Occupational Health Clinics for Ontario Workers

Centre de Santé des Travailleurs(ses) de l'Ontario

PREVENTING OCCUPATIONAL DISEASE BY PREVENTING EXPOSURES

SUBMISSION TO THE MINISTRY OF LABOUR REGARDING:

2016 PROPOSED CHANGES AFFECTING THE CONTROL OF HAZARDOUS SUBSTANCES UNDER THE OCCUPATIONAL HEALTH AND SAFETY ACT
JUNE 6, 2016

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

Executive Summary

We thank the Ontario Ministry of Labour (MOL) for the opportunity to comment on the proposed changes to the Occupational Exposure Limits (OELôs) and associated policy proposals. We also appreciate that the MOL strategic plan includes efforts to prevent occupational disease. As per our mandate, the Occupational Health Clinics for Ontario Workers (OHCOW) strives to prevent occupational disease by primary, secondary and tertiary prevention (preventing harmful exposures, screening for early signs of occupational disease and recognizing cases of work-related disease). The OEL update process is a significant opportunity to prevent future occupational disease.

The following table summarizes OHCOWôs recommendation concerning the 2016 OEL update proposals:

substance	current OEL	proposed OEL	OHCOW proposal	rationale
Acetaldehyde	100 ppm	25 ppm	25 ppm	ocular and respiratory irritation
Atrazine	5 mg/m^3	2 mg/m^3	1 mg/m^3	German MAK
1-Bromopropane	10 ppm	0.1 ppm	0.1 ppm	ACGIH Documentation
Ethyl Isocyanate	n/a	0.02 ppm	0.004 ppm	US EPA AEGL
Methomyl	2.5 mg/m^3	0.2 mg/m^3	0.05 mg/m^3	Using default uncertainty factors
Methyl Formate	100 ppm	50 ppm	50 ppm (ceiling)	Austrian MAK
Naphthalene	10 ppm	10 ppm (no STEL)	0.01-0.5 ppm	WHO IAQ Guidelines, OHCOW experience, Bailey et al. (2016)
Nickel Carbonyl	0.05 ppm (TWA)	0.05 ppm (ceiling)	0.001 ppm (TWA)	WorkSafe BC OEL, US EPA AEGL, NIOSH REL, OSHA PEL
Oxalic Acid	1 mg/m^3	1 mg/m^3	1 mg/m^3 (skin)	German and Austrian MAKôs
Pentachlorophenol	0.5 mg/m^3 (inhalable)	0.5 mg/m^3 (inhalable & vapour)	0.3 mg/m3 (inhalable & vapour)	Consider banning substance as per Stockholm Convention on POPôs (2013)
Peracetic Acid	n/a	0.4 ppm (STEL)	0.2 ppm (STEL)	US EPA AEGL
Triethylamine	1 ppm	0.5 ppm	0.5 ppm	ACGIH Documentation
(Dimethylamine)*	5 ppm	5 ppm	0.5 ppm	ACGIH Documentation interpreted more rigorously
Trichloroacetic Acid	1 ppm	0.5 ppm	0.5 ppm	ACGIH Documentation
1,2,3-Trichloropropane	10 ppm (skin)	0.005 ppm	0.005 ppm (skin)	DFG [Deutsche Forschungsgemeinschaft]

*Dimethylamine was on the 2014 ACGIH adopted list but not on the MOL list

The MOL also included a number of policy issues in the 2016 proposal. The following summarizes OHCOWôs recommendations on some of these issues:

1. **Proposed New Respiratory Protection Requirements**: agree with following the CAN/CSA-Z94.4-11 ï ñSelection, use, and care of respirators standardò, but expand application to exposures to pesticides and providing protective provisions against dermal absorption.

2. **Proposed New Air Sampling Requirements**: agree with references to accepted analytical methods, however, the requirement to perform air sampling and to use appropriate (non-biased) sampling strategies still needs to be addressed.

3. **Proposed New Equivalency Clause**: in theory, the equivalency clause is reasonable, however, in practice, one needs to hold those proposing such equivalencies accountable to support their claims with adequate evidence (requiring a minimum degree of expertise) ï given that most workplaces may not have access to such expertise to make or be consulted on such claims, perhaps the MOL should screen and publish accepted claims of equivalency.

4. **Proposed Changes to Medical Examinations in O. Reg. 278/05**: as per our previous submission on the O.Reg 490/09 codes of practice submission, the MOL should consider the role of the recommended use of CT scans for lung cancer screen, and develop a mechanism to respond to developments and changes in evidenced-based recommendations.

We would also like to reiterate previous OEL update proposals which were not adopted in the past ï particularly for formaldehyde (1992), flour dust (2004), refractory ceramic fibres (2004), wood dust (2004), crystalline silica (2004 & 2006), beryllium (2009), sulphur dioxide (2009), hydrogen sulphide (2010), nitrogen dioxide (2013), manganese (2014), and, noise (2015).

Furthermore, in response to the invitation to nominate additional matters for consideration, we include recommendations regarding exposures to nanoparticles and carbon nanotubes, asbestos, styrene, ozone, particulates not otherwise classified (PNOCôs), metalworking fluids, diesel exhaust and Stoddard solvent.

We believe our recommendations, if adopted, would contribute significantly to the future prevention of occupational disease in the province of Ontario.

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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 seven 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 provided 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 (JHSCôs), unions, employers, individual workers and others, helping them to interpret exposure assessments, develop assessment strategies, directly assess exposures, deal with issues underlying the requests for assessments (e.g. worker symptoms and health conditions), address 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 Occupational Exposure Limits (OELôs) 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 TLVs particularly with the role that industry plays in influencing the Committee[1-5]. Reviews have shown that often the level set for the TLVs 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 TLVôs. However, a different challenge has been launched against the TLVôs in the last few years which also threatens to affect the manner in which they are set. A number of lawsuits were launched against the TLVôs from both industry and industrial disease victims. These legal challenges have had a ñchillingò effect on the organization and seem to have introduced hesitancy in reacting to situations where there is limited evidence. The ACGIH has withdrawn 39 TLVôs since 2005 for a variety of reasons (ñinsufficient dataò, deferred to Appendices, or combined with other TLV). In this effort to become more scientifically exact, protection for exposed workers is lapsing for the sake of scientific precision and avoidance of lawsuits.

A core principle of the former Ontario Joint Steering Committee on Hazardous Substances in the Workplace (JSC) was to consider the limits adopted by other jurisdictions (where they were established based on available documentation of evidence and where workplace parties were consulted). Such sources included jurisdictions with similar processes for updating their OELôs. Some reliable sources which we continue to consult include the EU SCOELôs, the German MAK documentations, DECOS from the Netherlands, the Nordic Expert Group (NEG), NIOSH, OSHA, US EPA IRIS database, ATSDR and the HSDB.

Regardless of the many sources available worldwide, we still come across situations where either there is no OEL, or else workers experience symptoms due to exposure despite the personal exposure concentrations being below an established OEL. In these cases, 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/en/common/ministry/publications/reports/campbell06/campbell06.aspx) needs to be included in Regulation 833 to address situations where the OEL has been eliminated, 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 OELôs are based on preventing irritation which would not necessarily be objectively verifiable, thus there is the need to address worker health effects which are not clinically measureable.

A Lack of a Legal Requirement to Measure Exposures:

Setting lower OELôs will not necessarily lead to reductions in exposure in Ontario workplaces. In order for an OEL to effectively lower workplace exposures, measurements must take place in workplaces particularly where exposures may exceed the 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 a need for a regulatory requirement to perform sampling for the purpose of exposure assessments if the changes in OELôs 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. Most other jurisdictions have some type of legal requirement for employers to perform hazard/risk assessments in consultation with worker representatives. We would recommend that Ontario also require workplaces to perform some type of risk assessment related to chemical exposures which may require exposure measurements. The MOL publication titled ñDesignated Substance in the Workplace: A Guide to the Regulationsò would be a good model to apply to all significant chemical exposures.

Again we would recognize the tremendous amount of work that was done by the Joint Steering Committee on Hazardous Substances in the Workplace (JSC) which led up to the proposed regulation (Draft #19, April 21, 1995) for the ñAssessment and Control of Exposure to Regulated Substancesò and the accompanying ñCode of Practice for Air Sampling and Analysisò (draft Feb 28, 1995). While this work is somewhat dated, the principles of requiring representative, accurate exposure measurement would still be of benefit to workplaces today.

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 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. ñthey should have sampled when é ò). 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 code of practice attached to a draft regulation on exposure assessment strategies (ñCode of Practice for Air Sampling and Analysisò (draft Feb 28, 1995)) 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 JSCôs draft regulation on sampling strategy. He has also remarked[21] that the advice he has offered in his many publications has been generally ignored. Recent studies[20] in sampling strategy offer defined optimum approaches to evaluating compliance. 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 assessment 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 changes in the OELôs were accompanied with legal requirements to perform exposure assessments and 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 updating the US OELôs from 1968 to 1989 would have on the percentage of compliance. Her findings were that even 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 OELôs would generally have some impact on the level of exposure experienced by most workers, but not address all concerns. Ms. Senn noted that there were some exceptions, however. For instance, the proposed lowering of the silica OELôs 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 OELôs 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 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 the OELôs. In fact, if one follows the history of OELôs, one will notice a gradual decline in most OELôs over the years as more evidence of workers experiencing symptoms and diseases is 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 chemicalôs toxicity until valid evidence is produced to the contrary. The concerns about the exposures to nanoparticles are a case in point, particularly carbon nanotubes and their similarity to asbestos fibres.

The MOL-instituted policy which recognizes that just because exposure assessments demonstrate compliance is no reason to ignore workersô symptoms nor health problems associated with such exposures. The fact is that there are relatively few investigations assessing worker health in relation to exposures, especially when one considers the number of workers actually exposed. In fact, there are a number of organizations publishing papers to be used in defense of corporations being sued for damages.[23]

For other OELôs where there is sufficient human evidence, a conscious decision has been made by the ACGIH 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 OELôs are set at an ñacceptable/tolerableò rate of occupational disease. The US the Supreme Court when considering the benzene OEL, determined that 1 worker death due to benzene exposure-related leukemia per 1000 exposed (i.e. 10^{-3}) could be considered a ñsignificantò risk (i.e. worth the effort to avoid). Due to this decision, for many years OELs (including the ACGIH TLVôs) were set to keep the risk of developing cancer to below 10^{-3} working lifetime risk (whereas environmental standards are usually set to keep the life-time risk below 10^{-5} to 10^{-6}). The Netherlands now requires that occupational cancer risks be quantified against two specified risk levels: $4x10^{-3}$ and $4x10^{-5}$[28]. Taking all these limitations into consideration, it is very clear that compliance with OELôs is in no way a guarantee that no significant health effects may occur among workers exposed!

It should also be noted that the work of CAREX (CARcinogen Exposure) and the OCRC (Occupational Cancer Research Centre) is extremely valuable in the context of preventing occupational disease (and in particular cancer). A paper by Del Bianco and Demers (2013)[22] notes that the trend for Canada from 1997-2010 show that the number of accepted claims for deaths due to occupational cancer ñhave surpassed those for traumatic injuries and disordersò[22].

Sensitizers, Carcinogens and Reproductive Hazards in the Workplace:

Workersô health in Ontario would benefit if exposures to sensitizers and carcinogens by any route were prevented through methods including substitution, engineering controls, isolation, local ventilation and protective equipment. 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 ñas low as possibleò 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. Ideally these efforts could be coordinated with the requirements in the Ministry of Environmentôs Toxic Substance Reduction Act & regulations. OHCOW also recommends that, similar to the ACGIH practice, these chemical have a notation included to identify the fact that they are carcinogens, sensitizers and/or reproductive hazards.

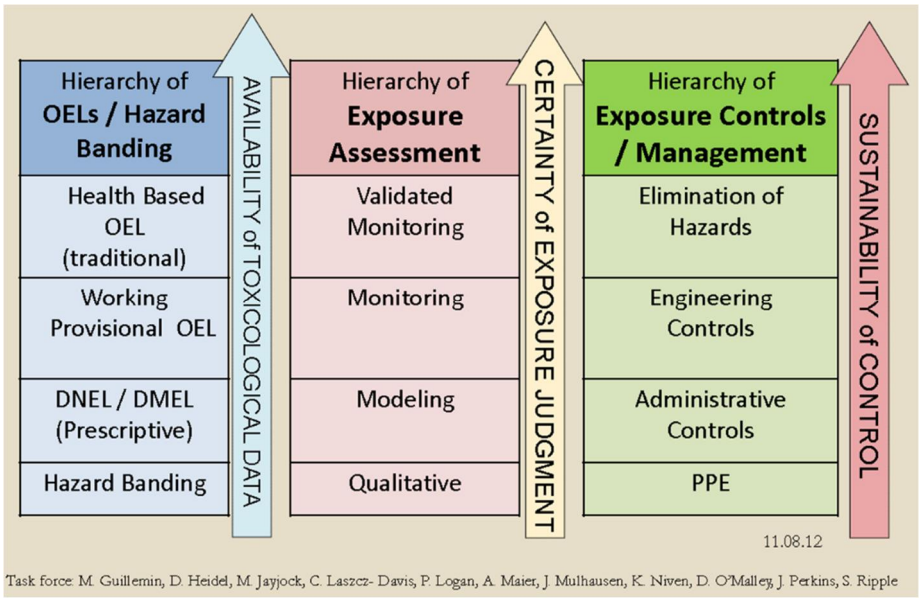
New Paradigms in Exposure Criteria:

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

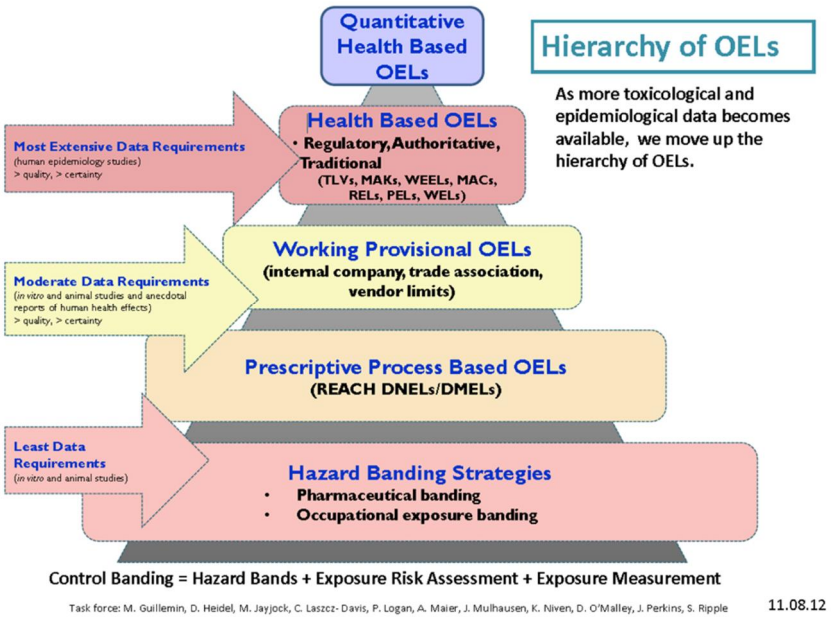
Similar graduated ranges have been established for volatile organic compounds (VOCôs)[13], although the main goal of measuring VOCôs is more to find the source and eliminate or control it to prevent exposure in the first place. Thus in the overall scheme of prevention, the single digit threshold concept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

Recently, new developments[24] have taken place in the field of occupational exposure limits. In addition to the traditional concept of a ñhierarchy of controlsò, the concept of a ñhierarchy of OELôsò and a ñhierarchy of exposure assessmentò have been put forward.



The hierarchy of OELôs begins at the lowest rung as hazard/exposure/control banding strategies which are based on qualitative (or at best semi-quantitative) categories of exposure determined by toxicological data and patterns of use (physical state, methods of use and quantities). Next comes a development required by the REACH (Registration, Evaluation, Authorization and Restriction of Chemicals) regulation in the European Union where chemical producers are required to follow a prescribed methodology to calculate Derived No-Effect Levels (DNELôs) or Derived Minimal-Effect Levels (DMELôs). While the ideals behind the legislation provided hope for the utility of the DNELôs in the workplace, actual practice has been somewhat disappointing[25, 26]. European Safety Data Sheets (SDSôs) require that DNELôs be included.

The next layer in the hierarchy of OELôs is provisional OELôs usually derived by manufacturers or trade associations in absence of an actual OEL or a protective OEL (e.g. Organization Resources Counselorsô (ORC) Metalworking Fluid Recommendations[27]). The highest rungs in the hierarchy are the legal OELôs and the health based OELôs (the top of the pyramid).



This framework for understanding OELôs is particularly useful for dealing with substances which do not have an OEL or for which the current OEL is not fully protective (e.g. wood dust, formaldehyde, silica, etc.).

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

It has also been recognized that most small or medium sized enterprises (SMEôs) 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 Health and Safety Executive (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 ñautomateò the decision logic exposure assessors would use to categorize exposures and recommend controls. The Ontario Ministry of Labour had a preliminary meeting with stakeholders a few years ago (Nov 23, 2001, chaired by John Vander Doelen) introducing the idea, however, nothing appears to have materialized from these efforts. The British Control of Substances Hazardous to Health (COSHH) Essentials is based on the GHS risk phrases ï as we moved to the GHS in Ontario, perhaps the MOL should give the control banding idea a second consideration particularly in light of providing support to small and medium sized enterprises (SME).

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 levels 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 JHSC levels.

OEL Update Process:

There have been a number of frustrations with the OEL Update process. We appreciate the recent efforts of the MOL to communicate with interested parties and the helpfulness of the OEL Update group in answering questions and explaining the proposals. Also the invitation to meet with the OEL Update group for more in-depth conversations is greatly appreciated.

In 2005 the MOL committed (website notice dated April 19, 2005) to a timeline for the process which involved publishing the proposal to update within 30 days of ACGIH publishing their changes, followed by a 60 day comment period. 45 days after the comment period the MOL would recommend adoption of non-contentious proposed OELs. If these timelines are too strict, then an amended timeline would be good to have rather than the current situation. However, since this is an annual process, the complete turnaround time should be at least within a year. It is also recommended that the MOL ñdouble-upò proposals to ñcatch-upò.

It would also be useful to get some response to the submissions (i.e. whether the suggestions were considered or if they were not, the reasons for dismissing them). OHCOW has repeated a number of suggestions some of which date back to the beginning of the process in 2004 and we still donôt know what response the MOL has to the recommendations.

It would also be useful to have clear statements on the website when the MOL decides not to update a particular OEL. Without directly talking to the OEL Update staff it is impossible to determine the status of the OEL proposals which the MOL has decided not to adopt (e.g. silica). If the MOL has reached such a conclusion, it serves the goals of transparency and accountability to publicly note such decisions. Otherwise, those parties who are interested will not know if a substance absent from the adopted list is ñunder further considerationò or has been dropped from the process.

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27. Cohen, H., and E.M. White, ñMetalworking Fluid Mist Occupational Exposure Limits: A Discussion of Alternative Methodsò, Journal of Occupational and Environmental Hygiene, 3:501ï 507 (2006)
28. Health Council of the Netherlands, ñGuideline for the calculation of occupational cancer risk valuesò, Report No. 2012/16E, The Hague, October 26, 2012.

Previous Proposals still Warranting Attention

The following are proposals that OHCOW has submitted in the past which have not been adopted, yet we think that significant occupational disease can be prevented if these were adopted. Where appropriate the submissions have been updated to reflect more recent advancements in the research.

Formaldehyde (1992)

The ACGIH has had a TLV of 0.3 ppm (ceiling) since 1992. In 1992 the Ontario Ministry of Labour published (Ontario Gazette Oct 31/92) a proposed limit of 0.5 ppm (8-hr TWA) for formaldehyde which was not adopted. This is quite disappointing since the scientific evidence in support of a lower OEL is overwhelming. The current Ontario OELôs for formaldehyde are 1 ppm (STEL) and 1.5 ppm (ceiling). While the ACGIH adopted a change the notation for the formaldehyde TLV to RSEN & DSEN (respiratory and dermal sensitizer) in 2015, the Ontario Ministry of Labour did not propose to update the Ontario formaldehyde OELôs in their 2016 proposal.

The 2015 ACGIH TLV Documentation is quite a detailed review of the existing literature and it recognizes that not all workers will be protected by the formaldehyde TLV:

> ñThe foregoing reports show that there is a substantial population, comprising up to 20% of the population, for whom airborne formaldehyde at concentrations on the order of 0.25 to 0.5 ppm is troublesome. It is plausible that a similar proportion (10% to 20%) who are more responsive, may react acutely to formaldehyde at very low concentrations, < 0.25 ppm,[(2)] and may be found among workers in schools, daycare centers, offices, laboratories, and other workplaces where formaldehyde or formaldehyde-containing products, such as particle board, insulation, etc., are used. Despite the intent of the TL V recommendations to protect most workers, it is believed that the recommended formaldehyde TLV-Ceiling of 0.3 ppm will not protect that portion of the workforce reported to be responsive to low ambient concentrations of this chemical.ò (page 23)

Health Canada (2006) has recommended a one hour Residential Indoor Air Quality Guideline of 0.1 ppm (based on a NOAEL of 0.5 ppm for 19 human subjects for 3 hours (Kulle, 1993)). The WHO has established an indoor air quality guideline of 0.1 mg/m^3 (0.08 ppm) based on a study (Lang et al., 2008) of 21 volunteers undergoing a 2 week randomized 4 hr exposures which identified a LOAEL of 0.5 ppm. While health effects were noted at 0.3 ppm, taking into account ñanxiousò personality traits accounted for the increased symptoms reports. The US EPA has published a draft report (IRIS, 2010) which has been reviewed by the National Research Council (NRC, 2011) which has a very extensive review of the evidence to 2009. Also when the NTP listed formaldehyde as a known human carcinogen, the NRC was asked to review their assessment and the review (NRC, 2014) support the NTPôs classification. In 2012 IARC recognized formaldehyde as a Group 1 Carcinogen (proven human carcinogen) citing sufficient evidence for cancer of the nasopharynx and leukemia, and also some evidence for sinonasal cancer.

Given this overwhelming documentation, OHCOW would recommend that the MOL adopt the current ACGIH TLV of 0.3 ppm (ceiling) and recognizing its limitations also give

serious consideration to adopting the either the Health Canada indoor residential standard of 0.1 ppm (1 hour), or the WHO formaldehyde guideline (0.08 ppm).

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- WHO, WHO guidelines for indoor air quality: selected pollutants, ñChapter 3: Formaldehydeò, pp.103-156, 2010.
- Lang, I., T. Bruckner, and G. Triebig, ñFormaldehyde and chemosensory irritation in humans: A controlled human exposure studyò, Regulatory Toxicology and Pharmacology 50:23ï 36 (2008).
- US EPA, ñToxicological Review of Formaldehyde - Inhalation Assessmentò, June 2, 2010.
- NRC, Review of the Environmental Protection Agency's Draft IRIS Assessment of Formaldehyde, Committee to Review EPA's Draft IRIS Assessment of Formaldehyde; National Research Council, 2011.
- NRC, Review of the Formaldehyde Assessment in the National Toxicology Program 12th Report on Carcinogens, Committee to Review the Formaldehyde Assessment in the National Toxicology Program 12th Report on Carcinogens; Board on Environmental Studies and Toxicology; Division on Earth and Life Sciences; National Research Council (2014)
- IARC, Chemical Agents and Related Occupations, ñFormaldehydeò, Volume 100F pp. 401-435, 2012.

Wheat Flour Dust (Total dust) (2004)

The current Ontario TWAEV for wheat flour dust is 3 mg/m^3 (total dust). The ACGIH refers to wheat flour dust as just flour dust and sets a TWA value of 0.5 mg/m^3 (inhalable fraction), where the TLV bases its critical effects on asthma, upper respiratory tract irritation and bronchitis. ACGIH also recognizes that flour dust is a sensitizer.

Cereal grains (e.g. wheat, oat, barley, rye, rice and corn) are collected and stored before they are prepared for human consumption. The grains are then milled to produce starch or flour for grain-based consumer products. Grain elevator workers, millers, flour packers, bakers and pastry chefs are some of the occupations where exposure to flour dust is inevitable. These workers can also be exposed to other sensitizers such as alpha-amylase, an enzyme that is found naturally in wheat flour; however, it is also added as a dough improver for baking. As a result of how flour is produced and stored, contaminants such as insects, mites and moulds can also induce respiratory allergy.

Reported illnesses associated with exposure to flour dust include conjunctivitis, rhinitis, dermatitis, and bakerôs asthma. Changes in lung function and increased risk of chronic bronchitis have also been observed from exposure to total flour dust. The more serious of these is bakerôs asthma. Currently, bakers along with automotive workers (exposed to isocyanates) are ranked amongst the highest occupations with reported numbers of occupational asthma. Aside from the morbidity of the disease, the economic cost and burden of managing asthma is staggering.

One study indicates wheat flour sensitization may occur at total dust levels as low as 0.5 mg/m^3. Other studies looking at exposure-response relationships, found that there are increased prevalence rates of sensitization at 1 mg/m^3. These studies indicate that the current TWAEV of 3 mg/m^3 is no longer sufficient to protect workers from becoming sensitized. Although these studies are usually based on exposures to wheat aeroallergens, studies indicate that there is cross-reactivity between different cereals ï suggesting the likely chance of multiple sensitizations. Therefore ACGIH recommends a TLV-TWA of 0.5 mg/m^3 for all types of flours.

Aside from sensitization, several studies noted increased prevalence of respiratory and asthmatic symptoms with exposures to total flour dust at levels approximately ranging from 1.35 to 3.57 mg/m^3. One other study also found that the frequency of symptoms generally increased with exposure intensity.

To protect workers who are exposed to flour dust, workplace exposures should be kept as low as reasonably achievable. Furthermore, the TWAEV should not exceed 0.5 mg/m^3.

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2. Smith T. Preventing bakerôs asthma: an alternative strategy. Occup Med. 2004; 54: 21-27
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5. Baur X. Bakerôs asthma: causes and prevention. Int Arch Occup Environ Health. 1999; 72: 292-296
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Refractory Ceramic Fibres (2004)

In 1999 the ACGIH[(1)] adopted the TLV of 0.2 f/cc for refractory ceramic fibres (RCF). This level was proposed by the MOL in 2004 but not adopted in favour of the current 0.5 f/cc OEL. The industry recommends a 0.5 f/cc exposure standard, however it also recognizes that OELôs as low as 0.1 f/cc have been adopted (e.g. Norway and France). Most provinces in Canada have lowered the RCF OEL to 0.2 f/cc consistent with the ACGIH TLV. NIOSH[(2)] has recommended a REL of 0.5 f/cc to prevent respiratory changes but it also noted that to prevent potential cancer risks the exposure should be kept below 0.2 f/cc. The SCOEL[(3)] has recommended an OEL of 0.3 f/cc. Verma et al (2004)[(4)] noted that construction exposures (including construction workers doing work in industrial settings such as removing and installing refractories) to RCFôs a decade ago were often (40%) over the ACGIH TLV. Given that some RCFôs under thermal stress will convert to crystalline silica, the combined exposure to RCFôs and crystalline silica warrants protection based on at least additive co-exposures. Given that both the OEL proposals for workers exposed to RCFôs and crystalline silica have not been adopted by the MOL, this leaves workers with combined RCF and silica exposure particularly vulnerable. OHCOW would, again, recommend the adoption of the ACGIH TLV for an OEL for RCFôs.

1. *ACGIH®,* Synthetic Vitreous Fibers: TLV® Chemical Substances 7th Edition *Documentation* Publication (2014).
2. NIOSH, ñCriteria for a Recommended Standard Occupational Exposure to Refractory Ceramic FibersòDHHS (NIOSH) Publication No. 2006ï 123 (2006)
3. Vanden Bergen E.A., P.S.J. Rocchi, and P.J. Boogaard, ñCeramic Fibers and Other Respiratory Hazards During the Renewal of the Refractory Lining in a Large Industrial Furnaceò Applied Occupational and Envrionmental Hygiene 9:32-35 (1994).
4. Verma, D.K., D. Sahai, L.A. Kurtz, and M.M. Finkelstein, ñCurrent Man-Made Mineral Fibers (MMMF) Exposures Among Ontario Construction Workersò, Journal of Occupational and Environmental Hygiene 1: 306ï 318 (2004).

Wood dust (2004):

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 ï 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 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 the 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 ï 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 than 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.

Ten years ago the SCOEL (the EU Scientific Committee on Occupational Exposure Limits) recommended(13) that the exposure limit for wood dust be lowered:

> "Taking into account the uncertainties and limitations of the available studies, it can be stated that exposure above 0.5 mg/m^3 induces pulmonary effects and should be avoided." (page 16)

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.

1. ACGIH [2005]. Wood dust. Documentation of the threshold limit values for chemical substances, 7th Edition. Cincinnati, OH: American Conference of Governmental Industrial Hygienists.
2. Demers P., Teschke K., Kennedy S. What to do about softwood? A review of respiratory effects and recommendations regarding exposure limits. Am J Ind Med. 1997; 31: 385-398
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13. SCOEL (Scientific Committee on Occupational Exposure Limits). ñRecommendation from the Scientific Committee on Occupational Exposure Limits: Risk assessment for Wood Dustò SCOEL/SUM/102 final December 2003

Silica (2004 & 2006)

In 2004 the MOL proposed lowering the silica OEL (or TWAEL as it is called in the designated substances regulation) 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. OSHA is now proposing to adopt a standard of 0.05 mg/m^3.

Silicosis is still a disease that affects Ontario workers(1) despite the fact that the knowledge and technology have long been available to prevent silicosis. The 2010 ACGIH documentation(2) for the silica TLV is quite sound in demonstrating that health changes are documented at levels between 0.05-0.06 mg/m^3 (early stages of fibrosis, silicosis and elevated rates of lung cancer). Thus the ACGIH recommendation is to lower the 8 hour time-weighted average (TWA) occupational exposure limit to 0.025 mg/m^3. We are not sure as to the reasons the MOL have decided not to adopt this OEL and thus we recommend it be reconsidered.

It is noted that in the current designated substances regulation incorporates the ALARA (as low as reasonably achievable) principle:

> ñEvery employer shall take all necessary measures and procedures by means of engineering controls, work practices and hygiene facilities and practices to ensure that a workerôs airborne exposure to each of the following designated substances and forms of designated substances is reduced to the lowest practical level and, in any event, does not exceed the TWA, STEL or C set out for the substance or form of substance in Table 1:ò (Section 16(2)) O. Reg 490/09)

We would endorse this approach, however we understand that legally there is opinion that the ALARA principle is not enforceable.

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.

1. Kachuri1, L., P.J. Villeneuve, M-É Parent, et al., ñOccupational exposure to crystalline silica and the risk of lung cancer in Canadian menò, International Journal of Cancer 135:138ï 148 (2014).
2. *ACGIH®,* Silica, Crystalline ï Ŭ-Quartz and Cristobalite: TLV® Chemical Substances 7th Edition *Documentation* Publication (2014).

Beryllium (2009)

Beryllium in particular has been a concern in Ontario workplaces with respect to sensitization associated with over-exposures. The ACGIH documentation(1) notes that cases of beryllium sensitization have occurred at lifetime weighted average exposures as low as 0.00005-0.0001 mg/m^3. Thus, an OEL of 0.00005 mg/m^3 seems to be the maximum at which one could set an

OEL for beryllium to avoid such cases of sensitization. Given the nature of dose-response relationships associated with sensitizers, it could be argued that even lower exposures may be associated with sensitization. A recent paper[2] has made the case for cost effective medical surveillance for early detection of beryllium disease, OHCOW would concur with this recommendation.

1. *ACGIH®,* Beryllium and Compounds: TLV® Chemical Substances 7th Edition *Documentation* Publication #7DOC-059 (2009).
2. Harber, P., S. Bansal and J. Balmes, ñProgression from Beryllium Exposure to Chronic Beryllium Disease: An Analytic Modelò, Environmental Health Perspectives 117:970ï 974 (2009).

Sulphur Dioxide (2009)

The ACGIH documentation[1] for the sulphur dioxide TLV cites a number of studies published as early as the 1960ôs up to the 1990ôs as evidence to support its TLV. It is somewhat dismaying that the studies cited to support the 0.25 ppm TLV-STEL were largely done 20 years ago and are only now being acted upon.

The SCOEL has a two tiered OEL for sulphur dioxide; for ñhealthy workersò the limit is 0.5 ppm (TWA) and 1 ppm (15 min STEL); and for ñasthmaticsò keep exposures below 0.2 ppm. Given that on average 8.5% of the population is asthmatic, OHCOW is not of the opinion that asthmatic workers should be excluded from the category ñhealthyò or ñgeneralò workers. Making this distinction for such a common variation in general health can lead to issues of discrimination ï focusing on the suitability of the individual to the workplace rather than the other way around. This approach would be a regressive policy step.

1. *ACGIH®,* Sulfur Dioxide: TLV® Chemical Substances 7th Edition *Documentation* Publication #7DOC-550 (2009).
2. SCOEL, ñRecommendation from the Scientific Committee on Occupational Exposure Limits for Sulphur Dioxideò, updated, December 2009.

Hydrogen Sulphide (2010)

In 2009 the ACGIH[1] adopted a 1 ppm 8-hr TWA TLV for hydrogen sulphide (H_2S) and a STEL of 5 ppm. The Ontario OEL of 10 ppm has been identified[2] as being associated with a significant decrease in oxygen uptake after as little as 30 minutes of exposure. This prompted the authors to question the ñscientific validityò of the 10 ppm OEL. The Dutch Expert Committee on Occupational Safety of the Health Council (DECOS) recommended[3] at health-based OEL of 1.6 ppm in 2006. Fielder et al (2007)[4] documented effects (likely due to odour) for subjects exposed to 0.05 ppm. Based on these considerations, OHCOW recommends that the MOL at least adopt the ACGIH TLV.

1. ACGIH, Hydrogen Sulfide. Documentation of the Threshold Limit Values and Biological Exposure Indices. American Conference of Governmental Industrial Hygienists. Cincinnati. 2014.
2. Bhambhani, Y., R. Burnham, G. Snydmiller, and I. MacLean, ñEffects of 10-ppm Hydrogen Sulfide Inhalation in Exercising Men and Women: Cardiovascular, Metabolic, and Biochemical Responsesò, Journal of Occupation & Environmental Medicine 39:122-129 (1997).

3. Dutch Expert Committee on Occupational Safety of the Health Council (DECOS), ñHydrogen sulphide: Health-based recommended occupational exposure limit in the Netherlandsò, 2006.
4. Fiedler, N., H. Kipen, P. Ohman-Strickland, et al., ñSensory and Cognitive Effects of Acute Exposure to Hydrogen Sulfideò, Environmental Health Perspectives 116:78ï 85 (2008).

Nitrogen Dioxide (2013)

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 EPAôs 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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1. ACGIH. (2012). Nitrogen Dioxide. Documentation of the Threshold Limit Values and Biological Exposure Indices. American Conference of Governmental Industrial Hygienists. Cincinnati. 2012.

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4. NIOSH. (2013). NIOSH Pocket Guide to Chemical Hazards. Nitrogen dioxide. Accessed March 2013. http://www.cdc.gov/niosh/npg/npgd0454.html

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Manganese (2014)

The major concern in relation to exposure to manganese is the development of neurological symptoms of hand tremor, reproductive effects, and psychological changes. A review of recent studies over the last 15 years including one conducted in Canada have indicated CNS effects occur at exposure levels 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}ï yrs of total manganese (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 these levels would be even lower! After many years of considering various proposals, in 2013, the ACGIH finally adopted the lower Mn exposure limit to 0.02 mg/m^3 for respirable particulate in light of a range of LOAELs between 0.0.032 and 0.038 mg/m^3.

Almost 8 years ago a group of concerned researchers issued a recommendation concerning the prevention of manganese health effects called the Brescia Declaration:

> ñOn 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.
>
> é
>
> The current occupational exposure standard may not protect workers against subclinical neurotoxicity. The value for air manganese concentration in inhalable/total dust of 100 ε g/m^3 should be adopted to protect the workers from prolonged exposure and consequent long-term effects.ò
> http://www.amm.se/icoh_sctm/pdf/Declaration%20of%20Brescia%20AJIM.pdf

The recommended level of 100 ε g/m^3 for inhalable/total dust is identical to the 2013 ACGIH TLV of 0.1 mg/m^3 (inhalable particulate matter).

The 2013 ACGIH TLV documentation(3) for manganese notes that:

> ñAccording to a statistical model of Roels et al. (1992), a level of 0.02 mg Mn/m^3 (respirable aerosol) would lead to impaired hand steadiness (detected with subtle tests but not clinically) in 2.5% of workers.ò (p. 2)

Thus, it is clear that even this OEL is not fully protective. Given our experience at OHCOW with welders(4) some of whom display these very symptoms, we can attest to the impacts that such ñimpaired hand steadinessò can have on the career of a welder whose livelihood depends on welding with a steady hand. Given the number of welders in Ontario, 2.5% would imply a great number of welders who would be losing their ability to do welding which requires fine motor control in the hand.

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Noise (2015)

In a previous OHCOW submission (2004) we cited a paper by Stekelenburg, (1982) which claims that an exposure to noise of 80 dBA for 40 years produces moderate hearing loss which in more than 10% of the exposed population will result in a difficulty in understanding speech after 10 years of retirement.

Rabinowitz (2012) suggests that NIHL is underdiagnosed and represents a significant public health issue. Nelson et al (2005) illustrate the global magnitude of the problem in terms of disability-adjusted life years, estimating that 18% (varying between 7-21% across sub-regions and being higher for men and workers in developing countries) of the burden of disabling hearing loss was attributable to noise exposure.

It is difficult to access publically reliable data regarding the extent of the noise induced hearing loss (NIHL) problem in Ontario. The online WSIB 2013 Statistical Supplements show a total of 43 allowed claims (for each year up to March 31st of the following year) ñdisorders of ear including deafnessò in 2005 and 31 ñdisorders of ear including deafnessò in 2009 (WSIB, 2014). In contrast, in 2011, the WSIB in a report to the Harry Arthurs Review showed a steady increase in NIHL registered claims from 3653 claims in 2005 to 5416 claims in 2009 (WSIB, 2011). This discrepancy may be due to the fact that for the purpose of compiling the Statistical Supplements, the WSIB only counts claims accepted by Mar 31st of the following year. Most occupational disease claims take much longer than that to settle.

In a paper published by Masterson et al. (2013), it was found that 18% of 1,122,722 worker audiograms collected from the NIOSH OHL Surveillance Project which met the NIOSH criteria for NIHL (>25 dB in either ear averaged over the 1, 2, 3, and 4 kHz frequencies). The data from this study is available online and when we applied the Ontario WSIB criteria (without making the adjustment for presbycusis) to the NIOSH data, the prevalence of Ontario WSIB-defined NIHL was 6% in this population. Obviously, the prevalence of NIHL is very dependent on the definition of NIHL applied to the data.

There is an additional problem of presbycusis masking the NIHL problem for workers older than 55 years old. Mahboubi et al., (2013) recently noted that, ñA limitation with almost all of NITS studies is that the presence of presbycusis will efface the notch, é ò (page 463), thus the ñnotchò in the audiogram of a worker with noise induced hearing loss will be masked by presbycusis resulting in the under-estimation of the prevalence of NIHL among older workers. Furthermore, a complicating factor associated with distinguishing between age-related hearing loss (AHL) and noise-induced hearing loss (NIHL) is the fact that noise exposure is cited as one of the four risk categories of AHL (Yamasoba, 2013).

Given the focus of the application of the noise regulation on the construction industry, it is worthwhile to note a number of recent studies of NIHL among construction workers (Leesen et al., 2011; Seixas et al., 2012; Leesen et al., 2014). Seixas et al. (2012) found in a prospective study of construction workers that:

> ñThe study provides evidence of noise-induced damage at an average exposure level around the 85 dBA level. The predicted change in HTLs was somewhat higher than would be predicted by standard hearing loss models, after accounting for hearing loss at baseline.ò (page 643)

Another researcher (Caciari et al., 2013) also noted a possible effect of air pollution on the hearing of workers working outdoors:

> ñDuring their working activity, outdoor and indoor workers are exposed to different noise levels LEX < 80 dB(A). At midï low frequencies (250ï 2000 Hz), the results show significant differences in the average values of hearing threshold between the two groups in both ears and for all age classes; there are no significant differences between the two groups at higher frequencies. The outdoor noise levels measured are not usually ototoxic and the hearing loss at midï low frequencies is not characteristic of the exposure to industrial noise. For these reasons the Authors hypothesize that the results may be due to the combined effect of the exposure to noise and to ototoxic air pollutants. The impairment of speech frequencies is disabling and involves the risk of missed forensic recognition.ò (page 302)

As mentioned above, in 1982 Stekelenburg noted that ñeven if 80 dBA is taken as a time weighted average limit - é - 10% of the exposed population will not be protected against impaired social hearing caused by noise.ò (page 408).

More recently, NIOSH describes the risks of NIHL associated with noise exposure as follows:

> ñé the 1997 NIOSH analysis of those frequencies likely to be affected by noise (1, 2, 3, and 4 kHz; é) demonstrates 1 in 4 workers (25%) will become hearing impaired at exposures to 90 dBA. By comparison, 1 in 12 workers (8%) are at risk of becoming hearing impaired at exposures to 85 dBA. The risk does not approach zero until exposures approximate 80 dBA.ò [Accessed at http://www.cdc.gov/niosh/programs/hlp/risks.html, on December 16, 2014]

These estimates are based on work that was published by Prince et al., in 1997 and became the basis of the NIOSH Criteria for a Recommended Standard - Occupational Noise Exposure (NIOSH, 1998).

While the NIOSH definition of NIHL is different from the Ontario WSIBôs definition (NIOSH: 25 dB averaged over 1, 2, 3, and 4 kHz, whereas for the WSIB: 22.5 dB averaged over 0.5, 1, 2, and 3 kHz), the point is quite obvious that if we want to prevent noise-induced hearing loss the noise exposure criteria should be lowered to 80 dBA $L_{ex,8}$. The scientific evidence clearly demonstrates that noise-induced hearing loss (NIHL) begins at noise exposures of $L_{ex,8}$ of 80 dBA. If the MOL is truly serious about preventing NIHL it is imperative to reduce the exposure limit to an $L_{ex,8}$ of 80 dBA.

Rabinowitz et al., (2007), reviewed the 10 year experience of a large industrial cohort and concluded:

> ñIn this modern industrial cohort, hearing conservation efforts appear to be reducing hearing loss rates, especially at higher ambient noise levels. This could be related to differential use of hearing protection. The greatest burden of preventable occupational hearing loss was found in workers whose noise exposure averaged 85 dBA or less. To further reduce rates of occupational hearing loss, hearing conservation programmes may

require innovative approaches targeting workers with noise exposures close to 85 dBA.ò (page 53)

The European Union Directive 2003/10/EC (which is over 10 years old) has a lower action level of 80 dBA $L_{ex,8}$ at which exposure employers must provide information and instruction, hearing protectors are to be made available, and, workers have a right to a preventive audiometric exam if a noise assessment indicates the possibility of a risk to hearing.

Furthermore, it is now well recognized that certain chemical exposures may induce ototoxic reactions making the worker more sensitive to NIHL (ACGIH, 2006).
The Nordic Expert Group (Johnson & Morata, 2010) classified three categories of ototoxic chemicals based on the strength of the evidence:

ñ1) Human data indicate auditory effects under or near existing OELs. There are also robust animal data supporting an effect on hearing from exposure.
2) Human data are lacking whereas animal data indicate an auditory effect under or near existing OELs.
3) Human data are poor or lacking. Animal data indicate an auditory effect well above existing OELs.ò (page 143)

Category 1 chemicals include, styrene, toluene, carbon disulphide, lead, mercury, and carbon monoxide. Category 2 chemicals include, para-xylene, ethylbenzene, and hydrogen cyanide. Thus workers working with exposures to these chemicals (some of which are quite common in industrial work environments), imply a higher risk for workers exposed to noise between 80-85 dBA.

The evidence is quite clear, if we are serious about preventing NIHL, the $L_{ex,8}$ needs to be lowered to 80 dBA.

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2016 OEL Update Substances

Acetaldehyde

A TLV Ceiling of 25 ppm is recommended to reduce the potential for ocular and upper respiratory tract irritation. A previous TLV of 100 ppm was established in 1973. The A3 classification was issued due to the identification of nasal and laryngeal carcinomas in rats and hamsters.
Acetaldehyde is possibly carcinogenic to humans (Group 2B Possibly carcinogenic to humans). NIOSH considers acetaldehyde to be a potential occupational carcinogen as defined by the OSHA carcinogen policy.
The change from an A3 carcinogen to an A2 carcinogen would be appropriate here.

Alarie Y [1981]. Dose-response analysis in animal studies: prediction of human responses. Environ Health Perspect 42:9-13.

Atrazine

Atrazine (6-chloro-N-ethyl-N'-(1-methylethyl)-1,3,5-Triazine-2,4-dia); CAS 1912-24-9) is used as an herbicide for the control of annual broadleaf and grassy plants (ACGIH, 2014; JMPR, 2009). Its use is banned in the EU (ECHA, 2012). The ACGIH TLV-TWA was 10 mg/m^3 when first adopted in 1976 and has been 5 mg/m^3 since 1983; the notation ñNot Classifiable as a Human Carcinogenò (Group A4) was added in 1995; and, two years ago the ACGIH (2014) changed its TLVï TWA for Atrazine from 5 mg/m^3 to 2 mg/m^3 ñInhalable Particulateò and changed to Group A3, Confirmed Animal Carcinogen with Unknown Relevance to Humans. The current TLVï TWA (ACGIH, 2014) is intended to protect against possible hematologic, reproductive, and developmental effects seen in animal studies.

In Germany, the OEL (MAK) for atrazine is 1 mg/m^3 ñInhalable Particulateò as a TWA (Germany, 2013). The ACGIH and German OEL-TWAs differ by a factor of 2 - the German MAK is essentially based on reports from the USEPA (2002) and a FAO/WHO report (JMPR 2009).

This difference between the German MAK and the current ACGIH TLV appears to stem from the selection of the research for the critical effect and the applying of uncertainty factors. The critical effect of atrazine in the German assessment is from a study of estrous cycle alterations and LH surge attenuation in Sprague Dawley rats in which the NOAEL was reported to be 1.8 mg/kg/day. This datum was from an unpublished study by Morseth (1996) that had been referenced in the reviews by the JMPR (2009) and the USEPA (2002). For this effect, the ACGIH documentation cites a study by Eldridge et al. (1999) in which the observed NOEL for estrous cycling and body weight changes was 50 ppm in the diet - equivalent to 3.3 mg/kg/day. The NOAELs in these studies differ by a factor of 1.8. In addition, the conversion of animal data to the equivalent dose in humans and the subsequent application of uncertainty factors is more clearly articulated in the German OEL documentation (Germany, 2013) than in the ACGIH documentation (2014).

The German documentation determined that the results for skin sensitization are inconsistent based on the FAO/WHO report (2009) which found that atrazine tested positive as a skin sensitizer in guinea pigs and the EPA (2002) review which found that atrazine was not a skin sensitizer. The ACGIH documentation is based only on EPA data showing atrazine to be non-sensitizing in a guinea pig model.

Products containing atrazine are available in Canada but are not manufactured in Ontario (PMRA, 2015; and communications with manufacturers and distributers: Syngenta, Bayer and BASF). CAREX Canada will be developing a profile for atrazine but does not have occupational exposure estimates for atrazine available on its website at this time (CAREX, 2011). Occupations with potential for exposure in Ontario would be herbicide supply workers and those applying them and in contact with crops e.g. farmers and farmworkers. Many Ontario workers who could potentially benefit from the regulation of atrazine appear to be excluded from doing so by Ontario Regulation 414/05 Farming Regulations.

Germany has lowered its OEL-TWA for atrazine from 2 mg/m^3 to 1 mg/m^3 (Germany, 2013). This level is based on research results that were not considered by the ACGIH and a more clearly articulated risk assessment methodology. This chemical has been banned in Europe. There is some lack of certainty regarding skin sensitization and the potential for cancer in exposed populations. It would be prudent at this time to adopt the more protective German OEL already in existence.

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1- Bromopropane

The old TLV (ACGIH) was TWA 10 ppm, no STEL, A3 notation- TLV basis listed as liver and embryofetal damage, neurotoxicity

2014 Adopted TWA 0.1 ppm, no STEL, A3 notation- TLV basis CNS impairment, peripheral neuropathy, haematological effect, developmental and reproductive toxicity (male and female)

OSHA-NIOSH Hazard Alert published 2013 contains the following information:
Solvent used in drycleaning, degreasing, spray adhesives- used in operations like asphalt production, aircraft maintenance, synthetic fibre manufacturing

Workers can be exposed by breathing in the vapour or mists of spray, or THROUGH SKIN ABSORPTION.
Impacts on health have been documented in workers exposed for as little as 2 days.

The National Toxicology Program considers 1-BP as reasonably anticipated to be a human carcinogen, based on increased lung, large intestine and skin cancers in rodents.

TURI Massachusetts Chemical Fact Sheet identifies 1-BP as a relatively new solvent, being used as outlined above. Use is increasing as an alternative to other solvents which are tightly regulated, such as perchlorethylene, trichloroethylene, and methylene chloride.

ToxNet cites neurological human health effects, in some cases under relatively short exposure periods (months)

High exposure levels have been recorded in a number of industries:
foam cushion gluers- 91-176 ppm
metal parts cleaner
18-381 ppm in plants where spray adhesive was used,
0.04-0.63 where used as a vapour degreaser
60-261 ppm where used as a solvent with a spray glue gun
Up to 27.8 (mean 1.42) cleaning metal and painted surfaces
Dry cleaning from 0.24 54.55 ppm

Conclusion
The animal studies are compelling in leading to the conclusion that there is a serious potential risk to exposed workers. Therefore reduction of the OEL for 1-BP to 0.1 ppm is warranted. As evidenced by the data presented above, segments of industry have allowed very high exposures which will be an ongoing concern.

The Toxic Use Reduction Institute (TURI), University of Massachusetts Lowell, Lowell Mass, www.turi.org. April 2014

Ethyl Isocyanate

Ethyl isocyanate is used as an intermediate in the manufacturing of pharmaceuticals and pesticides. It is a lacrymator and a severe eye, skin, and respiratory tract irritant which can cause acute inhalation injury. (Haz-Map)

ACGIH has adopted a TLV of 0.02 ppm with a STEL of 0.06 ppm to protect upper respiratory tract and eye irritation. ACGIH reported in the TLV documentation that the toxicity data of ethylene isocyanate is very limited for the TLV development purposes. Therefore, the TLV is derived from a similar low molecular weight chemical called methyl isocyanate. Although methyl isocyanate has a similar chemical structure, its toxicity is less than ethylene isocyanate (LC50: 27 ppm ï 82 ppm vs. 6.1 ppm for 6 hours). A notation of dermal sensitizer is also recommended. (TLV documentation)

DFG MAK does not have a TLV for ethylene isocyanate but it has assigned a MAK concentration of 0.01 ppm to methyl isocyanate. (MAK documentation is in German). Following are the AEGLs for ethylene isocyanate by US EPA:

Ethyl isocyanate (ppm)

	10 min	30 min	60 min	4 hr	8 hr
AEGL 1	NR	NR	NR	NR	NR
AEGL 2	0.20	0.065	0.034	0.0085	0.0040
AEGL 3	0.60	0.20	0.10	0.025	0.013

NR= Not recommended due to insufficient data

Source: AEGLs. Ethyl Isocyanate. http://www.epa.gov/oppt/aegl/pubs/rest231.htm .

US EPA has also mentioned in the AEGLs documentation that the toxicity data for ethylene isocyanate is limited to some rat lethality studies due to which AEGLs 2 and 3 are derived from methyl isocyanate AEGLs. An exposure above AEGL 2 (for 4 and 8 hours) and AEGL 3(for 8 hours) can cause adverse health effects in an individual corresponding to AEGL 2 and 3 type adverse health effects.

Since EPA has recommended an AEGL 2 of 0.004 ppm for 8 hours above which an individual may show reversible or serious adverse health effects, OHCOW recommends to adopt an OEL of 0.004 ppm with an STEL of 0.06 ppm. We support the dermal sensitizer designation.

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Methomyl

Methomyl (Lannate® (DuPont 1179; Methavin®; S-Methyl-N[(methylcarbamoyl)oxyl)thioacetimidate; Nudrin; CAS 16752-77-5) is a carbamate insecticide with anticholinesterase activity that is used on a wide variety of agricultural crops including vegetables that are thinned, pruned and harvested by hand such as broccoli, cabbage, cauliflower, Brussels sprouts, lettuce, peas, potatoes, snap beans, tomatoes, sweet corn that is de-tasseled by hand, fruit such as apples that are thinned by hand, soybeans, vines, hops, canola, grains including flax, wheat, barley and oats, field crops including cotton and ornamentals (ACGIH, 2014; Health Canada, 2010). It is also used to kill flies in barns, stables, poultry houses, feed lots, kennels, etc. (PMRA, 2015). Its use is banned in the EU (ECHA, 2010). The ACGIH TLV-TWA was 2.5 mg/m^3 with a skin notation when first adopted in 1977; the notation ñNot Classifiable as a Human Carcinogenò (Group A4) was added in 1996; and, two years ago the ACGIH (2014) changed its TLVï TWA for methomyl from 2.5 mg/m^3 to 0.2 mg/m^3 ñInhalable Fraction and Vaporò and retained the Group A4 notation and the skin notation. The previous TLV was intended to minimize the potential for symptoms of cholinesterase inhibition. The current TLVï TWA (ACGIH, 2014) is intended to protect against severe toxic responses: acetylcholinesterase inhibition; male reproductive damage and hematologic effects seen in humans and experimental animal studies.

Methomyl is marketed in the form of water-soluble powder, water miscible solution a wettable powder and fly bait pellets.

The change in the TLV is based is on the experimental work of Shalaby et al. (2010) who reported an LOAEL of 0.5 mg/kg bw for reproductive effects in male Sprague Dawley rats: decreased the fertility index, weight of testes and accessory male sexual glands, serum testosterone level and sperm motility and count, and increased sperm cell abnormality. The ACGIH (2014) extrapolated the LOAEL in rats to an airborne exposure level for humans of 3.5 mg/m^3 and then applied a factor of 17.5 to derive the TLV-TWA of 0.2 mg/m^3. Typical default assessment factors for inter-species (rat) and intra-species (worker) are 4 and 3 respectively; the typical default assessment factor of 3 is applied when extrapolating from a LOAEL instead of a NOAEL; the study by Shallaby et al. (2010) is subchronic and therefore extrapolation involves the application of a further factor with the default value of 2 (ECETOC, 2003). The product of these factors is a combined uncertainty factor of 72, which is four times that used by the ACGIH (2014), and would result in a TLV-TWA of 0.05 mg/m^3. Other schemes use typical default values of 10, 10 and 3, respectively for these uncertainty factors (Abadin et al., 2007).

Products containing methomyl are available in Canada but are not manufactured in Ontario (PMRA, 2015; and communications with manufacturers and distributers: Dupont, Troy, Wellmark and Engage). Occupations with potential for exposure in Ontario would be insecticide supply workers and those applying them and in contact with crops e.g. farmers and farmworkers. The many Ontario workers who could potentially benefit from the regulation of methomyl appear to be excluded from doing so by Ontario Regulation 414/05 Farming Regulations.

This chemical has been banned in Europe. Given the finding of severe male reproductive effects at levels that correlate with human airborne exposure of 3.5 mg/m^3, the ACGIH (2014) has lowered the exposure limit by a factor of 17.5 for a TLV-TWA of 0.2 mg/m^3. The ACGIH has not described their methodology; however, using accepted methods a factor of 72 is obtained, which, when applied results in a TLV-TWA of 0.05 mg/m^3. This more protective OEL is methodologically sound and should be adopted in Ontario.

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Methyl Formate

The ACGIH (2015) TLV-TWA for methyl formate of 50 ppm is based on the same research (BASF AG, 2003; ACGIH, 2015) and has the same value as those of recently updated OELs in several countries. These countries also include the skin notation. However, the ACGIH STEL of 100 ppm is twice the ceiling (instantaneous) value of the Austrian MAK, which is 50 ppm (Austria, 2007).

Human volunteers exposed to 100 ppm methyl formate for 8 hours had subjective effects of fatigue potentially increasing the risk of accidents (Sethre et al., 1998, 2000); no effects were seen in workers exposed to 36 ppm methyl formate and 44 ppm isopropanol (Sethre, 1998). As the available literature does contain the report of health effects on humans at 100 ppm, it would be prudent to consider the approach taken by Austria in setting 50 ppm as the Ceiling level rather than the TWA to ensure that Ontarioôs OEL is sufficiently protective.

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Naphthalene

The previous ACGIH TLV for naphthalene was 10 ppm (TWA ï since 1965) and 15 ppm (STEL since 1976). In 2011 the ACGIH proposed lower the TLV to 2 ppm. This proposal was then raised to 5 ppm in 2012 and raised again to 10 ppm in 2013; then adopted in 2014. The exposure limit of naphthalene has become very controversial with the focus on the relevancy of animal cancer studies. The WHO naphthalene guideline for indoor air quality is 0.01 mg/m^3 (or 0.002 ppm annual average (24 hrs 365 days) which would be equivalent to about 0.01 ppm for an 8-hr working day). This recommended level is based on an LOAEL derived from a rat study which showed almost all rats exposed to 53 mg/m^3 (10 ppm) for 6 hrs/day, 5 days/wk for 104 weeks experienced severe inflammation. Even industry based risk calculations have shown the benchmark dose level (BMDL) based on changes to the respiratory and olfactory epithelium to be in the range of 0.63-1.43 ppm (human equivalent concentration). Applying an uncertainty factor for inter-species extrapolation and inter-individual differences would suggest an uncertainty value of at least 30 to 100 (i.e. <0.02 ppm).

In our own OHCOW investigations we have observed, wide spread complaints due to naphthalene exposure at concentrations estimated to be just above the odour threshold (around 0.003-0.010 ppm). Based on the consideration of this literature and based on our direct experience investigation health complaints due to workplace exposures to naphthalene OHCOW recommends that the OEL for naphthalene be lowered to a level below 0.5 ppm and preferably 0.01 ppm.

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Nickel Carbonyl

Nickel Carbonyl is formed in a chemical process known as the Mond process by passing nickel over the carbon monoxide at 50-60 °C. It is used in refining metallic nickel, nickel plating, in the production of high purity fine nickel to be used for metallurgical and electronics industries, and in the synthesis of methyl and ethyl acrylates as a catalyst.

The ACGIH has adopted a ceiling TLV of 0.05 ppm in 2014 by changing its previous TLV-TWA of 0.05 ppm which was adopted in 1977. The ACGIH has changed the TWA TLV to a ceiling level due to nickel carbonylôs nature of causing acute adverse health effects. The basis of this ceiling limit is lung irritation. It is mentioned in the ACGIH TLV documentation that the primary acute effect of nickel carbonyl is on the lungs with initial symptoms of headache, nausea, chest tightness, and dizziness followed by delayed symptoms of shortness of breath, cyanosis, pulmonary edema, and low grade fever upon acute exposure. ACGIH has also

assigned a cancer rating of A3 to nickel carbonyl which means itôs a confirmed animal carcinogen with unknown relevance to humans.

NIOSH and OSHA have an exposure limit of 0.001 ppm TWA without a STEL. The WorkplaceSafe BC from British Columbia also has a similar exposure limit of 0.001 ppm TWA. Other jurisdictions and organizations such as DFG MAKs, AIHA-WEELs, HSE-WEL, REACH, and EU-SCOEL has no exposure limit for nickel carbonyl.

Acute Exposure Guidelines (AEGL) has also been derived by EPA from animal and human studies. Nickel carbonyl does not have AEGL-1 which means that exposure level above which discomfort and irritation occurs cannot be derived because of nickel carbonylôs severe toxic nature. The levels which should be noted here are AEGL-2 for 60 minutes, 4 hours, 8 hours, and AEGL-3 for 4 hours, and 8 hours.

Nickel carbonyl					
ppm					
	10 min	**30 min**	**60 min**	**4 hr**	**8 hr**
AEGL 1	NR	NR	NR	NR	NR
AEGL 2	0.10	0.072	0.036	0.0090	0.0045
AEGL 3	0.46	0.32	0.16	0.040	0.020

Source: AEGL ï Nickel Carbonyl http://www.epa.gov/oppt/aegl/pubs/results63.htm

The levels (AEGL-2 for 1 hour, 4 hours, and 8 hours) are below the adopted ceiling level of 0.05 ppm. These are the levels above which general and susceptible population could experience irreversible or other serious, long lasting adverse health effects and impaired ability to escape. Similarly, AEGL-3 for 4 and 8 hours are below the ceiling limit above which an individual can experience life threatening effects or death.

Shi (1994) studied toxic effects of nickel carbonyl exposure in animals and humans. Airborne concentrations of nickel carbonyl for causing acute toxicity are not available, however, it is reported in the study that the long term exposure to 0.003-0.08 mg/m^3 (0.0004-0.01 ppm) of nickel carbonyl can effect workersô health. The lung function was found to be abnormal in one third of the exposed workers who worked between 10-20 years. The lung function and blood gas analysis of workers exposed to low levels of nickel carbonyl was also studied by Shi (1994) on the same worker population. It is concluded in the study that exposure to low concentration of nickel carbonyl (0.001-0.07 ppm) can impair lung function of workers.

There are no levels recommended (NR) for AEGL 1 and AEGL category 2 and 3 has shown that exposures below the ACGIH adopted ceiling level of 0.05 ppm can cause irreversible and serious adverse health effects. Moreover, NIOSH has a REL of 0.001 ppm TWA for nickel carbonyl owing to its cancer causing potential. Shi (1994) has also reported abnormal lung function from nickel carbonyl exposure in the range of 0.0004 ï 0.01 ppm and 0.001-0.07 ppm, which is again lower than 0.05 ppm.

Keeping in mind the potential of nickel carbonylôs chronic and acute toxicity at low and higher concentrations respectively, OHCOW recommends to keep the exposure as low as reasonably possible (ALARA) or at least adopt an OEL-TWA of 0.001 ppm similar to NIOSH, OSHA, and WorkSafe BC with a ceiling or excursion limit of 0.005 ppm (5 times the exposure limit).

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Oxalic Acid

The 2015 ACGIH TLV for oxalic acid now distinguishes the anhydrous and the dihydrate forms, leaving the TWA and STEL unchanged at 1 mg/m^3 and 2 mg/m^3 respectively, applied to both forms. Other countries do have a lower STEL, for instance Germany has a STEL of 1 mg/m^3. In addition, both the German and Austrian OELs contain a skin designation (Austria, 2007; Germany, 2015); however, the ACGIH (2015) concluded that insufficient data were available to make this recommendation.

The ACGIH STEL for oxalic acid is based on the irritant potential of other carboxylic acids to the eyes, skin and respiratory tract. The STEL for oxalic acid is based on phosphoric acid; however, oxalic acid is a slightly stronger acid (ACGIH, 2015).

The German STEL and Austrian and German skin designation, which appear to be based on irritant potential, are inconsistent with the ACGIH TLV and should be considered at this time to ensure that Ontarioôs OEL is based on the most current and appropriate interpretation.

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Pentachlorophenol

Pentachlorophenol (PCP) is primarily used as a fungicide for industrial wood preservation, insecticide for termite control and a herbicide. It is used in Canada for utility poles, railroad ties, foundation pilings, timbers in highway construction, fence posts (Carex Canada, 2014). It has not been produced in Canada since the early 1980ôs but instead has been imported as a wood preservative (Carex Canada, 2014). PCP is only allowed for wood preservation with additional restrictions/regulations in Canada, Mexico and the United States (UN, 2013). Ontario has the highest worker population exposed to pentachlorophenol followed by Quebec, British Columbia and Alberta (Carex Canada).

Farm workers, electrical power line and cable workers are the occupations most exposed to this chemical. (Carex Canada,2014). The National Toxicology Program has declared ñPCP and itôs by-products to be reasonably anticipated to be a human carcinogen based on the limited evidence from studies in humans showing that a causal relationship between exposure to PCP and Non-Hodgkinôs Lymphoma is credible.ò IARC has declared combined exposures to the polychlorophenols or their sodium salts to be classified as Group 2B. IARC has declared the agent to be carcinogenic to animals, as it causes adrenal tumors in mice. (IARC monograph, Volume 53, 1991). In humans IARC has found that there are suggested links to non-Hodgkinôs lymphoma (NHL) and soft tissue sarcomas in those occupationally exposed. (IARC monograph, Volume 71, 1999). Demers et al., 2006 found strong dose-response relationships between pentachlorophenol exposure and NHL, multiple myeloma and kidney cancer. Elevations of lung cancer and non-hodgkin's lymphoma were found by Ruder and Yiin, 2007. Cooper et al., 2008 found a strong association between heamapoietic cancer and pentachlorophenol.

Dermal exposure is the most important route of exposure, as it was found in a study to account for 95% of exposure for workers in sawmills (Fenske, et al., 1987). Hayes, 1982 found that dermal absorption of PCP or its sodium salt can also lead to systemic toxicity and death, with time until death varying from 3- 30 hours after the first appearance of symptoms.

Non cancer health effects due to chronic high dose occupational exposures to PCP, which are of great concern, are listed as follows (Taken from ACGIH 2014 data on Pentachlorophenol): Upper respiratory tract inflammation, bronchitis (Baader and Bauer, 1951; Klemmer et al., 1980; Walls et al., 1998); Emphysema (Walls et a. 1998); Tuberculosis, Pleurisy or pneumonia (McLean et al., 2009)

Respiratory diseases are of great concern and need more attention than is offered by the Government of Canada at this time. More funding for research is required in terms of finding ways to prevent and cure the diseases. If we know that chemical agents can affect the respiratory system in irreversible or damaging ways, we should be obliged to minimize if not eliminate exposures to those agents for the protection of workers and their families. Mortality due to non-malignant respiratory disease was elevated in the McLean et al., study findings and COPD was found to be increased in the Ruder and Yiin, 2011 study, due to exposure to PCP.

As per the Stockholm Convention on Persistent Organic Pollutants report from the ninth meeting in November 2013, PCP had either no use or is banned in all E.U. member states, China, India, Indonesia, New Zealand, Russia and Switzerland. ñThe committee states that PCP as a result of its long-range environmental transport, can lead to significant adverse human health and or environmental effects and that global action is warranted.ò

Fisher, 1991 reports ñthat an estimated 96.5 percent of PCP in the environment will end up in the soil. Thus researchers looking at environmental sources of human exposure to PCP have calculated that the food chain especially fruits, vegetables and grains account for 99.9 % of

human exposure to PCP. Fisher also reports that in a study of pork and chicken in Canada, 60% of samples contained PCP. In a survey of cow milk in Ontario, 57 % of samples contained PCP at an average of 29 ppb. The exposure of dairy cattle to PCP was attributed to PCP contaminated wood shavings or saw dust used as litter. (Ryan et al, 1985; Frank et al., 1985). In a study of movement of PCP from treated wood posts that were in direct contact with soil, 95 % of the PCP remained in the wood posts after 2.5 months. PCP can persist in the soil from 14 days to 5 years depending on the soil microbes present (US EPA, 1978). Agriculture Canada has concluded the ñuse of PCP and other chlorophenols pose significant hazards to fish and fisheries in Canada, particularly British Columbia.ò A large majority of people with no obvious exposure have measurable residues (Fisher, 1991). Cheng et al., 2014 studied, ñcancer risks and long term community level exposure to PCP through drinking water.ò PCP was detected in 27 raw drinking water samples ranging from 11.21 to 684.00 ng/L. The authors conclude that PCP was probably associated with hemolymph neoplasm, neurologic tumors, and digestive system neoplasm. ò

Zheng, et al., 2013 ñconducted a systematic review to explore 2 kinds of associations, one was between the workers exposed to PCP and the said neoplasms, and the other was between the children with lymphoma and leukemia with their parents exposed to PCP. The authors found a significant association between lymphoma and workerôs occupational exposing to PCP and other studies suggested an increased risk of childhood leukemia because of their parental exposure to PCP at preconception.ò

EPA has declared this agent a carcinogen and IARC has declared it a possible carcinogen. The National Institute of Environmental Health Sciences has just recently declared this agent as reasonably anticipated to be a human carcinogen and it has been demonstrated that Ontario workers are by far the most exposed in all of Canada, followed by Quebec, Alberta and BC. More importantly, it is recommended that the authorities look to ban the use of this product entirely, and research a safer product/method and one that is more tolerable by the environment. The health impacts due to use and the human and environmental burden of this product are too toxic to ignore. More stringent controls at the worker level should be implemented with regards to training, personal protective equipment and work administration for workers who must work with this chemical or come in contact with it, and if use is going to persist, then the chemical should be placed on the designated substances lists so that the enforcement of engineering controls and personal protective equipment, biological monitoring is administered. Furthermore, in the 2011 Health Canada Re-evaluation decision, it is stated ñpentachlorophenol is acceptable for continued registration when used according to label directions.ò The practices are identified in the ñRecommendations for the Design and Operation of Wood Preservation Facilities Technical Recommendations Document (Environment Canada 2004) and were designed to minimize environmental and human health exposures. In reviewing several MSDSs, there is no clear indication that the warnings that are summarized in this re-evaluation are met by the MSDSs published by their suppliers, thus a failed communication to the end users.

If at this time a ban is not feasible, but is recommended, in the meantime, OHCOW recommends that the TWA of 0.3 mg/m^3 inhalable fraction and vapor (Demidenko at al., 1969) be adopted as it has been shown to cause irritant effects to the respiratory system above this level and the STEL should be 0.5 mg/m^3 inhalable fraction and vapor to minimize upper respiratory tract and eye irritation reported in workers exposed to greater than 1 mg/m^3 (Deichmann and Keplinger, 1981).

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Peracetic Acid

Peracetic acid is becoming an important chemical in healthcare infection control. With the rise of concerns over *C. difficile* and the problems associated with the use of chlorine (especially in environments where quaternary ammonium chlorides are also used), peracetic acid is being espoused as an alternative to disinfection with bleach solutions (references ï British paper about PA and PIDAC). Peracetic acid is also being considered as an alternative disinfectant for haemodialyzers (CDC reference, Bond et al 2011).

The proposed establishment of a new peracetic acid OEL for Ontario is based on the new ACGIH adoption of a STEL-TLV for peracetic acid of 0.4 ppm (1.24 mg/m^3), as measured as inhalable fraction and vapour, and classified as an A4 category carcinogen (ñNot Classifiable as a Human Carcinogenò). The ACGIH documentation seems to imply that the derivation for this STEL-TLV is based on the Gagnaire et al. (2002) paper which identifies a 50% respiratory depression rate (RD50) of 3.8-5.4 ppm for a peracetic acid mixture (similar to what is commonly found as a disinfectant in healthcare and food handling workplaces) and pure peracetic acid respectively. Since the recommendation (Nielsen, et al. (2007)) is to multiply the RD50 by 0.1 to derive a STEL (multiply by 0.03 to derive a TWA), the recommend STEL would be 0.038-0.054 ppm or 0.4 ppm (1.24 mg/m^3). Interestingly, the documentation cites a pair of unpublished studies (McDonagh (1997); and; Fraser and Thorbinson (1986)) involving worker exposures to corroborate this OEL based on animal data.

Generally, the practice in risk assessment is to give priority to human studies if they are of sufficient quality. This is what the NRCôs Committee on Acute Exposure Guideline Levels (AEGL) did when they considered the same studies and derived their AEGL-1 of 0.17 ppm (0.52 mg/m^3) for 10 minutes to 8 hours (the rationale behind the same level for short term exposures (i.e. 10 minutes) and full shift exposures (i.e. 8 hours) is that: ñ(1) effects of peracetic acid exposure correlate with concentration more than time, and (2) and peracetic acid is freely soluble in water; therefore, it should be effectively scrubbed in the nasal passagesò, (page 329). In the documentation the NRCôs Committee cited that: ñMcDonagh (1997) recommended 0.15 ppm (0.47 mg/m^3) as an acceptable 8-h occupational exposure limit for peracetic acid. This concentration would be perceptible, but not irritating or unpleasant.ò (page 332). The NRC Committee also discussed the Fraser and Thorbinson (1986) study of fogging in a chicken house. They cite the authors as reporting that ñthe physiological responses decreased from extreme discomfort of mucous membranes to mild discomfort at 0.5-1.0 ppm (1.56-3.12 mg/m^3) to no discomfort at Ò0.5 ppm (1.56 mg/m^3).ò (page 333).

The NRC defines the AEGL-1 as follows: ñAEGL-1 is the airborne concentration (expressed as parts per million or milligrams per cubic meter [ppm or mg/m3]) of a substance

above which it is predicted that the general population, including susceptible individuals, could experience notable discomfort, irritation, or certain asymptomatic, non-sensory effects. However, the effects are not disabling and are transient and reversible upon cessation of exposure.ò (http://www.epa.gov/oppt/aegl/pubs/define.htm accessed Sept 23/14).

Recently NIOSH (2015) has proposed an IDLH of 1.7 mg/m^3 (0.64 ppm) which is only slightly more than 3 times the proposed OEL. Furthermore in a recent report Hawley et al. (2016) noted ñcommonly reported health outcomes were watery eyes (46%), nasal problems (41%), asthma-like symptoms (28%), use of allergy medicine (16%), and shortness of breath (16%)ò, among exposed healthcare workers at concentrations of 6-511 ppb for hydrogen peroxide, 7-530 ppb for acetic acid and 1-48 ppb (0.001-0.048 ppm) for peracetic acid.

Based on these considerations, OHCOW would recommend that the OEL-STEL be lowered to at least 0.2 ppm to be consistent with the NRCôs interpretation of the studies which form the basis of the TLV and perhaps even lower given the experiences reported recently in the MMWR (Hawley et al., 2016). We would agree with sampling both the vapour and the inhalable fraction. While we agree with the A4 carcinogen designation, it is important to note that there is some concern that the commonly used peracetic acid/hydrogen peroxide/acetic acid mixture is a potent tumour promoter.

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Triethylamine & Dimethylamine

The change in the ACGIH TLV for dimethylamine (DMA) is simply the adding of the ñDSENò notation to the current OEL of 5 ppm (TWA) and 15 ppm (STEL) and thus it was not included in the MOL proposals even though it was listed as a change for the 2014 TLVôs. However, when we reviewed the ACGIH documentation we noticed a number of discrepancies:

1. One of the two studies cited (CIIT, 1990) as the basis of the TLV, was referenced as providing a NOEL (no observed effect level) of 10 ppm, however, there was a mild effect observed and therefore this should have been called a LOEL (lowest observed effect level) as per the interpretations of the documentations.
2. The ACGIH applied an uncertainty factor of 2 to the CIIT ñNOELò, whereas the other documentations use the same study and apply a 5-10 fold uncertainty factor on the basis of the CIIT ñLOELò
3. The second study referred to (Coon et al., 1970) also showed effects at an even lower level (5 ppm) and again this was interpreted both by the ACGIH and Coon et al., as a NOEL whereas other documentation recognize this appropriately as a LOEL.

4. There were errors in referencing outdated OELs for analogous amines (the documentation assumes erroneously that the TLVôs for methylamine and ethylamine are 10 ppm); furthermore, these are both primary amines, not secondary amines ï should tertiary amines (e.g. triethylamine) also be cited?

In contrast to the ACGIH 2014 documentation, the SCOEL 1991 documentation using the same CIIT, 1990 study derives a 2 ppm TWA OEL (STEL = 5 ppm) using an uncertainty factor of 5 based on the absence of human data and based on the absence of a NOAEL (they refer to the CIIT, 1990, 10 ppm observations as an LOEL).

Similarly, despite many of the agenciesô documentations concurring with the ACGIHôs interpretation of the Coon et al., 1970 study as being an NOEL, the AEGL 2008 documentation questions the authorsô (Coon et al., 1970) conclusion that: ñspecific chemically induced histopathological changes were not noted.ò. We would concur with this perspective that the Coon et al., 1970 study establishes an LOAEL and should not be considered an NOAEL. Using the IRIS methodology of deriving an RfC and converting it to an OEL, based on an LOAEL of 5-10 ppm (CIIT, 1990, and, Coon et al., 1970) an equivalent OEL of 0.1-0.2 ppm (without uncertainty factors applied) can be derived. If one were to apply the uncertainty factors applied by the SCOEL then the OEL adjusted for a 5 fold uncertainty factor would be: 0.02-0.04 ppm.

The German MAK 1993 documentation follows the SCOEL 1991 lead in using the CIIT 1990 study to establish a 2 ppm MAK (essentially concurring with the SCOEL uncertainty factor of 5) but they note that the OEL ñrequires substantiation from experience of human exposuresò. In a similar vein, the ACGIH 2014 documentation states ñIt should be noted that at the TLV concentration, the odor of dimethylamine may be sufficiently unpleasant to make working under those conditions not possible.ò Furthermore, the German MAK 1993 documentation cite a study by Sedov et al., 1980, which describes a set of human exposure experiments in the former USSR which establish a human NOEL of 0.5 ppm (which the 1986 ACGIH Documentation cites as the OEL for dimethylamine in the USSR). However, the German documentation discounts this finding because of poor documentation.

Given the findings of Sedov et al., 1980 and the ACGIH 2014 documentation note that working under TLV concentrations is ñnot possibleò, we would recommend that the OEL be lowered from 5 ppm to at least 0.5 ppm. Furthermore, using the IRIS RfC methodology (but using the SCOEL uncertainty factor of 5), one would derive an equivalent occupational OEL of 0.02-0.04 ppm. We agree with the addition of the ñDSENò notation.

The key acute health effect associated with dimethylamine exposure is glaucopsia (Kang, 2016), which is thought to be caused by a swelling of the cells on the surface of the eye which causes ñhaloò, or ñfoggyò vision disturbances.

Given that triethylamine (TEA) causes similar effects as dimethylamine, we find it quite interesting that the ACGIH has proposed to lower the TEA TLV from 1 ppm (TWA) to 0.5 ppm (TWA). The basis for this change is the work done by Järvinen et al. (1999), where 4 volunteers who normally worked with TEA were exposed in a laboratory setting for 4 hours to 0.72, 1.56, and 9.74 ppm of TEA. They reported no observed adverse effect (NOAE) at 4 hrs of 0.72 ppm (equivalent to 0.36 ppm 8-hr TWA) and observed ñblurred vision and a decrease in contrast sensitivityò at 4 hours of 1.56 ppm exposure (equivalent to 0.78 ppm 8-hr TWA). Based on this study the ACGIH set the 8 hour exposure limit at 0.5 ppm and the 15 minute short term exposure limit at 1 ppm. While this is very close to the NOAEL published by Järvinen et al. (1999), it should be noted that these observation are based on only 4 workers observed for only 4 hours on

a single occasion. To generalize for all workers based on an 8-hr TWA equivalent of 0.36 ppm for no effect and an 8-hr TWA equivalent of 0.78 ppm for an effect does not allow for much variation between workers (normal factor for human variation is somewhere between 3-10 fold). Thus, based on such a degree of variation between workers in their response to TEA (and/or dimethylamine) it would not be inconceivable to expect to find some workers who might well experience glaucopsia at exposure concentrations below the proposed OEL.

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Trichloroacetic Acid

TCA is used as a soil sterilizer and a laboratory intermediate or reagent in the synthesis of a variety of medicinal products and organic chemicals (HSDB, 2007). Medical applications of TCA include use as a reagent for the detection of albumin (Lewis, 1997), application as an antiseptic (Morris and Bost, 2002), and use as a skin peeling agent (Lee et al., 2002; Coleman, 2001). TCA is also used industrially as an etching and pickling agent for the surface treatment of metals and (in solution) as a solvent in the plastics industry ñTrichloroacetic acid is used therapeutically where removal of tissue by exfoliation or tissue destruction is desired. ACGIH 2014ò It is also found as a by-product during the chlorination of water, and thus may occur in swimming pools, (IARC, 2004).

IARC Monographs 106 has now declared Trichloroacetic acid as possibly carcinogenic to humans, Group 2B. EPA has declared trichloracetic acid as a possible human carcinogen based on carcinogenicity data in experimental animals. (IARC, 2014).The European Commission does not establish an OEL for this agent as they state there is limited animal data from which a NOAEL can be derived. So no OEL is made available by this organization.

OHCOW recommends adopting the ACGIH TLV for trichloracetic acid, to be 0.5 ppm versus the current OEL of 1 ppm. The TLV-TWA of 0.5 ppm ñshould be sufficient to protect

against the unwanted effects of TCA, which are tissue irritation and damage to points of contact ACGIHò The liver and kidney are the major target organs for trichloracetic acid exposure, as per the data from animal studies (IARC, 2014).

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1,2,3-Trichloropropane

CAS # 96-18-4, synonyms, allyl trichloride, glycerol trichlorohydrin, trichlorohydrin As per the Environmental Protection Agency, 1,2,3 Trichloropropane has been used as an industrial solvent and as a cleaning and degreasing agent, amongst several other uses such as chemical intermediate in the production of other chemicals (DHHS, 2011).

The ACGIH 2015 documentation indicated that the level of 0.005ppm for an 8 hr exposure should provide adequate protection from the effects seen in animal studies conducted. Taken directly from the documentation:

> ñRepeated exposure studies in rats have found the first sign of response to the agent to be irritation to mucous membranes with lacrimal discharge see in rats exposed to 0.5 ppm for 13 weeks. 1,2,3-trichloropropane is carcinogenic in rats and mice, producing a wide range of tumors via oral administration. The lowest does tested in these studies, 3mg/kg in rats and 6 mg/kg in mice produced significant number of forestomach tumors, suggested that the no effect level might be considerably lower than those used in the experiment. Short Term exposure may cause eye and throat irritation and long term exposure has led to liver and kidney damage and reduced body weight in animal studies, as per EPA. Exposure occurs through vapour inhalation, dermal exposure or ingestion (ATSDR 1995; DHHS 2011). US Department of Health and Human Services states that TCP is ñreasonable anticipated to be a human carcinogen based on sufficient evidence of carcinogenicity form studies in experimental animalsò (DHHS, 2011).ò

International Values:

IARC has classified this agent as Group 2A probably carcinogenic to humans. As it 1,2,3- Trichloropropane causes tumours at multiple sites and at high incidence in mice and rats. (ii) The metabolism of 1,2,3-trichloropropane is qualitatively similar in human and rodent microsomes. (iii) 1,2,3- Trichloropropane is mutagenic to bacteria and to cultured mammalian cells and binds to DNA of animals treated in vivo. (Taken from IARC).

EPA has classified TCP as ñLikely to be carcinogenic to humans.ò

The Institute for Occupational Safety and Health of the German Social Accident Insurance (IFA)ôs GESTIS Database lists 1,2,3, Trichloropropane and the most protective country with regards to 8 hour limit values are The Netherlands, at 0.00108 mg/m^3. The 2012 MAK value was not given because of the cancer risk to man, since a safe concentration range cannot be given. Furthermore a notation of H, danger of percutaneous is also assigned. The European SCOEL value 2011, for the 8 hr TWA is not assigned, a skin notation is assigned, and the SCOEL carcinogen group is A (Genotoxic carcinogen with no threshold). Their recommendation is that occupational contact should be avoided.
Similarly, the 1992 ATSDR Documentation also concludes the following taken directly from Section 2.3.1.3 of the report:

> ñ No quantitative information was located regarding absorption of 1,2,3-Trichloropropane in humans or animals following dermal exposure. However, internal pathology and death have been reported in animals exposure by the dermal route. Since vapor exposure was unlikely due to the occlusive covering of the treatment area, it can be concluded that dermal absorption occurs to some extent.ò Thus it is recommended to keep the SKIN notation as it was. Furthermore, as per section 2.2.2 of the SCOEL Documentation for this agent, it was reported that, ñthe LD50 for rats after dermal exposure and oral administration was observed indicating that it is absorbed by these routes. (DFG, 1997).ò

The Polish group, GECA, Group of Experts for Chemical Agents, studies and assigned skin notation to 13 agents, one of which was 1,2,3 Trichloropropane. Although their TWA is at 14 mg/m^3, they did assign a skin notation due to their findings of systemic effects of dermal exposure in rabbits at 250 mg/kg/rabbit.

IARC Monographs 106 has now declared Trichloroacetic acid as possibly carcinogenic to humans, Group 2B. EPA has declared trichloracetic acid as a possible human carcinogen based on carcinogenicity data in experimental animals. (IARC, 2014).The European Commission does not establish an OEL for this agent as they state there is limited animal data from which a NOAEL can be derived. So no OEL is made available by this organization.

OHCOW recommends adopting the ACGIH TLV for 1,2,3 Trichloropropane, to be 0.005 ppm versus the current OEL of 10 ppm. OHCOW recommends keeping the skin notation as per DFG, 1997. This agent is assigned an A2 classification, Suspected Human Carcinogen and exposure should be avoided.

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2016 Policy Issues:

Proposed New Respiratory Protection Requirements

The following comments are excerpted from OHCOWôs submission on O.Reg 490/09: While we find it rather unusual to be commenting on a conceptual presentation of the code rather than the exact proposed wording, in general we agree with the concept of following the CAN/CSA-Z94.4-11 - Selection, use, and care of respirators standard. We are pleased to note that in our submission on O.Reg. 490/09 we noted that ñit would be extremely helpful if this proposal were to be expanded to include the substances referenced in O.Reg 833 and also apply to occupational exposures to pesticides.ò Thus we are pleased to see that the MOL is proceeding along these lines.

Based on our own experience of characterizing a worker with leukemia whose exposure to benzene consisted mainly of the absorption of benzene through the skin from solvents which had benzene concentrations below the WHMIS reportable concentration (0.1%), we would suggest that dermal protective equipment should also be addressed.

Proposed New Air Sampling Requirements

Again, the following comments are excerpted from OHCOWôs submission on O.Reg 490/09: We believe, in general, it is wise to reference widely accepted methods that the field of occupational hygiene recognizes rather than specifying methods for the collection and analysis of airborne substances. There is a concern regarding the lack of validity of the sampling strategies used to collect exposure data for the purpose of individual exposure characterization. As with the methods of collection and analysis of airborne designated substances, the field of occupational hygiene has developed standards of practice with respect to sampling strategies. These are mentioned in the sections above titled ñA Lack of a Legal Requirement to Measure Exposuresò and ñA Lack of a Legal Requirement to Employ Unbiased Sampling Strategiesò. Thus, we would recommend that the Ministry of Labour not only address the analytical methods of exposure assessment using air sampling, but also address necessity to perform sampling and to use a valid sampling strategy.

Proposed New Equivalency Clause

While in theory and on the face of it, an ñequivalency clauseò with JHSC consultation seems very reasonable, in practice we are concerned that the JHSC may not have the familiarity and/or access to expertise to judge claims of equivalency. We would recommend that any claim of equivalency should be supported with the appropriate technical evidence and that someone with minimum specified qualifications (i.e. a certified occupational hygienist) take responsibility for the claim of equivalency. Furthermore, it may make things much easier for Ontario workplaces if the Ministry would publish a list of recognized equivalencies.

Proposed Changes to Medical Examinations in O. Reg. 278/05

Again, the following comments are excerpted from OHCOWôs submission on O.Reg 490/09 in relation to the review conducted by St. Michaelôs Hospital Occupational & Environmental Health Program:

In 2012, the ACCP with a number of other organizations published a guideline titled: ñThe role of CT screening for Lung Cancer in clinical practice - The evidence based practice guideline of the American College of Chest Physicians and the American Society of Clinical Oncologyò (supplementary material to Bach et al. (2012), ñBenefits and Harms of CT Screening for Lung Cancer - A Systematic Reviewò, JAMA. 2012;307(22):2418-2429). This guideline reverses the previous position which Holness et al (2010) reference as justification for not including low-dose CT lung cancer screening. In the US the CDC has produced a list of organizations with their positions on lung cancer screening (http://www.cdc.gov/cancer/lung/pdf/guidelines.pdf). In Ontario, Cancer Care Ontario has published a guideline recommending CT screening for high risk populations (Roberts H, Walker-Dilks C, Sivjee K, Ung Y, Yasufuku K, Hey A, et al. Screening high-risk populations for lung cancer. Toronto (ON): Cancer Care Ontario; 2013 April 18. Program in Evidence-based Care Evidence-based Series No.: 15-10.). Thus, it would seem appropriate to reconsider this recommendation in light of the newer recommendations.

OHCOW has considered the guideline produced by the National Comprehensive Cancer Network (NCCN), ñClinical Practice Guideline in Oncology for Lung Cancer Screeningò (Version 1.2016), because it includes occupational exposures to lung carcinogens as a risk factor included in the criteria. The organization has also developed a guide for patients to help them understand the risks and benefits associated with such screening (http://www.nccn.org/patients/guidelines/lung_screening/files/assets/common/downloads/files/lung_screening.pdf). This is particularly relevant to the Sarnia OHCOW Clinic, since it has been collaborating with Princess Margaret Hospital/University Health Network in Toronto on a screening study for asbestos-exposed workers since 2005. Thus we would endorse the comment made by Holness et al (2010):

> ñA mechanism to provide for the evolving evidence with respect to both health effects and screening tests would be advantageous so program recommendations could be modified as new evidence is found.ò (page 5)

Additional Substances Recommended for Improved OEL's:

Nanoparticles and Carbon Nanotubes

NIOSH has recently recommended[1] an REL for carbon nanotubes (CNT) and nanofibers (CNF) of 1 ε g/m^3, 8-hr TWA, for a 45 year working life. Included in the recommendations were provisions for measuring CNT/CNFôs and medical surveillance/screening. Schulte et al. (2014)[2] have also recommended some general guidelines which would apply to all nanoparticles invoking the precautionary principle. Given the fast pace at which nanoparticles are being disseminated in a diverse range of products, and given the poor knowledge base which exists about the health effects of these materials, OHCOW recommends that until evidence is provided to the contrary, these particles be treated with the highest degree of exposure control. An analogous situation would be the MOLôs treatment of polymeric isocyanates when it was established that there were no valid methods of measuring airborne polymeric isocyanates ï in response to this situation, the MOL mandated maximum PPE (full face-piece positive air supply) if polymer isocyanates were used in the workplace[3 c.f. p.28-29]. OHCOW recommends that an exposure registry be established for workers exposed to nanoparticles and that at minimum passive medical surveillance be established and where early research finding warrant, active medical surveillance be practiced. An exposure registry should also require employers to monitor exposures. By adopting the hierarchy of OELôs the MOL may be able to mandate exposure limits as they emerge by recommended practice under 25(2)(h) in the Act and section 3(1) of O. Reg. 833. Given the rationale provided by NIOSH for the REL for CNT/CNFôs, OHCOW recommends that the MOL adopt a 1 ε g/m^3 for these substances.

1. NIOSH, ñCurrent Intelligence Bulletin 65 - Occupational Exposure to Carbon Nanotubes and Nanofibersò, US Department of Health and Human Services, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, DHHS (NIOSH), Cincinnati (2013).
2. Schulte, P.A., C.L. Geraci, V. Murashov, et al., ñOccupational safety and health criteria for responsible development of nanotechnologyò, Journal of Nanoparticle Research 16:2153:p.1-17 (2014).
3. Ontario Ministry of Labour, Occupational Health and Safety Division, Designated Substances in the Workplace: A Guide to the Isocyanates Regulation, Queenôs Printer for Ontario, Toronto, 1987.

Asbestos

The recent developments in the cancer risk assessment of asbestos exposure have highlighted the fact that the risks associated with asbestos exposure (particularly amphiboles) are higher than the previous estimates on which the current TLV and OEL are based. Offermans et al (2014)[1] published a prospective cohort study (prospective cohort study designs are the strongest observation design), which showed statistically elevated hazard ratios for both mesothelioma and lung cancer associated with a cumulative estimated exposure (based on the Finnish Job Exposure Matrix (FINJEM)) of 0.20 f-yr/mL (median exposure estimate ï hazard ratios of 2.69 and 1.47 respectively). Assuming a 40 yr working life, this cumulative exposure level would translate into an average exposure level of 0.005 f/mL. This level is 20 times lower than the current asbestos OEL of 0.1 f/mL. Accordingly, OHCOW recommends that the asbestos OEL be

lowered to 0.005 f/mL. The Netherlands has an OEL of 0.002 f/cc (8-hr TWA) for chrysotile asbestos and 0.00042 f/cc for amphibole asbestos fibres. This has been in effect since Jan 1, 2014 (https://www.ser.nl/en/grenswaarden/asbest.aspx).

1. Offermans, N.S.M., R. Vermeulen, A. Burdorf, et al., ñOccupational Asbestos Exposure and Risk of Pleural Mesothelioma, Lung Cancer, and Laryngeal Cancer in the Prospective Netherlands Cohort Studyò, Journal of Occupational and Environmental Medicine 56:6-19 (2014)

Styrene

In 1996 the ACGIH adopted a TLV of 20 ppm (TWA) and 40 ppm STEL for styrene. The current Ontario OEL is 35 ppm (8-hr TWA) and 100 ppm (STEL). A relatively recent review from the Swedish Criteria Group for Occupational Standards came to the following conclusions regarding the effects of exposure to styrene:

> ñThe critical effects of occupational exposure to styrene are genotoxicity, hearing loss and effects on color vision. Styrene is probably genotoxic to humans and possibly also carcinogenic. Genotoxic effects have been observed at occupational exposures down to about 10 ppm. Effects on color perception have also been documented at occupational exposures around 10 ppm, and hearing loss is presumed to occur at approximately the same levels.ò (p.78)

Accordingly, OHCOW recommends the MOL adopt an OEL of at most 10 ppm for styrene exposure.

1. *ACGIH®,* Formaldehyde: TLV® Chemical Substances 7th Edition *Documentation* Publication (2014).
2. Swedish Criteria Group for Occupational Standards, ñScientific Basis for Swedish Occupational Standards XXXò Arbete och Hälsa nr 2010;44(5) p.44-88

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/pubs/gl_heat.php).

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 its intent to implement this much reduced OEL for respirable PNOCôs, 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 εg/m^3 (i.e. 0.121 mg/m^3) and suggested the threshold for this health effect was at a level of 60 εg/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 ñnuisanceò 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.

In 2013 a group of leaders in Occupational Hygiene research published a commentary[10] in the Annals of Occupational Hygiene recommending the OEL for PNOCôs be lowered, stating:

> ñé there is good evidence from epidemiology and toxicology studies that current dust exposures may still present a risk to workers and that for some of those who are affected, there are devastating health consequences.ò (p.685)

They recommend that occupational hygienists use an OEL of at most 1.0 mg/m^3 PNOC (respirable) until governments respond to this situation.

Furthermore, in 2012 Health Canada published a ñGuidance for Fine Particulate Matter ($PM_{2.5}$) in Residential Indoor Airò (http://www.hc-sc.gc.ca/ewh-semt/pubs/air/particul-eng.php) which indicated there was no threshold for health effects associated with particulate matter:

> ñIndoor levels of $PM_{2.5}$ should be kept as low as possible, as there is no apparent threshold for the health effects of $PM_{2.5}$.
> é any reduction in $PM_{2.5}$ would be expected to result in health benefits, especially for sensitive individuals, é ò

Given the evidence highlighted, OHCOW would recommend the Ministry of Labour should lower the PNOC respirable dust OEL to at least 1.0 mg/m^3 and preferably 0.4 mg/m^3 for the protection of the health of Ontario workers.

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2. Mermelstein, R. and R.W. Kilpper, ñRecent Chronic Inhalation Study Results and Their Implications on the Respirable Dust Standardò, Chapter 6 in 3rd Symposium on Respirable Dust in the Mineral Industries, edited by R.L. Frantz and R.V. Ramani, Society for Mining Metallurgy and Exploration, Littleton, Colorado (1991) pp.51-60.
3. Morrow, P.E., H. Muhle and R. Mermelstein, ñChronic Inhalation Study Findings as a Basis for Proposing a New Occupational Dust Exposure Limitò, Journal of the American College of Toxicology 10: 279-290 (1991).

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7. Daniels, M.J., F. Dominici, J.M. Samet and S.L. Zeger, ñEstimating Particulate Matter-Mortality Dose-Response Curves and Threshold Levels: An Analysis of Daily Time-Series for the 20 Largest US Citiesò, American Journal of Epidemiology 152:397-406 (2000).
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10. Cherrie, J.W., L.M. Brosseau, A. Hay, and K. Donaldson, ñLow-Toxicity Dusts: Current Exposure Guidelines Are Not Sufficiently Protectiveò, Annals Occupational Hygiene 57:685ï 691 (2013).

Metalworking Fluids

Metalworking fluids (MWF) were not on the list for updating, however, OHCOWôs 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(7) has shown similar comparisons with respect to statistically significantly elevated respiratory symptoms at total MWF aerosol concentrations of 0.1-0.2 mg/m^3. 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 UNIFOR that all new metalworking process 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 (although it appears that this OEL excludes MWFôs), 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 previously proposed (but never adopted) ACGIH TLV of 0.2 mg/m^3 for mineral oil in metalworking fluids, if not at least the NIOSH (GM/UNIFOR) OEL of 0.5 mg/m^3.

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Diesel Exhaust

The fact that IARC now recognizes diesel exhaust as a proven human carcinogen, underlines the importance of creating an appropriately protective diesel exhaust OEL.

The ACGIH[(1)] in 2002 proposed a TLV (TWAEV) of 20 εg/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 εm particle size measured by elemental carbon) would be up to 1-3 εg/m^3[(4)]. Thus, NIOSH effectively recommends a level below 1 εg/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 εg/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 εg/m^3 of DPM (roughly equivalent to 3.1-6.6 εg/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, VOCôs, etc.). The EPA[4] states ñEffects of DE exposure could be additive to or synergistic with concurrent exposures to many other air pollutants. é (e.g., potentiation of allergenicity effects, potentiation of DPM toxicity by ambient ozone and oxides of nitrogen)ò (page 1-7).

In the past year or two, a number of new papers[7,8] have been released particularly dealing with the lung cancer risks associated with exposure to diesel exhaust. At a recent conference Bob Park from NIOSH reviewed[9] the risk estimates associated with a working lifetime exposure to diesel exhaust and the risks of developing lung cancer. The range of lifetime equivalent concentrations to diesel exhaust associated with a risk of 1/1000 (maximum occupational risk benchmark) was 0.32-0.94 ε g/m^3 (respirable elemental carbon).

The Occupational Cancer Research Centre has recently presented[10] the results of study calculating the impact of diesel exposure on Canadian cancer rates:

> ñApproximately 1.4 million workers were exposed to DEE during the risk exposure period. The initial estimated AFs for DEE-related lung cancers are: 4.92% for males, 0.29% for females, and 2.70% overall.ò *(Reference 10, page A37; AF = attributable fraction, DEE = diesel engine exhaust)*

Given the ubiquitous exposure to diesel exhaust among Ontario workers, and given the MOL strategic plan to reduce occupational disease, it behooves the MOL to establish an OEL for diesel exhaust OEL below 10 ε g EC/m^3 in order to prevent lung cancer among exposure Ontario workers.

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Stoddard Solvent

The Ontario TWA OEL for Stoddard Solvent is 100 ppm, however in Europe, the Scientific Committee on Occupational Exposure Limits (SCOEL) has adopted an OEL of 20 ppm since 2007 (see: http://ec.europa.eu/social/BlobServlet?docId=3859&langId=en). This exposure limit was based on the experience of painters who were exposed to an average of 40 ppm. At this level of exposure workers experienced acute symptoms (nausea, irritation, vertigo and an impaired sense of smell), and had impaired results in reaction time and memory tests. The SCOEL also recommended a short-term exposure limit (STEL) of no more than 50 ppm for any 15 minute period of time during the work day (while also maintaining the full shift 8-hr TWA of 20 ppm).

A 2-year National Toxicology Program (NTP) animal study (2004 ï see: http://ntp.niehs.nih.gov/ntp/htdocs/lt_rpts/tr519.pdf) found that there was ñ*some evidence*ò that Stoddard Solvent (CAS # 64742-88-7) caused a very specific type of adrenal gland tumour only in male rats and, also, there was ñequivocal evidenceò of excess tumours in the livers of female mice. Male mice and female rats did not show any excess tumours. The interpretation of this study has been hotly debated in the scientific literature ï industry connected researchers and the US EPA saying that the mechanism of the kidney damage in male rats has no relevance to humans, while others questioning this claim because of the lack of evidence to support the hypothesized mechanism. There are case-control studies (Brautbar, 2004) indicating that long term solvent exposure can cause kidney problems in workers exposed.

Based on this evidence, OHCOW recommends that the OEL for Stoddard Solvent be reduced to no more than 20 ppm (TWA) and 50 ppm (STEL).

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