factor of 10 to derive the OEL of 0.2 ppm as a TWA; the STEL of 1 ppm is based on studies of human volunteers (particularly a study of health volunteers by Frampton et al, 2002). Neither the Dutch nor the SCOEL recommendations take into consideration the effect of nitrogen dioxide on asthmatics; the US EPA's Risk and Exposure Assessment (2008) found that the majority of asthmatics may experience nitrogen dioxide-related airway hyper-responsiveness following short-term exposures between 0.1 ppm and 0.3 ppm nitrogen dioxide.

From a meta-analysis of 19 controlled human exposure studies involving mild asthmatics, the US EPA (2008) report that the LOEL for nitrogen dioxide is of 0.1 ppm. As more severely affected asthmatics may be more susceptible than mild asthmatics to the effects of NO2 exposure, they concluded that lower end of the range of potential alternative 1-h daily maximum standards is 0.05 ppm. In addition, small but significant increases in nonspecific airway responsiveness were observed in the range of 0.2 to 0.3 ppm nitrogen dioxide for 30-minute exposures and at 0.1 ppm nitrogen dioxide for 60-minute exposures in asthmatics.

The ACGIH TLV TWA of 0.2 ppm is the same value as the Dutch and SCOEL TWA OELs except that neither of those two is intended to be protective for workers with asthma as the ACGIH has claimed to be. Taking the asthmatics into consideration, the US EPA report found the nitrogen dioxide LOEL for airway hyper-responsiveness is 0.1 ppm and that 0.05 ppm is needed to be protective for severely affected asthmatics.

Because asthmatic workers are a sensitive population, they need increased protection and Ontario should adopt a health-based nitrogen dioxide OEL that meets their needs. While a vast improvement over the previous TLV, and now in line with newer European standards, in light of the US EPA findings, the ACGIH TLV appears to have fallen short of its stated goal with regard to workers with asthma. A TWA of 0.05 ppm would be protective of all asthmatics; however, a STEL or CEILING approach would also be needed for peak exposures.

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Ethyl Formate

The ACGIH (2102) has changed its existing TLV for ethyl formate from a TWA to a STEL, keeping the same value of 100 ppm. The basis of the STEL is to minimize upper respiratory tract irritation as well as protect against central nervous system effects which occur at higher levels. In setting the TWA earlier, the ACGIH did not feel that there were sufficient data to recommend a STEL; in the absence of a STEL, the excursion limit would have been 300 ppm.

The current ACGIH STEL and the TWA before it is based on human health data. The STEL and the TWA are based on data from a 1931 German language book by Flury and Zernik (1931), who found that ethyl formate was an irritant at 330 ppm. In the current documentation for the STEL, the ACGIH also cites van Thriel et al. (2006) who list the irritant concentration as 321 ppm - this value based on the work of Ruth (1986), who reported the irritant threshold as 990 mg/m3 (327 ppm). The irritant threshold for ethyl formate in humans from these sources is in the range 321 to 330 ppm.

Jurisdictions such as Finland and the United Kingdom have OELs that specify both a TWA and STEL. The Norwegian OEL is a TWA of 50 ppm without a STEL. By specifying a STEL, it is possible to limit the magnitude of short-term peak exposures to a lower level than is possible for a TWA with excursion limits and therefore is better suited to controlling an acute irritant such as ethyl formate. Using the STEL approach, has the effect of reducing the TWA exposure to some extent.

Finland, the United Kingdom and Norway have industrial economies similar to Ontario. The STEL approach is already in use in Finland and the UK and Norway has an OEL TWA of 50 ppm that is half the ACGIH STEL. The ACGIH has set the STEL a factor of only 3 below the LOAEL in humans; however, Norway has used a factor of 6 in setting its TWA. It should be noted that a STEL of 100 ppm would still permit conditions to exist in which a worker could

theoretically be exposed at or above the irritation threshold (i.e. 321 to 330 ppm) for up to almost 5 out of the 15 minutes.

Since the use of the STEL approach would provide greater control of peak exposures as well as reduce average exposures, the use of the STEL should be adopted in Ontario to prevent cases of upper respiratory tract irritation. Based on Norway's example, it should be possible to further decrease the magnitude of the STEL and more nearly eliminate the possibility of respiratory irritation in exposed workers.

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Carbonyl Sulfide

The ACGIH (2012) has a new TLV for carbonyl sulfide of 5 ppm based on central nervous system impairment. No other OEL was found for this substance; however, reportedly there is a corporate (DuPont) "Adverse Exposure Level" of 2 ppm (ATSDR, 2007), one US state's interim

"Effects Screening Level" for carbonyl sulfide is 1.1 ppb (TCEQ, 2008) and the US EPA has a derived an interim AEGL-2 of 23 ppm.

The US EPA derived its "Acute Exposure Guideline Level-2" (AEGL-2) of 23 ppm for irreversible or other serious, long-lasting adverse health effects" in the general population by extrapolation from animal data. However, they found insufficient data to derive an AEGL-1 which applies to non-disabling, transient and reversible effects of "notable discomfort, irritation, or certain asymptomatic, non-sensory effects" in the general population.

The ACGIH documentation includes only one human toxicology reference: a case report of a worker accidentally exposed to a high level of a mixture of reduced sulfur gases including carbonyl sulfide. The TWA is based on the NOEL for central nervous system (CNS) effects in rats. The key animal study cited is by Roloff (1985) who reported that rats receiving eleven six-hour whole-body exposures of 51 ppm over a two-week period did not show dose-dependent signs of CNS dysfunction. The CNS effects seen in animals exposed to higher doses included ataxia, head- tilting, circling, tremors and convulsions. Additional signs of CNS dysfunction, from Roloff (1985) and noted in Bartholomaeus and Haritos (2005), were pivoting, prostrate and arched back postures, loss of muscle control and bulging and dilated eyes.

Rats exposed to sufficient doses of carbonyl sulfide develop neurodegenerative changes in numerous areas of the brain; the nature of these changes and mechanisms behind them are the subject of recent research by a group of researchers in the US. (Herr et al, 2007; Sills et al, 2004 and 2005; Morrison et al, 2009).

Kamstrup and Hugod (1979) exposed 18 rabbits to 54 ppm ± 13 ppm carbonyl sulfide by inhalation continuously for seven weeks. While 13 were clinically unaffected, 3 died after five days and 2 developed symptoms of CNS toxicity; the differences were interpreted as evidence of individual variability in sensitivity (at this dose level).

The ACGIH documentation did not address reproductive toxicity of carbonyl sulfide; however, Bartholomaeus and Haritos (2005) and the US EPA (2008) report reproductive toxicity NOELs in rats of 60 ppm in males and 182 ppm in females based on data from a 1987 study by Reyna and Ribelin. Based on the same reproductive toxicity data, the Texas Commission on Environmental Quality (2008) has applied a cumulative of uncertainty factor of 3000 to these data (Reyna and Ribelin, 1987) in deriving a long-term (annual average) Effects Screening Level (ESL) for (ambient) carbonyl sulfide of 1.1 ppb.

Carbonyl sulfide is produced in numerous industrial activities as well as naturally by living organisms. Carbonyl sulfide, carbon disulfide and hydrogen sulfide are related biologically: carbonyl sulfide is a metabolite of carbon disulfide and hydrogen sulfide is a metabolite of carbonyl sulfide (Sills et al, 2005). Carbonyl sulfide was demonstrated to be rapidly converted by isolated rat hepatocytes to hydrogen sulfide and this is the major metabolic pathway for carbonyl sulfide (Chengelis and Neal, 1979). Both carbonyl sulfide and hydrogen sulfide are neurotoxic and carbonyl sulfide neurotoxicity and while knowledge of the mechanism of carbonyl sulfide toxicity is incomplete, it considered to be due (at least in part) to the release of hydrogen sulfide (Chengelis and Neal, 1979; Sills et al, 2005; Bartholomaeus and Haritos, 2005; US EPA, 2008).

The mechanisms of carbonyl sulfide neurotoxicity are not completely known. Other jurisdictions have not developed an OEL; the US EPA has developed an interim health-based exposure level for the general population based on animal data but, due to insufficient data, it is only for irreversible effects: this level is 23 ppm, or a factor of approximately 5 times greater than the ACGIH TLV. An experiment involving rabbits has shown that there can be significant individual variability in sensitivity to the neurotoxic effects of carbonyl sulfide at lower doses. Reproductive toxicity in male rats has an NOEL that is in the same range as the rat NOEL for neurotoxicity.

Ontario should adopt a lower OEL to help protect exposed workers against possible neurological and reproductive toxicity. As carbonyl sulfide is primarily and rapidly metabolized to hydrogen sulfide and its neurotoxicity is thought to be at least partially attributable hydrogen sulfide, it would be prudent to apply the hydrogen sulfide TLV which is a TWA of 1 ppm and a STEL of 5 ppm. While the NIOSH REL for hydrogen sulfide is now in need of revision, the ceiling approach for an OEL that NIOSH adopted could also be considered as an alternative (NIOSH, 2013) to the STEL.

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O-phthalodinitrile (1 ,2-Benzenedicarbonitrile)

The ACGIH TLV (2012) for o-phthalodinitrile is TWA 1 mg/m3 (inhalable fraction and vapour), based on central nervous system toxicity (convulsions) and body weight effects (decreased). This is the first time the ACGIH has made a TLV for o-phthalodinitrile; however, Japan (2013) has had an OEL of 0.01 mg/m3 since 2010.

The ACGIH documentation for o-phthalodinitrile (2012) does not reference literature published since the publication of a 2001 review by the United Nations Environment Programme (UNEP, 2001). The UNEP review has a more comprehensive evaluation of animal studies; however, both cover the same occupational exposure literature which is in German and dates from the 1960's and 1970's, a time when o-phthalodinitrile was produced at one company in Germany. According to the literature reviews of these two documents, none of these four or five studies had exposure information and only one reported toxic effects in workers – headaches, dizziness,

nausea and vomiting, vertigo and seizures resulting from dermal and inhalation exposure – while the other studies reported no increase in morbidity, mortality or frequency of aberrant chromosomal metaphases. O-phthalodinitrile is absorbed through the skin (HSDB, 2013), and although this route has been documented by the ACGIH (2012), it has not been factored into the TLV.

O-phthalodinitrile is an acute neurotoxin in animal studies, with effects seen at relatively low doses; it is also toxic to the testes, female reproduction, the liver and kidneys in the species in which it has been tested.

The ACGIH (2012) and UNEP (2001) documents differ somewhat in the determination of the value of the NOAELs for repeat dose toxicity in animals; estimates have been based on literature in the German and Japanese languages. The key document for both appears to be a study by the Japanese Ministry of Health, Labour and Welfare (1996). The ACGIH has estimated the health risk to workers by extrapolating from an NOAEL of 1 mg/kg in rats which they have converted to an NOAEL of 7 mg/m3 for workers. The conversion is based on 8 hours inhalation exposure and adjusting for the differences in rat and human body weight and respiration. At 1 mg/m3, the TLV TWA is a factor of 7 lower than the value extrapolated directly from the rat NOAEL value. To determine an accurate interspecies and intraspecies uncertainty factor, information on the toxicokinetics and metabolism is also required. The Japanese OEL for o-phthalodinitrile is 0.01 mg/m3 which is 100 times lower than the ACGIH TLV. This difference might be attributable to the weight given to the seriousness of the health effects at low dose levels and the lack of the toxicokinetic and metabolic data; as the key research has been reported in the Japanese language, this may also have been a factor.

Japan has a modern industrial economy similar to Ontario's.

The Japanese OEL of 0.01 mg/m3 appears to be health-based and is more likely to provide adequate prevention for workers than the ACGIH TLV which is 100 times higher. Therefore we recommend that Ontario adopt the Japanese OEL of 0.01 mg/m3 rather than the ACGIH TLV.

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Piperazine

With the exception of Norway, Sweden and the Netherlands, most European countries do have a piperazine OEL of 0.1 mg/m3 (0.03 ppm), which is also the new ACGIH TLV TWA (2012). Japan does not have an OEL for piperazine; they have designated it as a sensitizer (Japan, 2013). There is no German MAK for piperazine on the basis of insufficient information - piperazine is a skin and airways sensitizer for which threshold concentrations do not apply (Germany, 2012). The ACGIH (2012) also recognizes that piperazine is a dermal and respiratory sensitizer and the basis of the TLV is respiratory sensitization and asthma.

The ACGIH (2102) has identified a relatively small number of uses for piperazine: manufacture of fibers, pharmaceuticals, and insecticides. It is also used in a wide variety of other applications and industries including: corrosion inhibitors, rubber additives, metalworking fluids, scrubber in gas-washer formulations, hardener for glue prepolymers, surface-active agent, chemical intermediate, additives in epoxy, urethane and polyamide resins, flame retardants, oil refining chemicals, sizing agents, disinfectants, optical brighteners, photographic colourants, electroless plating, ion exchange resins and microcapsules (Germany, 2012; HSDB, 2013; Huntsman, 2013; Delamine, 2013).

While the lowered TWA may lessen the level of risk for sensitization, it is not currently possible to define an OEL for an allergic reaction to protect already sensitized individuals (see discussion in this document on sensitizers). In addition, exposure to low levels that are insufficient to cause an allergic attack may worsen airway inflammation in already sensitized individuals (Arbete och Hälsa, 2003). With piperazine there is the further complication of cross-reaction with other substances with chemically similar structures such as ethylenediamine (HSDB, 2013).

The ACGIH documentation (2012) has designated piperazine as "Not Classifiable as a Human Carcinogen" on the basis of negative but inadequate animal studies that used piperazine alone. The documentation does not discuss the synergistic nitrosation of piperazine in the presence of nitrite (presumably in the stomach) to a carcinogenic nitrosamine compound, N-mononitrosopiperazine. A dose dependent increase in lung adenomas has been demonstrated in animal studies in which piperazine and nitrites were administered concurrently (Mirvish, 1975; Greenblatt and Mirvish, 1973). In a study involving human volunteers who inhaled piperazine at concentration of 0.3 mg/m3 and eating a nitrate-rich diet, Bellander et al (1988) found that 5% of the absorbed piperazine dose was converted to N-mononitrosopiperazine.

The lowered ACGIH TLV TWA of 0.03 ppm should be adopted in Ontario as it may lower the level of risk for sensitization; however, exposure may occur in a wide variety of workplaces, and, as it is a defined sensitizer and a potential carcinogen, use of piperazine should be discouraged

and substitution encouraged. The aim of regulation should be to avoid creating new cases for already defined allergens (Arbete och Hälsa, 2003).

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Nonane

With the exception of the Netherlands and Norway, which have a nonane OEL of TWA 100 ppm, Sweden (2011) which has an OEL of 150 ppm and Germany which has no OEL, most other jurisdictions in Europe, the US and Japan have a nonane OEL of TWA 200 ppm, which is the same as the ACGIH TLV TWA (since 1976). Additionally, Sweden and Finland have short term exposure limits of 200 ppm (since 1989) and 250 ppm, respectively.

The ACGIH (2012) has not changed the TLV this time; only the documentation has been updated. The basis of the TLV is central nervous system impairment. The health effects data are entirely extrapolated from animal experiments; no human exposure, metabolic or toxicokinetic data were used.

The key paper cited by the ACGIH (2012) is Carpenter et al. (1978), who report that in the animals given the LOAEL dose (1600 ppm), fine tremors, mild coordination loss as well as salivation occurred during the first 4 days; and, irritation of the eyes and salivation were seen during subsequent exposure periods. At the end of the exposure period - 13 weeks of exposure for 6 hours a day, 5 days a week - the mean body weights of the group of rats exposed to the LOAEL dose were statistically significantly lower than those of the controls and the amount of weight loss was approximately 10%.

STATOIL (1987) reports that in rats exposed to nonane levels similar to the LOAEL identified by Carpenter et al (1978), but more intensely per day and for a shorter term (12 hours per day for 14 days consecutively), they found reduced gross motor activity and aggression, reduced vigilance, and impaired motor performance and learning ability.

In their study, Carpenter et al. (1978) determined an NOAEL for nonane in rats is 590 ppm. In extrapolating to humans, the ACGIH has used a factor of 3 for adjustment for interspecies differences. This level was chosen because of the higher alveolar ventilation rates and cardiac outputs of rats and because the effects at the LOAEL in the same study (Carpenter et al.,1978) were considered to be "mild".

In addition to the species differences in alveolar ventilation rates and cardiac outputs as are mentioned by the ACGIH (2102),additional physiological parameters including metabolism and toxicokinetics are used to build predictive models. The Dutch documentation of their OEL (Netherlands, 2005) does include a discussion on rat and human biotransformation and kinetics and also has a more expansive discussion on health effects and toxic mechanisms in animals.

Based on the data from a more substantial health hazard assessment, the Dutch (Netherlands, 2005) have extrapolated from the NOAEL in rats reported by Carpenter et al. (1978) using an intra- and interspecies uncertainty factor of 9, and, rounding according to the preferred value approach, have produced a health based OEL for nonane of 100 ppm. The ACGIH TLV TWA of 200 ppm is used in Sweden as a short term limit.

The Netherlands, Norway and Sweden have modern industrial economies similar to Ontario. As described in the ACGIH documentation (2012), nonane is a constituent of many hydrocarbon fuels and solvents, and therefore occupational exposure typically occurs simultaneously with exposure to other paraffinic and aromatic hydrocarbons. The group guidance value for C9 through C15 alkanes is 1200 mg/m3, which is essentially the same as the ACGIH TLV TWA for nonane. Therefore, lowering the TLV for nonane should also result in lessening exposures to other alkanes in the same group as well as to the other constituents of the hydrocarbon mixtures commonly encountered in the workplace.

To prevent central nervous system impairment, this review suggests that Ontario follow the health-based European examples described and reduce the TWAEV to 100 ppm, keeping the 200 ppm level as an STEV.

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Diacetyl (2,3-butanedione)

The ACGIH (2102) has produced its first TLV for diacetyl which is a TWA of 0.01 ppm and a STEL of 0.02 ppm. In the previous year NIOSH (2011) released their draft criteria document for diacetyl (and a potential substitute, 2,3-pentanedione) with the REL of TWA 0.005 ppm (action level 0.0026 ppm) and a STEL of 0.025 ppm.

The basis of the TLV and the REL for diacetyl is to prevent bronchiolitis-like lung disease; additional adverse health effects reported in humans include asthma and irritation of the skin, eyes and respiratory tract. Among other uses, synthetic diacetyl is a food flavour additive for popcorn, margarine, candy, etc. and health issues have been reported primarily from workers in diacetyl manufacturing and food production.

The results of animal testing have not been helpful in determining an NOAEL: in rats the upper airways are the target and in most reports, effects were seen in animals at every dose level. These OELs are therefore based on a quantitative risk assessment of human health data and industrial hygiene data accumulated over the past decade. A STEL is required because potentially very high peak exposures can occur, and these could be more toxic than the same cumulative dose spread out over a longer period of time (NIOSH).

NIOSH (2012) has determined that the REL is achievable with appropriate engineering controls and describes local exhaust systems with proven effectiveness. The USA has a modern industrial economy similar to Ontario.

The ACGIH has documented the literature it considered but not the process it has used to determine the TLV; the NIOSH REL is based on a worker having a less than 1 in a 1000 chance of developing reduced lung function due to diacetyl exposure. While both OELs are health-

based, the ACGIH TLV process is less well documented than and the TLV is twice as high as the NIOSH REL. We suggest that Ontario adopt the more protective NIOSH REL of TWA 0.005 ppm and either the NIOSH or ACGIH STEL.

Given that exposure causes severe irreversible lung disease and the high level of engineering control required to achieve this low level, substitution with a safer flavouring would be preferable. According to the NIOSH, 2,3-pentanedione is not a substantially safer product and REL and engineering controls needed are of the same order.

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Previous Submission (2004): Wood dust

Regulation 833 classifies wood dust into two categories:

1) certain hard woods as beech and oak with an eight hour exposure limit of 1 mg/m^3;
2) soft woods with an eight hour exposure limit of 5 mg/m^3 and a short-term exposure limit (STEL) of 10 mg/m^3.

ACGIH however lists wood dust two categories: Western Red Cedar, a softwood species but allergenic, with a TWA of 0.5 mg/m^3; and all other species having a TWA 1 mg/m^3 and removing (adopted 2004).

Wood dust can result from the process of cutting, milling, sawing, sanding and so forth of natural or processed wood. Wood is composed of polymeric compounds such as cellulose, polyoses, lignin, and a variety of smaller molecules know as extractives. These extractives are often defense mechanisms for trees to survive; however, some are toxic and allergenic to humans.

Exposure to wood dust can often be in combination with a variety of other hazards such as fungi, bacteria and pesticides. In other wood-related industries, workers can also be exposed to formaldehyde from adhesives and resins. Although the focus is on wood dust exposure, it is important to consider other exposures that may have potential ill health effects.

In 1965, an excess of sino-nasal adenocarcinoma was observed among furniture workers exposed to wood dust. This prompted further research which found an excess risk among other workers employed in wood-related industries such as logging, sawmills, furniture making, and carpentry. The highest risk of sino-nasal adenocarcinoma was observed in workers who were exposed to hardwoods such as beech and oak. However, a majority of the research, although examining the risk of cancer, did not specify the type of wood. Furthermore, wood workers are often exposed to mixed woods – not just one. Based on this information, IARC classifies wood dust as a Group 1 human carcinogen. IARC further states that this evaluation was based on workers exposed to hardwood dusts.

Several case-control studies indicate that there may be an excess risk of cancer of sino-nasal adenocarcinoma among workers exposed to softwood dusts. Unfortunately, in some cases there was confounding exposure to hard wood dusts. At this time, studies examining the exposure of softwood dusts and the risk of cancer are inadequate to estimate an OEL. There is however, sufficient data regarding nonmalignant respiratory effects of wood dust.

Upper and lower respiratory symptoms, airflow obstruction (other than asthma), and asthma have been reported in workers exposed to softwood species – particularly Western Red Cedar. Several studies found eye, upper and lower respiratory tract irritation, and altered lung function in sawmill workers exposed to concentrations of softwood dust at levels as low as 0.5 mg/m^3 up to a high of 32 mg/m^3. One other study of 315 sawmill workers exposed to other softwood dust (such as Douglas fir, Western hemlock, spruce, and balsam) experienced pulmonary function abnormalities and respiratory symptoms at dust levels ranging from 0.1 to 2.7 mg/m^3. Other studies have demonstrated that the risk of developing asthma to cedar dust increases as wood dust exposure levels increase. For the workers who developed asthma, the levels of exposure were on average less then 2 mg/m^3.

Based on these studies, workers exposed to softwood dust are still experiencing ill health effects at levels below the recommended TWAEV. It appears the changes to the TLV adopted by the ACGIH in 2004 are well founded. In addition, exposures levels to allergenic species of wood dust should be kept as low as reasonably achievable.

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Additional Substances Recommended for Improved OEL's:

Ozone

The ACGIH has adopted a lower standard for ozone which is based on the degree of physical activity the worker is engaged in:

Heavy work	0.05 ppm
Moderate work	0.08 ppm
Light work	0.10 ppm
All workloads for <2 hrs	0.20 ppm

While we are not convinced of the scientific evidence for the raising of the OEL for periods of less than 2 hours (a modified STEL?), we agree with the need for a more protective OEL for ozone that is graduated for the level of physical activity. We understand that the MOL is reluctant to adopt this ACGIH OEL due to the fact that ambient levels of ozone in Ontario can exceed these levels (particularly on hot summer days when the winds come from the south). It should be noted that the effects of ozone on the health of workers is the same whether the source of exposure is ambient as opposed to originating in the workplace. Despite the ambient source of ozone, employers are still able to take reasonable precautions in the circumstance for the protection of workers. For instance, during high ambient ozone conditions, employers can reduce workloads of outdoor workers to ameliorate the effect of ozone on the lungs (a similar approach is taken for the heat stress/strain TLV). Such a reduction in workload may also be required to address heat stress since high ozone episodes often coincide with hot weather. For indoor workplaces, there are simple adjustments that can be made to outdoor intake (a thin layer of activated charcoal filter) to remove or reduce ozone levels coming into enclosed workplaces. Thus we would challenge the MOL suggestion that an OEL should not be adopted if the ambient air quality conditions might on occasion exceed the OEL. Heat stress would also serve as an example of another exposure which is related to environmental conditions external to the workplace and yet exposure limits are enforced (http://www.labour.gov.on.ca/english/hs/guidelines/gl_heat.html).

Manganese

The major concern in relation to exposure to manganese is the development of neurological symptoms of hand tremour, reproductive effects, and psychological changes. A review of recent studies over the last 15 years including one conducted in Canada have indicated CNS effects below 0.2 mg/m^3 [(1)]. A key study which the ACGIH have relied upon for their determination of the TLV has been the study by Roels, et al.[(2)]. In this study the authors found that the upper 95th confidence limit of the lifetime integrated exposure metric corresponded to 3.575 mg/m^3–yrs of total Mn dust exposure and 0.73 mg/m^3-yrs of respirable Mn exposure. Assuming 40 years working life, these values would translate into 0.09 mg/m^3 for total Mn dust and 0.02 mg/m^3 of respirable Mn dust. If one uses the midpoint of the integrated exposure metric instead of the upper 95th confidence limit (as would be more appropriate) these levels would be even lower! In 2003, the ACGIH proposed to further lower the Mn exposure limit to 0.03 mg/m^3 in light of a

calculated LOAEL of between 0.15 and 0.035 mg/m^3, however, they specified that this was only applicable to respirable dust. Upon strong objections to the respirable designation (it was considered that non-respirable range particles should be included), they pulled back the recommendation and are in the process of revising it. Despite these ongoing considerations it is quite clear that pre-clinical neurological symptoms can be detected below the current 0.2 mg/m^3 TLV, and therefore it is recommended that this level be lowered to at least 0.03 mg/m^3 of total Mn dust to prevent the development of such symptoms.

A more recent development concerning the prevention of manganese health effects is the Brescia Declaration:

"On 17-18 June 2006, the Scientific Committee on Neurotoxicology and Psychophysiology and the Scientific Committee on the Toxicology of Metals of the International Commission on Occupational Health (ICOH) convened an International Workshop on *Neurotoxic Metals: Lead, Mercury and Manganese – From Research to Prevention (NTOXMET)* at the University of Brescia. Scientists and physicians from 27 nations participated. Data were presented for each of the three metals on environmental sources, fate and distribution; human exposure; clinical, subclinical and developmental neurotoxicity; epidemiology; risk assessment; and prospects for prevention. Ongoing and future studies were described and discussed.

…

The current occupational exposure standard may not protect workers against subclinical neurotoxicity. The value for air manganese concentration in inhalable/total dust of 100 µg/m^3 should be adopted to protect the workers from prolonged exposure and consequent long-term effects." (www.ntoxmet.it/**declaration**.pdf)

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Particulates Not Otherwise Classified (PNOC's)

An unpublished paper by Mermelstein and Kilpper titled "Xerox Exposure Limit for Respirable Dust (N.O.S.)" suggests that in order to prevent this overloading of the lung's defences, the exposure level to "nuisance" dust should kept below 0.4 mg/m^3 of respirable dust [1,2].

In another paper[3], the researchers retained by Xerox, calculated a 1 mg/m^3 respirable dust OEL but then suggested lowering this value by applying a safety factor since the calculation is conservative and leaves no allowance for errors in the assumptions. This would result in a greater than 10 fold reduction in the present OEL (occupational exposure limit). This paper also references Xerox's exposure limit for respirable dust of 0.4 mg/m^3. While Xerox internally experienced much apprehension when it stated it's intent to implement this much reduced OEL for respirable PNOC's, they have largely been successful in implementing it and have even

noticed a side benefit of improved morale due to the stringent housekeeping and exposure control needed to achieve this limit. There have been reports however, of workers who still experience symptoms even when this lower exposure limit is achieved.

Susan Woskie[4] reviewed the issues around the exposure standards for particulate in an article. In this review she suggests that using established models, 4 years of exposure to 0.25 mg/m^3 would lead to an accumulated dust burden in the lungs equivalent to the amount causing a 50% decline in lung clearance. Similarly, J. N. Pritchard[5] suggested the TLV of 10 mg/m^3 is two orders of magnitude (i.e. 100 X) too large.

An article by Chestnut et al.[6] provides some environmental epidemiological support for the recommendations to lower the nuisance dust OEL. This paper suggests that a significant decrease in forced vital capacity (FVC) is associated with exposures to total suspended particulate 121 $\mu g/m^3$ (i.e. 0.121 mg/m^3) and suggested the threshold for this health effect was at a level of 60 $\mu g/m^3$ (i.e. 0.06 mg/m^3). It should be emphasized that these dust measurements include materials other than insoluble mineral dust. It should also be noted that these levels are total dust concentrations. These findings have since been corroborated by numerous other studies[7] of ambient particulate and various health parameters.

An occupational epidemiological study related to this issue was published by N.S. Seixas et al.[8], in which they reviewed the exposure of coal miners to respirable coal dust since 1970. The authors found a significant association of obstructive lung disease with cumulative respirable dust exposures of 20 mg/m^3-years or more. Assuming a 45 year working life this cumulative respirable dust exposure would translate into a 0.44 mg/m^3 average lifetime exposure after which a significant health effect would be expected. Again it should be noted that coal dust is not considered a "nuisance" dust due to its silica content. However, it does seem to corroborate well with the animal study-based OEL recommendations. As a note of interest, the ACGIH in 1997 adopted a change to its TLV for coal dust lowering it from 2.0 mg/m^3 to 0.4 mg/m^3 for anthracite, and, to 0.9 mg/m^3 for bituminous coal (assuming less than 5% silica content).

A more recent review[9] has focussed in on the increased toxicity associated with ultrafine particulate, reinforcing previous recommendations for reductions in the PNOC exposure limits.

Given the evidence highlighted, the Ministry of Labour should seriously consider the need to lower the PNOC respirable dust OEL for the protection of the health of Ontario workers.

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Metalworking Fluids

Metalworking fluids were not on the list for updating, however, OHCOW's experience with workers affected by MWF and our own participation in MWF research has brought the need for a new OEL to our attention.

There have been three main published studies of cross-shift decrements of FEV_1 among metalworking exposed workers. Kennedy et al. found effects (5% cross-shift decrement) above a threshold of 0.2 mg/m^3 [(1)]. Kriebel et al., found effects (5% cross-shift decrement) at exposures above 0.15 mg/m^3 [(2)]. Robbins et al. found effects (10% cross-shift decrement) among a group of workers exposed to an average of 0.41 mg/m^3 [(3)].

With respect to occupational asthma, Kennedy et al. found significant new bronchial hyper-reactivity among apprentices after two years of exposure to an average exposure of 0.46 mg/m^3 [(4)]. Rosenman et al. reporting from data from an occupational asthma surveillance system in Michigan found metalworking fluids to be one of the major causes of reported occupational asthma[(5)]. Follow-up sampling showed all workplaces were below the 5 mg/m^3 exposure limit. Eisen et al.[(6)] found that exposure to 1 mg/m^3 of mineral oil mist had the same impact as smoking on FVC.

Our own work has shown similar comparisons with respect to respiratory symptoms[(7)]. NIOSH has recommended an exposure limit of 0.5 mg/m^3 [(8)] recognizing that health effects have been confirmed below this level. GM Canada has an agreement with the CAW that all new metalworking process installed will meet a 0.5 mg/m^3 exposure standard and that exposures related to existing processes will not exceed 1 mg/m^3. Given the current Ontario OEL of 5 mg/m^3, and given the large number of Ontario workers exposed to metalworking fluids, furthermore, given the OHCOW clinics experience with patients with lung problems due to metalworking fluids, we would strongly recommend adopting the new proposed ACGIH TLV of 0.2 mg/m^3 for mineral oil in metalworking fluids.

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Diesel Exhaust

The ACGIH[(1)] in 2002 proposed a TLV (TWAEV) of 20 μg/m^3 measured as elemental carbon (the proposal was withdrawn[(2)] in 2003 and never replaced). NIOSH[(1)] in 1988 recommended that diesel exhaust be treated as a human carcinogen. NIOSH suggests[(2)] that occupational exposures be controlled to as low as feasible. In essence, they require that sampling be done in unexposed areas, for example, the air outside the building, and that levels inside the building not exceed those of outside. The US EPA estimates that the ambient outdoor level of diesel exhaust (<10 μm particle size measured by elemental carbon) would be up to 1-3 μg/m^3[(4)]. Thus, NIOSH effectively recommends a level below 1 μg/m^3.

NIOSH has published a method[(5)] which they recommend to be used to measure the elemental carbon associated with diesel exhaust so as to distinguish it from other carbon sources such as cigarette smoke. In their analysis of exposures in the trucking industry NIOSH[(6)] estimated that a 13 μg/m^3 working life exposure was associated with a 1-2% (10-20/1000) excess risk of lung cancer above the 5% background lung cancer risk.

The EPA[(4)] has developed a reference concentration (RfC) for diesel exhaust of 5 μg/m^3 of DPM (roughly equivalent to 3.1-6.6 μg/m^3 of diesel exhaust as determined by elemental carbon) which was derived on the basis of dose-response data on inflammatory and histopathological changes in the lung from rat inhalation studies. .

Finally, there is the question of exposure to other gases (sulphur compounds, other nitrogen oxides, VOC's, etc.). The EPA[(4)] states "Effects of DE exposure could be additive to or synergistic with concurrent exposures to many other air pollutants. … (e.g., potentiation of

allergenicity effects, potentiation of DPM toxicity by ambient ozone and oxides of nitrogen)" (page 1-7).

Given the ubiquitous exposure to diesel exhaust among Ontario workers, we would strongly recommend the Ministry of Labour adopt at minimum the 2002 ACGIH proposed TLV if not the NIOSH Recommended Exposure Limit.

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