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, TRAINING AND SKILLS DEVELOPMENT

REGARDING:

2021 OCCUPATIONAL HEALTH CONSULTATION

JULY 9, 2021

Executive Summary

We thank the Ontario Ministry of Labour, Training, Skills and Development (MLTSD) for the opportunity to comment on the proposed changes to the Occupational Exposure Limits (TWAs) and associated policy proposals. We also appreciate that the MLTSD 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 TWA update process is a significant opportunity to prevent future occupational disease.

The following table summarizes OHCOW's recommendations concerning the 2021 TWA update proposals:

Substance	Current TWA	Proposed TWA	OHCOW Endorsed	OHCOW Recommendation	Rationale
Respirable crystalline silica (RCS)	TWA: 0.1 mg/m^3	TWA: 0.025 mg/m^3	Yes	*OHCOW recommends lowering it further: TWA 0.010 mg/m^3*	Reduce the occupational burden of chronic obstructive pulmonary disease (COPD), silicosis, and lung cancer. *Citation: CAREX Canada*
Hydrogen sulfide [7783-06-4]	TWA: 10 ppm STEL: 15 ppm	TWA: 1 ppm STEL: 5 ppm	Yes: TWA is endorsed No: STEL is not endorsed	*Ceiling (C) 5 ppm*	OHCOW recommends a ceiling limit of 5 ppm. If there was no STEL, Reg. 833. Sec. 4.3.ii would apply: a TWA without a STEL or C shall never exceed 5 x TWA at any time. A C of 5 ppm would align with the current legislation. This recommendation is made due to the GHS Hazard Code: H330 acute toxicity (fatal if inhaled). *Citation: ECHA*
4,4'–Methylene bis (2-chloroaniline) (MBOCA; MOCA®) [101-14-4]	TWA: 0.0005 ppm = 0.005 mg/m^3 Skin notation	TWA: 0.01 ppm (IFV) Skin notation	No: Raising the TWA of a carcinogen is not endorsed	*OHCOW recommends maintaining the existing TWA of 0.0005 ppm = 0.005 mg/m^3 Skin notation*	Exposure to carcinogens should not be raised without excellent evidence, and should be as low as reasonably achievable (ALARA). MOCA is an IARC Group 1 carcinogen, with GHS Hazard Code: H350 may cause cancer. *Citations: IARC, ECHA*

We would also like to reiterate previous TWA update proposals which were not adopted in the past in 2004, 2009, 2010, 2013, 2014, 2015, and 2018.

OHCOW strongly recommends that Ontario needs to move forward and adopt the ACGIH notations for chemicals, RSEN and DSEN, as well as carcinogen (ACGIH notations). Furthermore, in response to the invitation to nominate additional matters for consideration, we include recommendations regarding exposures to agents not on the list.

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

Outline:

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 7 clinics in Ontario, a team of nurses, occupational hygienists, ergonomists, and physicians see Ontario workers to 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 (JHSCs), 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 (TWAs) play in prevention (or the lack of prevention when illnesses occur even when exposures comply with the TWA).

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 TLVs. However, a different challenge has been launched against the TLVs subsequently which also threatens to affect the manner in which they are set. A number of lawsuits were launched against the TLVs from both industry and industrial disease victims.

These legal challenges have had a “chilling” effect on the organization and seem to have introduced hesitancy in reacting to situations where there is limited evidence. The ACGIH has withdrawn 39 TLVs since 2005 for a variety of reasons (“insufficient data”, 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. Stouten et al.[31], in 2008 published a review of the Netherlands’ TWA and came to the conclusion that the toxicological evidence for about 40% their TWA (which were largely based on the ACGIH TLVs) did not meet DECOS’ criteria for a health-based TWA. The concluded

that "Many older MAC values were either too high or not scientifically supported and therefore not health-based" (*page 407*).

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 TWAs. Some reliable sources which we continue to consult include the EU SCTWAs, 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 TWA, or else workers experience symptoms due to exposure despite the personal exposure concentrations being below an established TWA. 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 TWA has been eliminated, no TWA has been established, or, the TWA 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 TWAs 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 TWAs will not necessarily lead to reductions in exposure in Ontario workplaces. In order for an TWA to effectively lower workplace exposures, measurements must take place in workplaces particularly where exposures may exceed the TWA. 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 TWAs are to impact Ontario workplaces. Without such a legal requirement, employers fearing being found to be 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 quantitative risk assessment related to chemical exposures which may require exposure measurements. The MLTSD publication titled "Designated Substance in the Workplace: A Guide to the Regulations" 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 "Assessment and Control of Exposure to Regulated Substances" and the accompanying "Code of Practice for Air Sampling and Analysis" (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 air 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. "they should have sampled when …"). In fact it has been shown[6] that mathematically modeling exposures[7] is more accurate than a sampling campaign that covers three or fewer workdays (most sampling campaigns cover only a single day). The Joint Steering Committee on Hazardous Substance Regulations (JSC, 1987-1995) recognized this situation and brought forward a draft code of practice attached to a draft regulation on exposure assessment strategies ("Code of Practice for Air Sampling and Analysis" (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 JSC's 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 then studies[20] in sampling strategy have offered 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 framing 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 TWA as a Means for Improving Workplace Conditions:

If changes in the TWAs 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 TWAs 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 TWAs 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 TWAs 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 TWAs in Preventing Occupational Disease:

Even though most workplaces are in compliance with current TWAs 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 TWAs. In fact, if one follows the history of TWAs, one will notice a gradual decline in most TWAs 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.

All the above 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 is interpreted as evidence supporting non-toxicity), we would adhere to the assumption of a chemical's 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 MLTSD-instituted policy which recognizes that just because exposure assessments demonstrate compliance is no reason to ignore workers' 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 TWAs 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 TWA (a certain percentage of exposed workers will go on to develop asthma in spite of maintaining exposures below the TWA). Carcinogens often do not have a threshold and thus TWAs are set at an "acceptable / tolerable" rate of occupational disease. The US the Supreme Court when considering the benzene TWA, determined that 1 worker death due to benzene exposure-related leukemia per 1000 exposed (i.e. 10^{-3}) could be considered a "significant" risk (i.e. worth the effort to avoid).

Due to this decision, for many years TWAs (including the ACGIH TLVs) 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]. In its Current Intelligence Bulletin 68 (Chemical Carcinogen Policy)[29] NIOSH stated it will set its Risk Management Limit for Carcinogens (RML-Ca) "… at the estimated 95% lower confidence limit on the concentration (e.g., dose) corresponding to 1 in 10,000 (10^{-4}) excess lifetime risk, when analytically possible to measure." (*page vi*). Taking all these limitations into consideration, it is very clear that compliance with TWAs 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 "have surpassed those for traumatic injuries and disorders"[22]. It should also be noted that most occupational cancers go unrecognized as being caused by occupational exposures. For the year 2011 the burden of lung cancer and mesothelioma due to occupational and para-occupational exposures in Canada was estimated to 427 cases of mesothelioma and 1904 lung cancer cases[30]. The authors estimated that 54% of mesothelioma costs and only 10% of lung cancer costs were covered by Canadian workers' compensation systems.

New Paradigms in Exposure Criteria:

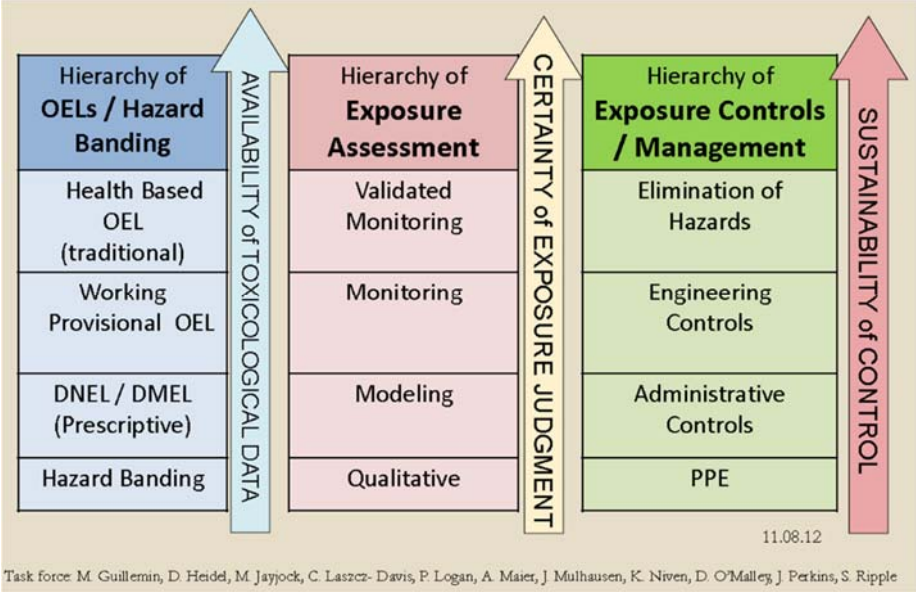
The dose-response relationship is more of a continuum than a straight line with a sudden discontinuity at the TWA. The heat stress TWA is graduated response as the WBGT rises. New paradigms in exposure assessment criteria have surpassed the single digit representation of the dose-response relationship which the TWA 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

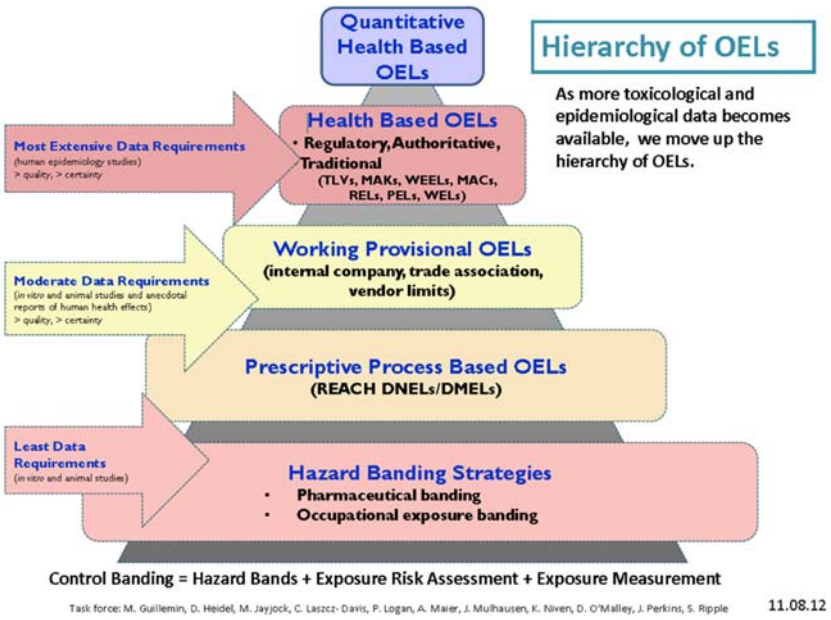
Similar graduated ranges have been established for volatile organic compounds (VOCs)[13], although the main goal of measuring VOCs is more to find the source and eliminate or control it to prevent exposure in the first place. Thus in the overall scheme of prevention, the single digit threshold concept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

New developments[24] have taken place in the field of occupational exposure limits. In addition to the traditional concept of a "hierarchy of controls", the concept of a "hierarchy of TWAs" and a "hierarchy of exposure assessment" have been put forward.



The hierarchy of TWAs 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 (DNELs) or Derived Minimal-Effect Levels (DMELs). While the ideals behind the legislation provided hope for the utility of the DNELs in the workplace, actual practice has been somewhat disappointing[25, 26]. European Safety Data Sheets (SDS) require that DNELs be included.

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



This framework for understanding TWAs is particularly useful for dealing with substances which do not have an TWA or for which the current TWA 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 (SMEs) 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 implementing exposure controls 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 "automate" the decision logic exposure assessors would use to categorize exposures and recommend controls. The MLTSD (formerly MOL) had a preliminary meeting with stakeholders a few years ago (Nov 23, 2001, chaired by John Vander DTWAen)

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 – as we have now adopted the GHS system in Ontario, perhaps the MLTSD 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. Adapting these tools to the Ontario context represents an untapped potential for exposure (and occupational disease) prevention especially for small and medium sized organizations. We would point to OHCOW's AirAssess smartphone application (https://www.ohcow.on.ca/airassess.html) as an example of new technology enabling any workplace to assess and diagnose indoor air quality concerns.

TWA Update Process:

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

In 2005 the MLTSD 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 MLTSD would recommend adoption of non-contentious proposed TWAs. If these timelines are too strict, then an amended timeline would be good to have rather than the current "*ad hoc*" situation. However, at one time, this was an annual process, which would have allowed the complete turnaround time within a year.

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 don't know what response the MLTSD has to the recommendations.

It would also be useful to have clear statements on the website as to the rationale the MLTSD uses when it decides not to update a particular TWA. Without directly talking to the TWA Update staff it is impossible to determine the status of the TWA proposals which the MLTSD has decided not to adopt (e.g. silica). If the MLTSD has reached such a conclusion, it serves the goals of transparency and accountability to publicly explain such decisions. Otherwise, those

parties who are interested will not know if a substance absent from the adopted list is "under further consideration" or has been dropped from the process.

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2021 TWAs

Respirable Crystalline Silica (RCS) (2021)

Based on the current evidence, OHCOW supports the MLTSD proposal to lower the TWA for Respirable Crystalline Silica (RCS) to be in line with the ACGIH[(1)]. However, after reviewing the evidence in the published peer-reviewed literature of occupational hygiene and occupational epidemiology, OHCOW has concluded that the proposed TWA of 0.025 mg/m^3 may be insufficient to protect Ontario workers. Based on the current evidence, OHCOW recommends Ontario adopt a TWA of 0.01 mg/m^3.

According to IARC (2018)[(IARC)], "silica dust crystalline in the form of quartz or cristobalite" is carcinogenic to humans (Group 1). Applying the NIOSH OEB and COSHH Essentials criteria for an acceptable exposure range that is health-based exposures should be maintained ≤ 0.01 mg/m^3. Notably, both NIOSH and COSHH Essential recommend carcinogen exposures be as low as reasonably achievable (ALARA).

One of the limitations for sampling and analysis of RCS with a low enough limit of quantitation (LOQ), to measure RCS below 0.025 mg/m^3 is the sampling conventional flow rate is typically close to 2 L/min (varies dependent on the type of cyclone sampler). Typically, an instrumental limit of quantification of 10 µg per sample is achievable by the use of either infrared (IR) or X-ray diffraction (XRD) analysis in laboratories. Studies have shown that samples collected with high flow rate samplers could provide precise analytical results, i.e. significantly above the LOQ by increasing the mass collection on filters[(Lee et al)]. *Thus there are now higher flow pumps available now in addition to sampling and analysis that allow higher flow rates with increased sensitivity, so that routine sampling would in fact be able to detect exposures below the proposed TWA of 0.01 mg/m^3.*

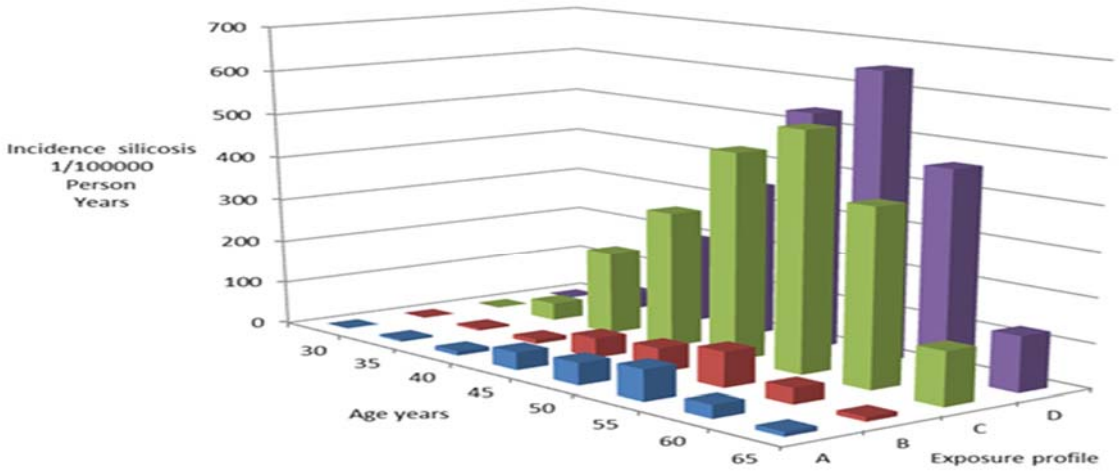
This proposal to lower RCS exposure is not new, the MLTSD has been proposing this change for nearly 20 years. In 2004 the MLTSD proposed lowering the RCS TWA (or TWAEL as it is called in the designated substances regulation at that time) to 0.05 mg/m^3, however it was not adopted. In 2006 (after the ACGIH lowered its TLV again), the MLTSD again listed silica in its annual TWA update but this time proposing to lower the TWAEL to 0.025 mg/m^3, but again, it has not been adopted.

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[(OSHA)]. The 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).

In 1997, a monograph published by IARC concluded that there is now sufficient evidence in humans confirming the carcinogenicity of inhaled crystalline silica in the form of quartz or cristobalite from occupational sources[(IARC)].

The National Toxicology Program (NTP) has labeled this agent a human carcinogen. It is now very clear from international studies that lung health of workers continues to be affected at relatively low RCS exposures well below the current Ontario TWA of 0.1 mg/m^3.

Nine studies reported by the IARC showed an excessive risk for lung cancer. Sun et al. (2011) in their study of 3250 Chinese pottery workers estimated the exposure response relationship between RCS and **the incidence of category 1/1 silicosis** for worker's long term exposure to relatively low concentrations of airborne silica dust known as RCS is known to lead to silicosis, a progressive fibrosis of the lungs. An increased risk of silicosis was demonstrated ≤ 0.05 mg/m^3.



Code	Exposure profile
A	Average ≤ 0.05 mg/m^3; Maximum annual average ≤ 0.05 mg/m^3
B	Average 0.05 – 0.1 mg/m^3; Maximum annual average ≤ 0.1 mg/m^3
C	Average 0.05 – 0.1 mg/m^3; Maximum annual average 0.1 – 0.5 mg/m^3
D	Average 0.1 – 0.15 mg/m^3; Maximum annual average 0.1 – 0.5 mg/m^3

Figure 1: Incidence of silicosis in Chinese pottery workers, showing estimates of exposure and the age at which silicosis was determined.
(Adapted from Sun et al. 2011, p.2931[3].)

As the risk greatly increases as exposure to RCS exceeds 0.1 mg/m^3 - it is really important that exposures be maintained << 0.010mg/m^3 (TWAs).

Besides silicosis and lung cancer, RCS exposure has also been linked to pulmonary tuberculosis, chronic obstructive pulmonary disease (COPD), and autoimmune disease (chronic arthritis).

OHCOW endorses lowering the MLTSD proposal to lower the TWA. OHCOW further recommends that Ontario adopt a TWA of 0.01 mg/m^3.

1. OCRC (2017) Burden of Occupational Cancer in Ontario.
2. OSHA (2016) Occupational Safety and Health Administration (OSHA) Federal register final rule respirable crystalline silica (RCS).
3. Kachuri1, L., P.J. Villeneuve, M-É Parent, et al., "Occupational exposure to crystalline silica and the risk of lung

cancer in Canadian men", International Journal of Cancer 135:138–148 (2014).
4. *ACGIH,* Silica, Crystalline – α-Quartz and Cristobalite: TLV Chemical Substances 7th Edition *Documentation* Publication (2014).
5. Sun, Y, Bochmann, F, Marfield, P, Ulm, K, Liu, Y, Wang, H et al. 2011, 'Change of exposure response over time and long term risk of silicosis among a cohort of Chinese pottery workers', *J. Environ. Res. Public Health*, vol. 8, pp. 2923-2936.
6. Queensland Australia Mining and Quarrying Safety and Health Act 1999, Mining Health and Safety Regulation. https://www.legislation.qld.gov.au/view/pdf/2017-09-01/sl-2017-0166
7. Hedges, K. (2016). Assessment and control of respirable crystalline silica in quarries and dimension stone mines. http://researchdirect.uws.edu.au/islandora/object/uws%3A36593/datastream/PDF/view
8. IARC. (2018). Silica Dust, Crystalline, in the form of Quartz or Cristobalite. Volume 100C. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. https://monographs.iarc.who.int/wp-content/uploads/2018/06/mono100C-14.pdf
9. Lee et al. (2016), Silica Measurement with High Flow Rate Respirable Size Selective Samplers: A Field Study. Ann Occup Hyg 2016 60 (3): 334 – 347. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4779386/

Hydrogen Sulfide (2021)

In 2009 the ACGIH[(1)] adopted a 1 ppm TWA TLV for hydrogen sulfide (H_2S) and a STEL of 5 ppm. The Ontario TWA 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 "scientific validity" of the 10 ppm TWA. The Dutch Expert Committee on Occupational Safety of the Health Council (DECOS) recommended[(3)] at health-based TWA of 1.6 ppm in 2006. Fielder et al (2007)[(4)] documented effects (likely due to odour) for subjects exposed to 0.05 ppm. At this exposure, some people may temporarily experience mild symptoms of discomfort, including nausea, headache, and irritability due to the odour. Asthma symptoms may be triggered. In addition, at 0.75ppm there may be transient lung function changes in healthy, moderately exercising, non-asthmatic individuals. From 0.01–0.3 ppm, the odour threshold is highly variable between individuals[(5)]. From 1–5 ppm, there is moderate offensive odour, which may cause nausea, tearing of the eyes, headaches, loss of sleep with prolonged exposure[(5)].

If there was no STEL, Reg. 833. Sec. 4.3.ii would apply: a TWA without a STEL or C shall never exceed 5 x TWA at any time (sometimes called excursion limits – ceiling). A Ceiling (C) of 5 ppm would align with the current legislation. **OHCOW endorses the MLTSD proposal of TWA of 1 ppm, but does not endorse the STEL of 5 ppm. OHOCW recommends a C of 5 ppm.**

1. ACGIH, Hydrogen Sulfide. Documentation of the Threshold Limit Values and Biological Exposure Indices. American Conference of Governmental Industrial Hygienists. Cincinnati. 2009.
2. Bhambhani, Y., R. Burnham, G. Snydmiller, and I. MacLean, "Effects of 10-ppm Hydrogen Sulfide Inhalation in Exercising Men and Women: Cardiovascular, Metabolic, and Biochemical Responses", Journal of Occupation & Environmental Medicine 39:122-129 (1997).
3. Dutch Expert Committee on Occupational Safety of the Health Council (DECOS), "Hydrogen sulphide: Health-based recommended occupational exposure limit in the Netherlands", 2006.
4. Fiedler, N., H. Kipen, P. Ohman-Strickland, et al., "Sensory and Cognitive Effects of Acute Exposure to Hydrogen Sulfide", Environmental Health Perspectives 116:78–85 (2008).

5. Guidotti TL (1994), Occupational exposure to hydrogen sulfide in the sour gas industry some unresolved issues. Int Arch Occup Environ Health (1994) 66: 153 – 160.

4,4'-Methylene bis (2-chloroaniline) (MBOCA; MOCA®) (2021)

The MLTSD is proposing raising the TWA from 0.0005 ppm (0.005 mg/m^3) to 0.01 (IPV).

Exposure to carcinogens should not be raised without excellent evidence, and should be as low as reasonably achievable (ALARA). MOCA is an IARC Group 1 carcinogen(1), with GHS Hazard Code: H350 may cause cancer(2). *Citations: IARC, ECHA*

OHCOW does not endorse this proposed change, and recommends the TWA remains at 0005 ppm (0.005 mg/m^3).

1. IARC. (2010). Some Organic Amines, Organic Dyes, and Related Exposures: 4,4'metylenebis(2-chloroaniline). Volume 99. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. https://publications.iarc.fr/_publications/media/download/2958/04f67887be058cb7ae62b10572bf41e37643b15c.pdf
2. European Chemical Agency (ECHA) Profile: 5,4'metylenebis(2-chloroaniline). https://echa.europa.eu/brief-profile/-/briefprofile/100.002.654

New Agents Not Under MLTSD Review in 2021:

Chemical	Current TWA	Recommended TWA	Basis
Asbestos*	0.1 f/cc	TWA 0.002 f/cc	Netherlands HC
1-methyl-pyrrolodine (CAS 872-50-4)	TWA 100 ppm	TWA 10 ppm (STEL 20 ppm) Skin notation	EU TWA
Silver and compounds (CAS 7440-22-4)	Metal, dust and fume TWA 0.1 mg/m^3	Metal, dust and fume TWA 0.01 mg/m^3	NIOSH
	Soluble compounds, as Ag TWA 0.01 mg/m^3	No change: TWA 0.01 mg/m^3	NIOSH
Silver nanoparticles	No TWA	TWA 0.9 µg/m^3	NIOSH
Trichloroethylene (CAS 79-01-6)	TWA 10 ppm (STEL 25 ppm)	TWA 10 ppm (STEL 25 ppm) Skin notation	CAREX Canada

*see below next section for additional discussion

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 TWA 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/cc (median exposure estimate – 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/cc (fibres per cubic centimetre, sometimes referred to as fibres per milliliter).

The Ontario TWA is based on the 1998 ACGIH TLV of 0.1 f/cc, which was designed to prevent workers from developing asbestosis (fibrosis of the lung – the documentation of the TLV assumes that if asbestosis is prevented then lung cancer will also be prevent – a theory that has been to be false by subsequent studies). It should be noted that this is quite an old exposure limit, and while it is the legal standard in Ontario, there has been a lot of research in the intervening years, particularly regarding the risks associated with low level exposures to asbestos.

The jurisdiction which has the most up-to-date TWA for asbestos is the Netherlands. In 2010 the Health Council of the Netherlands published[(2)] an extensive review titled: Asbestos: Risks of environmental and occupational exposure (https://www.gezondheidsraad.nl/sites/default/files/201010E.pdf). Based on the conclusions of this extensive review of the literature and its detailed risk assessment, in 2014 the government in the Netherlands lowered their 8 hour time weighted (TWA) occupational exposure limit for asbestos from the previous limit of 0.01 f/cc, down to 0.002 f/cc for chrysotile asbestos (the most commonly encountered form of asbestos). The purpose of the standard was to limit worker exposures to asbestos to prevent lung cancer and/or mesothelioma.

Accordingly, OHCOW recommends that the asbestos TWA be lowered to a **TWA of 0.002 f/cc**.

1. Offermans, N.S.M., R. Vermeulen, A. Burdorf, et al., "Occupational Asbestos Exposure and Risk of Pleural Mesothelioma, Lung Cancer, and Laryngeal Cancer in the Prospective Netherlands Cohort Study", Journal of Occupational and Environmental Medicine 56:6-19 (2014)
2. Health Council of the Netherlands. Asbestos: Risks of environmental and occupational exposure. The Hague: Health Council of the Netherlands, 2010; publication no. 2010/10E.

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.

Wheat Flour Dust (Total dust) (2004)

The current Ontario TWA 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 baker's 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 baker's 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 TWA 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 – suggesting the likely chance of multiple sensitizations. Therefore, ACGIH recommends a TLV 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.

The Dutch Expert Committee on Occupational Standards (DECOS) has recently updated their 2004 recommendation of 0.12 mg/m^3 of inhalable wheat flour dust to 0.2 mg/m^3. This value was calculated as the level at which it is estimated that the risk of sensitization will increase 1% from the background level. The Committee explains the increase in the TWA as a result of the fact that "the Committee has combined new data and data used previously, which increases the reliability of the estimation." (*page 6*) In another section of this submission we reference a paper by Daniels (2018) which calculates a benchmark dose for TDI using a benchmark dose associated with a risk of developing asthma at the incidence of 1 in 1000 (0.1%). OHCOW is of the position that the rate of 1 worker developing asthma among 100 exposed is an unacceptable level of risk.

To protect workers who are exposed to flour dust, OHCOW recommends that workplace exposures should be kept as low as reasonably achievable (ALARA), and OHCOW recommends a **TWA of 0.2 mg/m^3**. Please note that even at this lower TWA the risk of exposed workers developing occupational asthma is still unacceptably high.

1. ACGIH [2001]. Flour Dust. Documentation of the threshold limit values for chemical substances, 7th Edition. Cincinnati, OH: American Conference of Governmental Industrial Hygienists.
2. Smith T. Preventing baker's asthma: an alternative strategy. Occup Med. 2004; 54: 21-27
3. Elms J., Beckett P., Griffin P., et al. Job categories and their effect on exposure to fungal alpha-amylase and inhalable dust in the U.K. baking industry. AIHAJ. 2003; 64: 467-471
4. Baur X. Are we closer to developing threshold limit values for allergens in the workplace? Ann Allergy Asthma Immunol. 2003; 90: 11-18 (suppl.)
5. Baur X. Baker's asthma: causes and prevention. Int Arch Occup Environ Health. 1999; 72: 292-296
6. Nieuwenhuijsen M., Heederik D., Doekes G., et al. Exposure-response relations of α-amylase sensitization in British bakeries and flour mills. Occup Environ Med. 1999; 56: 197-201
7. Houba R., Doekes G., Heederik D. Occupational respiratory allergy in bakery workers: a review of the literature. Am J Ind Med. 1998; 34: 529-546
8. Health Council of the Netherlands. Dutch Expert Committee on Occupational Standards. Wheat and other cereal flour dusts. The Hague: Health Council of the Netherlands, 2004; publication no. 2004/02OSH.
9. Health Council of the Netherlands. Wheat and other cereal flour dusts. Health-based recommendation on occupational exposure limits The Hague: Health Council of the Netherlands, 2017; publication no. 2017/10.
10. Daniels, R. D. (2018). Occupational asthma risk from exposures to toluene diisocyanate: A review and risk assessment. American Journal of Industrial Medicine, 61(4), 282-292.

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 MLTSD in 2004 but not adopted in favour of the current 0.5 f/cc TWA. The industry recommends a 0.5 f/cc exposure standard, however it also recognizes that TWAs as low as 0.1 f/cc have been adopted (e.g. Norway and France). Most provinces in Canada have lowered the RCF TWA 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 SCTWA[(3)] has recommended an TWA 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 RCFs a decade ago were often (40%) over the ACGIH TLV.

Given that some RCFs under thermal stress will convert to crystalline silica, the combined exposure to RCFs and crystalline silica warrants protection based on at least additive co-exposures. Also given that both the TWA proposals for workers exposed to RCFs and crystalline silica have not been adopted by the MLTSD, this leaves workers with combined RCF and silica exposure particularly vulnerable. OHCOW recommends the adoption of the ACGIH TLV for an TWA for RCFs: **TWA 0.2 f/cc**.

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

Wood dust (2004):

Reg. 833 classifies wood dust into two categories:

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

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

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

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

In 1965, an excess of sino-nasal adenocarcinoma was observed among furniture workers exposed to wood dust. This prompted further research which found an excess risk among other workers employed in wood-related industries such as logging, sawmills, furniture making, and carpentry.

The highest risk of sino-nasal adenocarcinoma was observed in workers who were exposed to hardwoods such as beech and oak. However, a majority of the research, although examining the risk of cancer, did not specify the type of wood. Furthermore, wood workers are often exposed to mixed woods – not just one. Based on this information, IARC classifies wood dust as a Group 1 human carcinogen. IARC further states that this evaluation was based on workers exposed to hardwood dusts.

Several case-control studies indicate that there may be an excess risk of 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 TWA. 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 – 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 TWA.

Ten years ago the SCTWA (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. OHCOW recommends: **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**.

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
3. Chan-Yeung M. Western Red Cedar and other wood dusts. In Bernstein L., Chan-Yeung M., Malo J., Bernstein D. Asthma in the workplace. 1993. New York. Marcel Dekker. pp. 503-531

4. Chan-Yeung M., Ashley M., Corey P., et al. A respiratory survey of cedar mill workers. I. Prevalence of symptoms and pulmonary function abnormalities. J Occup Med. 1978; 20: 323-327
5. Goldsmith D., Shy C. Respiratory health effects from occupational exposure to wood dusts. Scand J Work Environ Health. 1988; 14: 1-15 (review)
6. Noertjojo H., Dimich-Ward H., Peelen S., et al. Western Red Cedar dust exposure and lung function: a dose-response relationship. Am J Respir Crit Med. 1996; 154: 968-973
7. Schlünssen V., Schaumburg I., Heederik D., et al. Indices of asthma among atopic and non-atopic woodworkers. Occup Environ Med. 2004; 61: 504-511
8. Schlünssen V., Schaumburg I., Taudorf E., et al. Respiratory symptoms and lung function among Danish woodworkers. JOEM. 2002; 44(1): 82-98
9. Holmström M., Wilhelmsson B. Respiratory symptoms and pathophysiological effects of occupational exposure to formaldehyde and wood dust. Scand J Work Environ Health. 1988; 14: 306-311
10. Vedal S., Chan-Yeung M., Enarson D., et al. Symptoms and pulmonary function in Western Red Cedar workers related to duration of employment and dust exposure. Arch Environ Health. 1986; 41(3): 179-1833
11. Pisaniello D., Connell K., Muriale L. Wood dust exposure during furniture manufacture – Results from an Australian survey and considerations for threshold limit value development. AIHAJ. 1991; 52: 485-492
12. Whitehead L., Freund T., Hahn L. Suspended dust concentrations and size distribution and quantitative analysis of inorganic particles form woodworking operations. AIHAJ. 1981; 42: 461-467
13. SCTWA (Scientific Committee on Occupational Exposure Limits). "Recommendation from the Scientific Committee on Occupational Exposure Limits: Risk assessment for Wood Dust" SCTWA/SUM/102 final December 2003

Sulphur Dioxide (2009)

The ACGIH documentation(1) for the sulphur dioxide TLV cites a number of studies published as early as the 1960s up to the 1990s 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 30 years ago and still have not been acted upon. It should be noted that the Ontario STEL is 20 times for sulphur dioxide higher than the ACGIH STEL!

The SCTWA has a two tiered TWA for sulphur dioxide; for "healthy workers" the limit is 0.5 ppm (TWA) and 1 ppm (15 min STEL); and for "asthmatics" 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 "healthy" or "general" workers. Making this distinction for such a common variation in general health can lead to issues of discrimination – focusing on the suitability of the individual to the workplace rather than the other way around. This approach would be a regressive policy step.

OHCOW recommends a TWA of **0.2 ppm**.

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

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 TWA (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 (SCTWA, 2012) is for a TWA of 0.2 ppm with a short term exposure limit of 1 ppm (SCTWA, 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; SCTWA, 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 SCTWA (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 TWA 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 SCTWA recommendations take into consideration the effect of nitrogen dioxide on asthmatics; the US EPA's Risk and Exposure Assessment (2008) found that the majority of asthmatics may experience nitrogen dioxide-related airway hyper-responsiveness following short-term exposures between 0.1 ppm and 0.3 ppm nitrogen dioxide.

From a meta-analysis of 19 controlled human exposure studies involving mild asthmatics, the US EPA (2008) report that the LTWA 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 SCTWA TWA TWAs 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 LTWA 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 TWA 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. **OHCOW recommends a TWA of 0.05 ppm**, which would be protective of all asthmatics; however, a STEL or CEILING approach would also be needed for peak exposures.

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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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 RTWAs, et al.(2). In this study the authors found that the upper 95th confidence limit of the lifetime integrated exposure metric corresponded to 3.575 mg/m^3–yrs of total 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:

> "On 17-18 June 2006, the Scientific Committee on Neurotoxicology and Psychophysiology and the Scientific Committee on the Toxicology of Metals of the International Commission on Occupational Health (ICOH) convened an International Workshop on *Neurotoxic Metals: Lead, Mercury and Manganese*

> – *From Research to Prevention (NTOXMET)* at the University of Brescia. Scientists and physicians from 27 nations participated. Data were presented for each of the three metals on environmental sources, fate and distribution; human exposure; clinical, subclinical and developmental neurotoxicity; epidemiology; risk assessment; and prospects for prevention. Ongoing and future studies were described and discussed.
>
> …
>
> The current occupational exposure standard may not protect workers against subclinical neurotoxicity. The value for air manganese concentration in inhalable/total dust of 100 μg/m^3 should be adopted to protect the workers from prolonged exposure and consequent long-term effects."
> http://www.amm.se/icoh_sctm/pdf/Declaration%20of%20Brescia%20AJIM.pdf

The recommended level of 100 μg/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:

> "According to a statistical model of RTWAs 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." (p. 2)

Thus, it is clear that even this TWA 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 "impaired hand steadiness" 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) "disorders of ear including deafness" in 2005 and 31 "disorders of ear including deafness" 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, "A limitation with almost all of NITS studies is that the presence of presbycusis will efface the notch, …" (page 463), thus the "notch" 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:

> "The 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." (page 643)

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

> "During their working activity, outdoor and indoor workers are exposed to different noise levels LEX < 80 dB(A). At mid–low frequencies (250–2000 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 mid–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." (page 302)

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

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

> "… the 1997 NIOSH analysis of those frequencies likely to be affected by noise (1, 2, 3, and 4 kHz; …) 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." [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 WSIB's 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 MLTSD 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:

> "In 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." (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:

> "1) Human data indicate auditory effects under or near existing TWAs. 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 TWAs.
> 3) Human data are poor or lacking. Animal data indicate an auditory effect well above existing TWAs." (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.

Vyskocil et al. (2012) evaluated the weight of evidence of ototoxic potential of industrial chemicals by conducting a literature review of 224 ototoxic substances related to animal and human studies. Materials such as ethyl benzene, n-hexane, and xylene were classified as potentially ototoxic individually, while materials such as lead, styrene, toluene, and trichloroethylene were classified as ototoxic individually without the consideration of combined noise exposures. Noise and toluene in combination was classified as presenting evidence of increased risk for NIHL at noise exposures below 85 dBA Lex,8. Toluene is commonly used in a number of industry sectors.

An extensive review of the literature was conducted by Campo et al. (2009). Epidemiological data, animal studies, and case reports were studied. The following were shown to have "strong" weight of evidence for ototoxicity:

- toluene,
- ethylbenzene,

- n-propyl benzene,
- styrene,
- trichloroethylene,
- xylene,
- n-hexane,
- carbon disulfide,
- carbon monoxide,
- hydrogen cyanide,
- acrylonitrile,
- lead,
- mercury

Further, cadmium, arsenic, bromates, and halogenated hydrocarbons were classified as having a "fair" weight of evidence for ototoxicity.

From a paper published by Schaal et al. (2018) which evaluated the combined effect of metal/solvent and noise on hearing loss as compared to noise exposure alone in a population of shipyard personnel, it was found simultaneous exposures classified as high for metal/solvent/noise appear to damage hearing more than exposure to noise alone. Hearing changes were significantly worse at 1000Hz. High metal/solvent exposure is classified with an exposure profile with an exceedance fraction at least 5% for each metal concentration in accordance with OSHA action levels. A noise level of at least 85dBA was considered high exposure. It was suggested that the hearing conservation program should take into consideration combined exposures to metals, solvents, and noise, not simply just noise. The metal/solvent of focus were lead, cadmium, arsenic, toluene, and xylene. As there is no combined exposure limit established for lead, cadmium, arsenic, toluene, and xylene considering ototoxicity as an endpoint, with and without co-exposure to noise, and Material Safety Data Sheets (MSDS) generally do not provide information on ototoxicity (AIOH, 2016), therefore regular audiometric testing is recommended for workers who are exposed to ototoxic substances. Recommending 80 dBA $L_{ex,8}$ is considered practical and will especially identify any potential NIHL from the combination of noise and ototoxic agents.

Currently, under O. Reg. 381/15: Noise of Occupational Health and Safety Act, audiometric testing is not mandatory in workplace. The legislative requirement has a requirement for health monitoring of workers exposed to hazards who rely on hearing protection for risk control. The Alberta government had recognized the importance of audiometric testing being an important component leading to the success of a noise management program. In fact, it is the only way to actually determine if occupational hearing loss is being prevented by the noise management control measures. As required in Section 223 of Part 16 of the Alberta Occupational Health and Safety (OHS) Code, audiometric testing component of a noise management program is made mandatory if a noise exposure assessment confirms that workers are exposed to excess noise at the work site, the employer must develop and implement a noise management program. The noise management program must include audiometric testing and an annual review of the effectiveness of the noise management program to preventing hearing loss. This was published - July 01, 2009 (Government of Alberta).

In addition to Alberta government, the following jurisdictions in Canada set out requirements for audiometric testing:

Province/Territory	Legislation
British Columbia	Occupational Health and Safety Regulation Part 7 Section 7.8 and 7.9
Saskatchewan	The Occupational Health and Safety Regulations, 1996 Part VIII Section 113
Manitoba	Workplace Safety and Health Regulations Part 12 Section 12.5 and 12.6
Quebec	Regulation respecting occupational health and safety Division XV Section 136
New Brunswick	Underground Mine Regulation Part III Section 12
Prince Edward Island	General Regulations Part 8 Section 9.8 and 8.10
Newfoundland and Labrador	Occupational Health and Safety Regulations Part VI Section 68
Northwest Territories	Occupational Health and Safety Regulations Part 8 Section 116 and 117
Nunavut	Consolidated Occupational Health and Safety Regulations Part 8 Section 116 and 117
Yukon Territories	Occupational Health Regulations Section 6

Taken from CCOHS, Updated on March 16, 2017.

Internationally, the Australia Standards AS/NZS 1269 Set provides an integrated approach to establishing, implementing and evaluating an occupational noise management program. Audiometric testing of persons exposed to excessive noise may be used for four distinct purposes as mentioned in following:

> " (a) The early detection of deterioration of hearing in persons exposed to excessive noise, so that noise management measures can be reviewed and action taken to prevent further deterioration.
> (b) The identification and documentation of existing hearing loss.
> (c) The prompt direction of those individuals who are identified as having a hearing loss to an appropriate rehabilitation program.
> (d) The supply of any special communication or warning system that may be required within the workplace for an individual with a hearing loss."

The Australian Model Code of Practice for Managing Noise and Preventing Hearing Loss at Work (Safe Work Australia, 2011) states that the daily noise exposure of workers exposed to ototoxic agents should be reduced to a level of no more than 80 dBA. Additionally, it also states

that regular audiometric testing should occur for workers who are exposed to ototoxic substances with airborne exposures at more than 50% of the Australian national exposure standards regardless of noise exposure level. In cases where workers are exposed to an 8-hour noise exposure level greater than 80 dBA (Leq) and/or C-weighted peak noise level Lc, peak greater than 135 dB with any level of ototoxic chemical exposure – audiometric testing should be carried out on a regular basis. The American Conference of Governmental Industrial Hygienists (ACGIH) recommends that periodic audiometry should be carried out and the results should be carefully reviewed for combined exposure (Fuente et al., 2012). The United States Army also recommends annual audiometric testing when chemical exposure (disregarding the use of respiratory protection) is equal to or greater than 50% of the most stringent criteria for occupational exposure limits, regardless of the noise level (Fuente et al., 2012).

Recent publication by OSHA further acknowledge the effect of induced ototoxic reactions by certain chemical exposures and indicated:

> "OSHA's occupational noise exposure standard at 29 CFR 1910.95 only requires audiometric testing at the noise action level (i.e., an 85-decibel 8-hour time-weighted average). However, wearing hearing protection and using audiometric testing to detect early signs of hearing loss, even on workers exposed below the action level and ototoxic chemicals below the PEL, may prevent hearing loss from their synergic effects" [Accessed at https://www.cdc.gov/niosh/docs/2018-124/pdfs/2018-124.pdf, on April 30, 2018]

Mandatory audiometric testing is an important component of successful hearing conservation program to effectively take action to prevent further deterioration as part of continuous monitoring. Audiometry is an essential part of a hearing conservation program and requirements for audiometry should consider the impact of ototoxic agents on NIHL.

Furthermore, 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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DSR Updates (2015)

OHCOW submitted a response to the MLTSD (formerly MOL)'s Consultation on Proposed Changes to Ontario Regulation 490/09 – Designated Substances and the Requirements for Medical Surveillance, Respiratory Protection and Measuring. Our submission was titled Preventing Occupational Disease through the Designated Substance Codes for Exposure

Measurement, Respiratory Protection and Medical Surveillance (July 6, 2015). At OHCOW we have a numerous time asked each other if anyone knows the status of these proposals since our work with designated substance both from a medical and hygiene perspective is so frequent. Also, since the designated substances regulation (O.Reg 490/09) is tightly related to O.Reg. 833 we reiterate the summary recommendations contained in this submission:

The Occupational Health Clinics for Ontario Workers Inc. (OHCOW) has extensive experience with designated substance assessment and control programs, including devising strategies for collecting exposure information, specifying what needs to be included in a control program and developing and delivering medical surveillance programs. OHCOW also has extensive clinical experience with workers who have suffered illness or injury due to exposures to various designated substances. Based on our experience, and the review of materials supplied by the MLTSD, we are providing the following observations and recommendations regarding the "Consultation on Proposed Changes to Ontario Regulation 490/09 – Designated Substances and the Requirements for Medical Surveillance, Respiratory Protection and Measuring".

1. Our experience indicates that the current status of designated control programs in Ontario has deteriorated through neglect over the years since they were first initiated – we recommend that the MLTSD reinstate the deployment of occupational nurses, physicians and hygienists to audit control programs to ensure they meet the regulatory requirements.

2. We agree with the MLTSD that all workers working in workplaces subject to a designated substance program should fall under the provisions of such a program whether or not they are third party contractors, construction workers, or any worker as defined by the Occupational Health and Safety Act.

3. We agree with the proposal to update the code for measuring designated substances. However, we recommend that it include the requirement to conduct such sampling using appropriate sampling strategies (as defined in current occupational hygiene practice) – allowing for a range of qualitative to quantitative techniques as outlined in the hierarchy of exposure assessment.

    3.1. We recommend that the reporting of exposure data be centralized to ensure consistent data collection (the MLTSD?, CAREX?) to form the basis of valid and reliable personal exposure records.

    3.2. We have particular concerns regarding the measurement of non-monomeric isocyanates and would recommend that their measurement be required in the regulation.

4. We also concur with the strategy of using the CAN/CSA-Z94.4-11 – "Selection, Use, and Care of Respirators" as the pattern to establish appropriate respiratory protection programs.

5. Given the general approach of the proposed codes for measuring and prescribing respiratory protection for exposures to the designated substances, we recommend that this

approach also be applied to the chemical agents listed in O.Reg 833, Control of Exposure to Biological or Chemical Agents.

6. In general, while we endorse the changes to the specific medical surveillance codes, we have a number of concerns about the outdated bases for the recommendations in the supporting documentation produced by Holness et al (2010):
   6.1. The proposal to not use low-dose CT scans for high risk workers is outdated; since the time the Holness et al. review was written, the cited organizations have reversed their position – therefore, we recommend this issue be revisited and would endorse the authors' recommendation to find some mechanism that could respond to the evolving nature of the evidence for these techniques.
   6.2. The suggestion that isocyanate biological monitoring is not yet mature is no longer true – again, we recommend that this issue be reconsidered given the current availability of valid techniques.
   6.3. While we commend the reductions in the blood lead and urine mercury levels, we note that the current literature shows that health effects are associated with exposures resulting in blood lead and urine mercury levels lower than the proposed criteria; also, there is evidence to show an increase of genetic mutations in workers exposed to cumulative levels of vinyl chloride below current TWA levels.
   6.4. While the use of a recent X-ray is commendable for reasons of reducing radiation exposure, we recommend, however, that the X-ray be re-read in light of the designated substance exposure information.
   6.5. As per the principles outlined by L' Institut national de santé publique du Québec in their 2011 publication, "Reference Framework for Screening and Medical Surveillance in Occupational Health"; workers undergoing any medical tests/exams, must be made aware of all the screening benefits and disadvantages (including economic and quality of life implications) prior to consenting to participate in the medical surveillance program. We recommend that this type of informed consent be required for every occasion that a medical surveillance activity take place.

7. We suggest that the model on which the designated substance control programs were initially established is aging and in need of update – we recommend that the MLTSD consider the model of the Ontario Health Study and the use of e-health records to modernize the medical surveillance programs (both "active" and "passive"). We note that "passive" monitoring using Sentinel Health Events (Occupational) as screening criteria could be extended to cover substances listed in O.Reg 833 for which we have evidence of association with specific health outcomes.

8. Finally, we recommend that the MLTSD update its guidance publications related the designated substances (i.e. "Designated Substances in the Workplace: A General Guide to the Regulations", and the specific guides for physicians conducting medical surveillance tests and exams) – we also offer OHCOW's assistance in bringing these publications up to date.

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

2,4-Dichlorophenoxyacetic acid (2,4-D) (2018)

First introduced for agricultural use in 1944, 2,4-D is widely used as an herbicide for the control of broadleaf plants and as a plant growth regulator (ACGIH 2017, IARC 2017). The ACGIH TLV has been 10 mg/m^3 since it was first adopted in 1956. While there had been a STEL, this was withdrawn in 1986. The ACGIH (2017) changed its TLV for 2,4-D from 10 mg/m^3 to 10 mg/m^3 "Inhalable Fraction" in 2013 (ACGIH, 2013) and this has been retained in 2017, the classification of "Not Classifiable as a Human Carcinogen" (Group A4), which was added in 1996, has also been retained; however, the "Skin Notation", which was added in 2013, was removed in 2017. The current TLV–TWA (ACGIH, 2017) is intended to protect against possible thyroid and the kidney effects seen in animal studies.

Several jurisdictions, including Ontario (Gestis, 2018), have an TWA-TWA of 10 mg/m^3, which is based on the ACGIH TLV that has changed little since 1956. Several countries have recently adopted TWAs and almost all of these are from ~2 to 10 times lower. In Denmark, Austria, Germany, Hungary, the TWA-TWA is 1 mg/m^3 with STELs that range from 2 mg/m^3 to 8 mg/m^3 (Denmark, 2007; Austria, 2007; Germany, 2018; Hungary, 2018). In Switzerland, Norway, Romania and Poland the TWA-TWA ranges from 4 mg/m^3 to 7 mg/m^3 with STELs from 8 mg/m^3 to 10 mg/m^3 (Norway, 2011; Romania, 2006; Switzerland, 2016; Poland, 2014). Skin absorption is noted in the TWAs for Germany (2013; 2018), Austria (2011), Denmark (2007), Hungary (2018) and Switzerland (2016).

The German MAK (2013) is 5 times lower than the ACGIH TLV (2017) due apparently to differences in the interpretation of the data from animal studies. The TLV has used a two-year rodent chronic feeding study (Charles, 1996a) to determine an NOAEL of 5 mg/kg, while the MAK is based on an NOAEL of 1 mg/kg body weight from a one-year feeding study in the dog (Charles, 1996b). This appears to be the fundamental difference between the two approaches; however, the application of uncertainty factors may also have contributed.

In the previous documentation of the TLV, the ACGIH (2013) had determined, that "acute systemic effects can occur following dermal exposures in humans" and referenced Bradberry et al. (2000). In the 2017 TLV documentation, citing the same and similar reviews by the same group - Bradberry et al. (2000; 2004) - they found that this information "does not suggest skin absorption would lead to systemic effects", which is the opposite interpretation. The authors, Bradberry et al., are from a Hospitals Poison Control centre and are concerned with clinically significant exposures. As the process for developing TWAs considers exposures causing subclinical effects, i.e. at the NOAEL, these reviews are not informative for this purpose.

The documentation for the German MAK (2013) and for the ACGIH TLV (2017) both cite Grover (1986), who studied herbicide sprayers and found that inhalation exposure accounted for

approximately 2% of the total absorption of 2,4-D, while >98% was due to dermal exposure, particularly the hands which accounted for 80% to 90%. While the TLV cites this as evidence that primary route of exposure is dermal, the MAK has interpreted this to mean that skin absorption could lead to a dose greater than their NOAEL. They determined that inhalation of 2,4-D at an exposure level of the MAK-TWA, which is 2 mg/m3, would result in a dose of 285 µg/kg body weight, which is at the level of the NOAEL in a worker. Since skin absorption is so much greater, they reasoned that the NOAEL could be exceeded and so the skin absorption notation was appropriate (Germany, 2013).

As the dermal route of exposure is the primary route for herbicide applicators, and as toxicity reportedly does occur at levels below those considered by the ACGIH TLV (2017), the skin notation currently used in Ontario and several other jurisdictions is an important indicator that effective controls are needed to limit skin exposure.

IARC (2017) has classified 2,4-D in Group 2B, "Possibly Carcinogenic to Humans", finding limited evidence in experimental animal studies (reticulum cell sarcomas, pulmonary adenoma, astrocytoma and lymphoma) but inadequate evidence in humans (most notably, non-Hodgkin-lymphoma, leukaemia, soft tissue sarcoma, and glioma). To illustrate, statistically significant increased risks of non-Hodgkin lymphoma associated with 2,4-D exposure have been reported in a Canadian population case control study and among Hispanic farm workers in California (McDuffie et al., 2001; Mills et al., 2005); and, Beane Freeman et al. (2013) recently reported from the prospective Agricultural Health Study of US licensed pesticide applicators (through 2008) that 2,4-D use was associated with elevated risks of brain cancer and statistically significant elevated risks of gastric cancer.

There is no domestic production of 2,4-D in Canada (CAREX, 2016) and therefore the occupations with greatest potential for exposure in Ontario would be herbicide supply workers and those doing the application such as farmers, farmworkers, lawn maintenance workers, etc. Peters et al. (2018) have selected 2,4-D as a high priority occupational carcinogen for creating new CAREX Canada profiles and for recommending new or updated occupational exposure estimates. CAREX Canada does not have occupational exposure estimates for 2,4-D available on its website at this time.

The ACGIH has specified a STEL for 2,4-D in the past, and STELs are currently used in several other jurisdictions (Denmark, 2007; Austria, 2007; Germany, 2018; Hungary, 2018; Romania, 2006; Switzerland, 2016). The German MAK specifies a STEL of 4 mg/m^3, which is 2 times the level of the TWA, because it is an irritant in certain formulations. (Germany, 2013; NPIC, 2008).

Germany has had an TWA-TWA for 2,4-D of 1 mg/m^3 for 20 years, and more recently Denmark, Austria, and Hungary have adopted that level, which is a clear indication that this level is both technically achievable economically feasible; Ontario can adopt this significantly more protective TWA without concern for negative repercussions. Ontario should keep the skin notation that it has currently and apply this to the suggested lower TWA-TWA of 1 mg/m^3. As is the case in Germany, Denmark, Hungary Austria, Switzerland and Romania, Ontario should also

adopt a STEL (based on irritation rather than systemic effects). As 2,4-D is possibly carcinogenic to humans (IARC, 2017), lowering the TWA would be a prudent step towards cancer prevention.

Ontario workers, such as those in agriculture and landscaping, who use 2,4-D and could potentially benefit from the regulation of 2,4-D, appear to be excluded from doing so by Ontario Regulation 414/05, which applies to Farming Operations. The Ministry of Labour should address this gap.

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Formaldehyde (2018)

The basis for the ACGIH recommendations was to minimize the potential for sensory irritation to namely the eye and upper respiratory tract, as well as nasopharyngeal cancer and leukemia (ACGIH, 2017). The Lowest Observed Adverse Effect Levels (LOAEL) for eye and upper respiratory tract irritation for humans involved both continuous and peak exposures (Lang et. al 2008).

TLV Chronology (Documentation from ACGIH for Formaldehyde):

- The 2017 ACGIH adopted a TLV of 0.1 ppm and the TLV –STEL of 0.3 ppm, RSEN and DSEN Notation, A1 Confirmed Human Carcinogen;
- Previously in 2015-2016 there was a TLV-CEILING 0.3 ppm, RSEN and DSEN, A2 Suspected Human Carcinogen (this is what OHCOW recommends adopting).

The ACGIH notations should also be adopted to alert workers of the health hazards with regards to exposure to Formaldehyde namely as a dermal sensitizer and a respiratory sensitizer. Formaldehyde has also been linked to occupational asthma. The fact that Formaldehyde is recognized as an A1 Confirmed Human Carcinogen (IARC, 2012), suggests strongly that Ontario needs to move forward and adopt the ACGIH notations for chemicals, RSEN and DSEN specifically for Formaldehyde (respiratory sensitization and dermal sensitization).

As per CAREX Canada, 152000 Canadians are occupationally exposed to formaldehyde, of which 66% are male. Out of all the provinces, Ontario workers are the highest exposed. The

main industries involved are wood product manufacturing workers, due to their use of formaldehyde containing resins and glues. Women are exposed to formaldehyde in hospitals, schools and clothing manufacturing. Generally speaking, other workers affected include, cooks exposed at low levels during the grilling of some foods, those exposed to emissions from solutions such as embalming fluids, pathology lab workers, wood and pulp and paper processing workers, those exposed to combustion sources (such as wood stoves), machinists, and health care professionals who may be exposed during medical device clean up (CAREX Canada).

In terms of irritant effects, Arts et al. (2006) found that workers had irritant effects to the eyes, nose and throat at levels above 1 ppm. As per Feldman and Bonashevskaya (1971), there were no reported health affects observed at Formaldehyde concentrations of 0.05 ppm. Neurophysiologic effects were observed at 0.05–1.50 ppm by the same authors. The Odour threshold is observed at 0.05–1.0 ppm in the air as per Hemminki et al., 1982. NIOSH has recommended a REL of 0.016 ppm (TWA) and a ceiling of 0.1 ppm, based on risk evaluations using human or animal health effects data and on analytical limits of detection or technological feasibility (NIOSH carcinogen policy). The NIOSH version of the International Chemical Safety Card (ICSC) identifies formaldehyde as a severe eye, skin and respiratory tract irritant following short term exposure. The identified effects of long-term or repeated exposure are skin sensitization and asthma-like symptoms; formaldehyde is also carcinogenic to humans. The NIOSH REL of formaldehyde was last updated on 2003.

Table 1 in The TLV Documentation for Formaldehyde 2017, "Human Adverse Health Effects Associated with Inhalation of Various Concentrations of Formaldehyde" (U.S. National Research Council, 1981) lists that from 0.10–25 ppm upper airway irritation and increased nasal airway resistance was observed by US NIOSH 1997 (Yefremov, 1970 et al). Other observations in the table included: lower airway and chronic pulmonary obstruction at 5-30 ppm; pulmonary edema, inflammation, pneumonia at 50-100ppm (U.S. NIOSH, 1997a, Zanni and Russo, 1957); and death at 100 plus ppm (Popa, et al., 1969, and Vaughn, 1939). With this information, it would appear preventative that a Ceiling value also be implemented so as to prevent any reactions amongst workers exposed to peak levels of formaldehyde exposure, especially given the fact that this is a respiratory and dermal sensitizer.

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 "anxious" personality traits accounted for the increased symptoms reports.
OHCOW's recommendations are the following:

Given this overwhelming documentation, OHCOW recommends that the MLTSD adopt the 2015 ACGIH TLV as the TWA, which is **0.3 ppm (ceiling)**; and recognizing its limitations also give serious consideration to adopting either the Health Canada indoor residential standard of 0.1 ppm (1 hour), or the WHO formaldehyde guideline (0.08 ppm).

We also recommend the MLTSD to adopt the ACGIH notations RSEN and DSEN for formaldehyde and chemicals in general.

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Toluene di-isocyanate (TDI) (2018)

TDI exposures are a concern in Ontario workplaces with respect to sensitization associated with over-exposures. TDI is a known respiratory sensitizer linked to occupational asthma. Workers at risk of exposure to isocyanates via the skin and respiratory tract include automotive painting, foam-blowing, and the manufacture of various polyurethane products such as furniture, adhesives, and insulation. Isocyanates remain the most common cause of occupational asthma worldwide, Redlich (2010). Exposure can also lead to irritation of both the skin and respiratory system.

The Current TWA for TDI is 0.005 ppm with a Ceiling of 0.02 ppm. The proposed TWA is 0.001 ppm with a STEL of 0.005 ppm and a skin notation added. A benchmark dose (BMD) was calculated in a 2017 study titled: "Occupational asthma risk from exposures to toluene di-

isocyanate: A review and risk assessment”**.** The benchmark dose (BMD) is defined as the dose that corresponds to a specific change in an adverse response compared to the response in unexposed subjects. The BMD-based TWA was 0.0004 ppm. The TWA based on low-dose extrapolation to working lifetime extra risk of developing asthma due to TDI exposure of 1/1000 corresponded to an exposure of 0.0003 ppm. Lowering the TWA closer to the BDM level would reduce worker exposure and reduce the chances of sensitization. There is real time instrumentation that can quantify TDI levels at the single-digit ppb level (1 ppb =0.001 ppm).

Given this overwhelming evidence, OHCOW would recommend that Ontario adopt the ACGIH TLVs, specifically a **TWA of 0.001 ppm with a STEL of 0.005 ppm**; with the caution that there still may be individual workers who will become sensitized thus warranting the application for the ALARA principle. Also, as per our recommendations concerning substitution, any workplace working with a sensitizer should investigate the possibilities of substituting a less toxic substance for the sensitizer. Based on our clinic experience of workers exposed to TDI and developing sensitization we perceive a significant need to reduce the TWA for TDI.

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Diesel Exhaust (2018)

There is no TWA for diesel particulate matter cover general workplaces under Regulation 833 so OHCOW commends the MLTSD for proposing an TWA. OHCOW’s position is that the current limit for mining (Regulation 834) of 0.4 mg/m^3 (TC) and the proposed limit for other workplaces (Regulation 833) of 0.16 mg/m^3 (TC) is not suitably protective and presents an unacceptable risk to health.

Ultimately a longer term strategy should be established to reduce all exposures towards a health based limit of 0.001 mg/m^3 (EC).

In 2017 Dr. Vermeulen provided a presentation through the OCRC in which he concluded that the “acceptable risk” and “maximum tolerable risk” levels for diesel exhaust would be below 1 µg/m^3 EC. Such limits are below current occupational exposure levels, and in some instances even below environmental exposure levels. These results bring into question if diesel engines using older technologies can be used in workplaces.

OHCOW acknowledges that it will take many years to phase out Traditional Diesel Engines (TDE), therefore, a strategy may practically be warranted to reduce diesel emissions. OHCOW endorses the policy recommendations in the OCRC report "Burden of Occupational Cancer in Ontario OCRC 2017, p.25[1.])

The fact that IARC now recognizes diesel exhaust as a proven human carcinogen, underlines the importance of creating an appropriately protective diesel exhaust TWA. The ACGIH in 2002[1.] proposed a TLV (TWA) of 20 µg/m^3 measured as elemental Carbon (EC) (the proposal was withdrawn in 2003 and never replaced[2.]). NIOSH[1.] in 1988 recommended that diesel exhaust be treated as a human carcinogen. NIOSH recommends that occupational exposures be controlled to as low as feasible[2]. In essence, they require that sampling be done in unexposed areas, for example, the air outside the building, and that levels inside the building not exceed those of outside. The US EPA estimates that the ambient outdoor level of diesel exhaust (<10 µm particle size measured by elemental carbon) would be up to 1-3 µg/m^3 [4.] Thus, NIOSH effectively recommends a level below 1 µg/m^3. NIOSH has published a method[5.] which they recommend to be used to measure the elemental carbon associated with diesel exhaust so as to distinguish it from other carbon sources such as cigarette smoke. In their analysis of exposures in the trucking industry NIOSH[6.] estimated that a 13 µg/m^3 working life exposure was associated with a 1-2% (10-20/1000) excess risk of lung cancer above the 5% background lung cancer risk. The EPA[4.] has developed a reference concentration (RfC) for diesel exhaust of 5 µg/m^3[.] of DPM (roughly equivalent to 3.1-6.6 µg/m^{3}of diesel exhaust as determined by elemental carbon) which was derived on the basis of dose-response data on inflammatory and histopathological changes in the lung from rat inhalation studies.

Finally, there is the question of exposure to other gases (sulphur compounds, other nitrogen oxides, VOCs, etc.). The EPA[4] [(page 1 – 7).] states:

> "Effects of DE exposure could be additive to or synergistic with concurrent exposures to many other air pollutants. … (e.g., potentiation of allergic effects, potentiation of DPM toxicity by ambient ozone and oxides of nitrogen)"

In the recent past, a number of new papers[7,8,] have been released particularly dealing with the lung cancer risks associated with exposure to diesel exhaust. In 2014 Bob Park from NIOSH reviewed the risk estimates associated with a working lifetime exposure to diesel exhaust and the risks of developing lung cancer [9]. The range of lifetime equivalent concentrations to diesel exhaust (measured as respirable elemental carbon REC) associated with a risk of 1/1000 (maximum occupational risk benchmark) was 0.32-0.94 µg/m^3[.]

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

> "Approximately 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." (Reference 10, page A37; AF = attributable fraction, DEE = diesel engine exhaust)

Five percent of Canada's working population is exposed to cancer-causing diesel engine exhaust (DEE) at work, according to CAREX Canada.

Here in Ontario, more than 300,000 workers are exposed. A recent report concluded these exposures cause 170 lung and 45 bladder cancers in workers annually. This same report also highlighted a significant regulatory gap in Ontario citing a complete lack of occupational exposure limits (TWA) for whole diesel exhaust or diesel particulate matter. With regard to the public consultation on a proposed TWA for diesel, the level the MLTSD is proposing **falls well short of what scientists believe is needed to protect exposed workers**. The Ministry proposes an TWA of 160 µg/m^3 for diesel particulate matter (DPM) measured as total carbon; while the globally-recognized Occupational Cancer Research Centre (OCRC) recommends lowering the TWA to 20 µg/m^3 (elemental carbon) for the mining industry and 5 µg/m3 (elemental carbon) for all other workplaces. In addition, relying on total carbon as a surrogate for diesel particulate matter (DPM) has been demonstrated to be a less sensitive and less accurate measure than elemental carbon. Elemental carbon (EC) is a better measure of exposure and less prone to interferences than total carbon (TC), therefore the limit should be set as EC, not TC. According to Debia et al. (2017):

> "The variability observed in the TCR/ECR ratio shows that interferences from non-diesel related organic carbon can skew the interpretation of results when relying only on Total Carbon data".

Because the mechanisms of lung cancer in humans are likely to be multifactorial, including direct genotoxicity, DEP induced oxidative stress and pulmonary inflammation, according to Taxell and Santonen 2017, it is currently not possible to identify a threshold level for carcinogenicity. Taxell and Santonen 2017, on behalf of the Finnish Institute of Occupational Health, Helsinki, Finland a is proposed that the TWA for DE be based on the cancer risk level calculated on the basis of recent epidemiological evidence. In addition, when the pulmonary inflammatory response seen in controlled human studies after 1–2 h exposure at 100 µg DEP/m3 (approximately 75 µg EC/m3) is taken into account, the TWA should be well below this level. There is sparse data available link high exposure to new technology DE with pulmonary inflammatory effects, without indicating genotoxicity or carcinogenicity (Bemis et al., 2015; Hallberg et al., 2015). In the long-term rat inhalation study, the LOAEL for inflammatory and histopathological changes in the lungs was 4.2 ppm NO_2 (12 mg DEP/m^3, approximately 3 mg EC/m^3) and the NOAEL was 0.9 ppm NO_2 (5 mg DEP/m^3, approximately 1 mg EC/m^3) (McDonald et al., 2015). The observed effects were mainly associated with NO_2, making NO_2 a good exposure indicator candidate for new technology DE. Because the emissions of DEP and the DEP-associated genotoxic compounds of new technology diesel engines are significantly lower than those of older technology diesel engines, the cancer risk (per kWh) is expected to decrease with new diesel technology. This is supported by the negative findings of the carcinogenicity study on rats (McDonald et al., 2015) and the available (although limited) in vivo genotoxicity data (Bemis et al., 2015; Hallberg et al., 2015). As the age and type of the engines and exhaust after-treatment systems applied vary within and between workplaces, it may be

appropriate to set an TWA value for DE as both respirable EC and NO_2. Neither of these values should be exceeded at workplaces where diesel engines are applied. OHCOW agrees with Taxell and Santonen (2017) that the TWA should be well below 75 µg EC/m^3.

The Dutch Expert Committee on Occupational Safety (DECOS), a committee of the Health Council of the Netherlands, derives so-called health-based calculated occupational cancer risk values 4 (HBC-OCRVs) associated with excess cancer risk levels of 4 per 1,000 and 4 per 100,000 as a result of working life exposure to substances. It concerns substances which are classified by the Health Council or the European Union in category 1A or 1B and which are considered stochastic genotoxic carcinogens. The Committee estimates that the concentration of elemental carbon (EC) from diesel engine exhaust in the air, which corresponds to an excess cancer risk level of:

- 4 deaths per 1,000 for 40 years of occupational exposure, equals to 1.03 µg EC/m^3,
- And 4 deaths per 100,000 for 40 years of occupational exposure, equals to 0.011 µg EC/m^3.

Since the estimated HBC-OCRV of 1.03 µg EC/m^3 falls in the range of the ambient urban air levels (0.4 – 2.0 µg EC/m^3), and the HBC-OCRV of 0.011 µg EC/m^3 is even far below these levels, DECOS recommends that workers should not be exposed to diesel engine exhaust at levels higher than the background levels.

Given the ubiquitous exposure to diesel exhaust among Ontario workers, and given the MLTSD strategic plan to reduce occupational disease, it is recommended that the MLTSD set a longer term target (perhaps over 5 - 10 years) to reduce diesel exhaust particulate matter to in order to greatly reduce the unacceptably high attributable fraction of lung and bladder cancer among Ontario workers. OHCOW agrees with the OCRC burden of cancer report. OHCOW also agrees that in the mid-term (over 5 years) that the following policy recommendation by OCRC be adopted "**Adopt occupational exposure limits of 20 µg/m^3 elemental carbon for the mining industry and 5 µg/m^3 elemental carbon for other workplaces" OCRC 2017**. http://www.occupationalcancer.ca/2017/news-occupational-burden-ontario-report/

In addition, the longer term strategy should drive exposures below background < 1.0 µg EC/m^3 and notwithstanding a lower TWA based on elemental carbon (EC) should be promulgated. OHCOW acknowledges that it will take some time before the above mentioned targets are achieved. As part of this submission and in line with the recommendation for the changes brought about from the current submission:

To drive harmonization, and ensure that there is international alignment the following is highly recommended:

- For new technology with significantly reduced diesel engine exhaust and elemental carbon mass concentrations, elemental carbon may not be an equally useful exposure indicator. Nitrogen dioxide is likely to be a more relevant exposure indicator for new technology diesel engine exhaust. Since the age and type of engines and exhaust after-treatment systems applied vary within and between workplaces, it may be appropriate to

set an occupational exposure limit value for diesel exhaust both as respirable elemental carbon and as nitrogen dioxide.

In summary, OHCOW concurs, in the interim, with the OCRC recommendation of the Finnish TWA be adopted in Ontario for EC TWA: **0.020 mg/m^3 for mining operations and 0.005 mg/m^3 for non-mining exposures**. But we also note (with the OCRC) that ambient levels of diesel exhaust from old diesel technology carries an unacceptable risk, we also draw attention to a number of international recommendations to ban old technology diesel engines in recognition that they will not be able to achieve a level of risk traditionally associated with TWAs. We also recognize that a leading mining company has instituted a goal of trying to reduce exposure levels to as low as reasonably achievable (ALARA) with a view of achieving at least EC TWA of 0.030 mg/m^3 for mining operations. Given the disruptive nature such an TWA might have particularly in the mining sector, OHCOW suggests a graduated approach to the lowering of the TWA for mining for diesel exhaust with a long term goal of eliminating old diesel technology, and ultimately reducing *all* Ontario exposures to diesel exhaust to below EC TWA of 0.001 mgm^3.

1. OCRC (2017) Burden of Occupational Cancer in Ontario.
2. ACGIH, 2002 TLVs and BEIs, ACGIH, Cincinnati, OH (2002).
2.ACGIH, 2003 TLVs and BEIs, ACGIH, Cincinnati, OH (2003).
3.National Institute for Occupational Safety and Health (NIOSH), "Current Intelligence Bulletin 50: Carcinogenic Effects of Exposure to Diesel Exhaust", NIOSH, Cincinnati, OH, Aug/88.
4.EPA, Health Assessment Document for Diesel Emissions, EPA/600/8-90/057F, Washington, DC, May 2002.
5.Zabest, D.D., D.E. Clapp, L.M. Blade, D.A. Marlow, K. Steenland, R.W. Hornung, D. Scheutzle, and J. Butler, "Quantitative Determination of Trucking Industry Workers' Exposures to Diesel Exhaust Particles", American Industrial Hygiene Journal 52:529-541 (1991).
6.Steenland, K., J. Deddens and L. Stayner, "Diesel Exhaust and Lung Cancer in the Trucking Industry: Exposure-Response Analyses and Risk Assessment", American Journal of Industrial Medicine 34:220-228 (1998)
7. Silverman D.T., C.M. Samanic, J.H. Lubin, et al., "The Diesel Exhaust in Miners Study: A Nested Case – Control Study of Lung Cancer and Diesel Exhaust", Journal of the National Cancer Institute 104:855-868 (2012)
8. Vermeulen R., D.T. Silverman, E. Garshick, et al., "Exposure-Response Estimates for Diesel Engine Exhaust and Lung Cancer. Mortality Based on Data from Three Occupational Cohorts", Environmental Health Perspectives 122:172–177 (2014).
9. Park, R.M., "Diesel Engine Emissions and Risk Assessment at NIOSH", presentation at the Health Effects Institute – Boston MA – March 6, 2014
10. Kim J., C.E. Peters, C. McLeod, S. Hutchings, L. Rushton, M. Pahwa1, and P.A. Demers1,4 "Burden of cancer attributable to occupational diesel engine exhaust exposure in Canada", Occup Environ Med 2014;71:A37
11. Taxell, P and Santonen, T (2017) Diesel Engine Exhaust: Basis for Occupational Exposure Limit. TOXICOLOGICAL SCIENCES, 158(2), 2017, 243–251
12. Vermeulen (2017) "Is diesel equipment in the workplace safe or not? A quantitative risk assessment for diesel engine exhaust", https://www.occupationalcancer.ca/2017/special-oeh-seminar-is-diesel-equipment-in-the-workplace-safe-or-not/

Substitution (Sensitizers, Carcinogens and Reproductive Hazards in the Workplace) (2018):

Workers' 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 hierarchy of controls deems that the best way to prevent exposures is at the source[(1)]. Any workplace where sensitizers, carcinogens and/or reproductive hazardous substances are used should be required to demonstrate, on a regular basis, that it is actively involved in an ongoing process to identify alternative 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[(2)], that exposures are "as low as reasonably achievable" (ALARA) 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 Environment's 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. Thus, OHCOW fully endorses the inclusion of the adding the mention of substitution in the list of controls in section 3 of O.Reg 833.

1. AIHA Exposure Assessment Strategies Committee, A Strategy for Assessing and Managing Occupational Exposures: Fourth Edition, ed. S.D. Jahn, W.H. Bullock, and J.S. Ignacio, AIHA Press, Fairfax VA (2015).
2. RTWAofs, C., Preventing Hazards at the Source, AIHA Press, Fairfax VA (2007).

Additional Substances Recommended for Improved TWAs:

Nanoparticles and Carbon Nanotubes

NIOSH has recently recommended[(1)] an REL for carbon nanotubes (CNT) and nanofibers (CNF) of 1 μg/m^3, TWA, for a 45 year working life. Included in the recommendations were provisions for measuring CNTs/CNFs 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 MLTSD's treatment of polymeric isocyanates when it was established that there were no valid methods of measuring airborne polymeric isocyanates – in response to this situation, the MLTSD 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 TWAs the MLTSD 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 CNTs/CNFs, OHCOW recommends that the MLTSD adopt a 1 $\mu g/m^3$ for these substances.

1. NIOSH, "Current Intelligence Bulletin 65 - Occupational Exposure to Carbon Nanotubes and Nanofibers", 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., "Occupational safety and health criteria for responsible development of nanotechnology", 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, Queen's Printer for Ontario, Toronto, 1987.

Styrene

In 1996 the ACGIH adopted a TLV of 20 ppm (TWA) and 40 ppm STEL for styrene. The current Ontario TWA is 35 ppm (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:

> "The 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." (p.78)

Accordingly, OHCOW recommends the MLTSD adopt an TWA of **10 ppm** for styrene.

1. *ACGIH,* Formaldehyde: TLV Chemical Substances 7th Edition *Documentation* Publication (2014).
2. Swedish Criteria Group for Occupational Standards, "Scientific Basis for Swedish Occupational Standards XXX" 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 TWA for periods of less than 2 hours (a modified STEL?), we agree with the need for a more protective TWA for ozone that is graduated for the level of physical activity. We understand that the MLTSD is reluctant to adopt this ACGIH TWA 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 TWA). 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 MLTSD suggestion that an TWA should not be adopted if the ambient air quality conditions might on occasion exceed the TWA. 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).

Thus OHCOW recommends Ontario adopt TWAs: heavy work - 0.05 ppm, moderate work - 0.08 ppm, light work - 0.10 ppm.

Particulates Not Otherwise Classified (PNOCs)

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 TWA, 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 TWA (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 TWA for respirable PNOCs, 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 TWA. This paper suggests that a significant decrease in forced vital capacity (FVC) is associated with exposures to total suspended particulate 121 µg/m^3 (i.e. 0.121 mg/m^3) and suggested the threshold for this health effect was at a level of 60 µg/m^3 (i.e. 0.06 mg/m^3). It should be emphasized that these dust measurements include materials other than insoluble mineral dust. It should also be noted that these levels are total dust concentrations. These findings have since been corroborated by numerous other studies[7] of ambient particulate and various health parameters.

An occupational epidemiological study related to this issue was published by N.S. Seixas et al.[8], in which they reviewed the exposure of coal miners to respirable coal dust since 1970. The authors found a significant association of obstructive lung disease with cumulative respirable dust exposures of 20 mg/m^3-years or more. Assuming a 45 year working life, this cumulative respirable dust exposure would translate into a 0.44 mg/m^3 average lifetime exposure after which a significant health effect would be expected. Again it should be noted that coal dust is not considered a "nuisance" dust due to its silica content. However, it does seem to corroborate well with the animal study-based TWA 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 TWA for PNOCs be lowered, stating:

> "…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." (p.685)

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

Furthermore, in 2012 Health Canada published a "Guidance for Fine Particulate Matter ($PM_{2.5}$) in Residential Indoor Air" (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:

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

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

1. Mermelstein, R. and R.W. Kilpper, "Xerox Exposure Limit for Respirable Dust (N.O.S.)", Xerox Corp., Webster NY, Presented at the 1990 American Industrial Hygiene Conference.
2. Mermelstein, R. and R.W. Kilpper, "Recent Chronic Inhalation Study Results and Their Implications on the Respirable Dust Standard", 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, "Chronic Inhalation Study Findings as a Basis for Proposing a New Occupational Dust Exposure Limit", Journal of the American College of Toxicology 10: 279-290 (1991).
4. Woskie, S.R., "Issues in Assessing and Regulating Particulate Exposures in Occupational Environments", Applied Occupational and Environmental Hygiene 13:593-599 (1998).
5. Pritchard, J.N., "Dust Overloading - A Case for Lowering the TLV of Nuisance Dust?", Journal of Aerosol Science 20: 1341-1344 (1989).
6. Chestnut, L.G., J. Schwartz, D.A. Savitz and C.M. Burchfiel, "Pulmonary Function and Ambient Particulate Matter: Epidemiological Evidence from NHANES I", Archives of Environmental Health 46: 135-144 (1991).
7. Daniels, M.J., F. Dominici, J.M. Samet and S.L. Zeger, "Estimating Particulate Matter-Mortality Dose-Response Curves and Threshold Levels: An Analysis of Daily Time-Series for the 20 Largest US Cities", American Journal of Epidemiology 152:397-406 (2000).
8. Seixas, N.L., "Exposure-Response Relationships for Coal Mine Dust and Obstructive Lung Disease Following Enactment of the Federal Coal Mine Health and Safety Act of 1969", American Journal of Industrial Medicine 21:715-734 (1992).
9. Rödelsperger, K. and M. Roller, "Pulmonary carcinogenicity of granular bio-durable particles without significant specific toxicity (GBP): relevance for occupational safety", in: BIA Report 7/2003e – BIA-Workshop "Ultrafine aerosols at workplaces", HVBG, Sankt Augustin, Germany 2004 (pp. 181-199).
10. Cherrie, J.W., L.M. Brosseau, A. Hay, and K. Donaldson, "Low-Toxicity Dusts: Current Exposure Guidelines Are Not Sufficiently Protective", Annals Occupational Hygiene 57:685–691 (2013).

Metalworking Fluids

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

The Health Council of the Netherlands[9] has recommended a health based occupational exposure limit for metalworking fluids containing mineral oils of 0.1 mg/m^3. Given the current Ontario TWA of 5 mg/m^3 (although it appears that this TWA excludes MWFs), 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 health-based DECOS (Dutch Expert Committee on Occupational Safety) recommendation[9] of **0.1 mg/m^3** for mineral oil in metalworking fluids, if not, then at least the NIOSH (GM/UNIFOR) TWA of **0.5 mg/m^3**.

1. Kennedy, S.M., I.A. Greaves, D. Kriebel, E.A. Eisen, T.J. Smith and S. Woskie, "Acute Pulmonary Responses Among Automobile Workers Exposed to Aerosols of Machining Fluids", American Journal of Industrial Medicine 15: 627-641 (1989).
2. Kriebel, D, S.R. Sama, S. Woskie,D.C. Christiani, E.A. Eisen, S.K. Hammond, D. Milton, M. Smith and M.A. Virji, "A Field Investigation of the Acute Respiratory Effects if Metalworking Fluids. I. Effects of Aerosol Exposures", American Journal of Industrial Medicine 31: 756-766 (1997)
3. Robins, T., N. Seixas, A. Franzblau, L. Abrams, S. Minick, H. Burge and M.A. Schork, "Acute Respiratory Effects on Workers Exposed to Metalworking Fluid Aerosols in an Automotive Transmission Plant", American Journal of Industrial Medicine 31: 510-524 (1997)
4. Kennedy, S.M., M. Chan-Yeung, K. Teschke and B. Karlen, "Change in Airway Responsiveness among Apprentices Exposed to Metalworking Fluids", American Journal of Respiratory Critical Care Medicine 159: 87-93 (1999).
5. Rosenman, K.D., M.J. Reillly and D. Kalinowski, "Work-Related Asthma and Respiratory Symptoms Among Workers Exposed to Metal-Working Fluids", American Journal of Industrial Medicine 32: 325-331 (1997).
6. Eisen, E.A., T.J. Smith, D. Kriebel, S. Woskie, D.J. Myers, S.M. Kennedy, S. Shalat and R.R. Monson, "Respiratory Health of Automobile Workers and Exposures to Metal-Working Fluid Aerosols: Lung Spirometry", American Journal of Industrial Medicine 39: 443-453 (2001).
7. Oudyk, J.D., A.T. Haines and J. D'Arcy, "Investigating Respiratory Responses to Metalworking Fluid Exposure", Applied Occupational and Environmental Hygiene 18: 939-946 (2003).
8. NIOSH, Criteria for a Recommended Standard: Occupational Exposure to Metalworking Fluids, DHHS (NIOSH) Publication No. 98-102, Cincinnati, OH (1998)
9. Health Council of the Netherlands. Aerosols of mineral oils and metalworking fluids (containing mineral oils). Health-based recommended occupational exposure limits. The Hague: Health Council of the Netherlands, 2011; publication no. 2011/12.

Stoddard Solvent

The Ontario TWA TWA for Stoddard Solvent is 100 ppm, however in Europe, the Scientific Committee on Occupational Exposure Limits (SCTWA) has adopted an TWA 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 SCTWA 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 TWA of 20 ppm).

A 2-year National Toxicology Program (NTP) animal study (2004 – see: http://ntp.niehs.nih.gov/ntp/htdocs/lt_rpts/tr519.pdf) found that there was "*some evidence*" that Stoddard Solvent (CAS # 64742-88-7) caused a very specific type of adrenal gland tumour only in male rats and, also, there was "equivocal evidence" 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 – 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 TWA for Stoddard Solvent be reduced to no more than TWA of **20 ppm** and STEL of **50 ppm**.

- SCTWA, "Recommendation of the Scientific Committee on Occupational Exposure Limits for "White Spirit"", SCTWA/SUM/87, August 2007.
- Brautbar, N., "Industrial Solvents and Kidney Disease", Int J Occup Environ Health 10:79–83 (2004).
- NTP, NTP Technical Report on the Toxicology and Carcinogenesis Studies of Stoddard Solvent IIC (CAS No. 64742-88-7) in F344/N Rats and B6C3F$_1$ Mice (Inhalation Studies), September 2004.

Dimethylamine

The change in the ACGIH TLV for dimethylamine (DMA) is simply the adding of the "DSEN" notation to the current TWA of 5 ppm (TWA) and 15 ppm (STEL) and thus it was not included in the MLTSD proposals even though it was listed as a change for the 2014 TLVs. 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 ACGIH TLV, was referenced as providing a NTWA (no observed effect level) of 10 ppm, however, there was a mild effect observed and therefore this should have been called a LTWA (lowest observed effect level) as per the interpretations of the documentations.
2. The ACGIH applied an uncertainty factor of 2 to the CIIT "NTWA", whereas the other documentations use the same study and apply a 5-10 fold uncertainty factor on the basis of the CIIT "LTWA"

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 NTWA whereas other documentation recognize this appropriately as a LTWA.
4. There were errors in referencing outdated TWAs for analogous amines (the documentation assumes erroneously that the TLVs for methylamine and ethylamine are 10 ppm); furthermore, these are both primary amines, not secondary amines – should tertiary amines (e.g. triethylamine) also be cited?

In contrast to the ACGIH 2014 documentation, the SCTWA 1991 documentation using the same CIIT, 1990 study derives a 2 ppm TWA TWA (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 LTWA).

Similarly, despite many of the agencies' documentations concurring with the ACGIH's interpretation of the Coon et al., 1970 study as being an NTWA, the AEGL 2008 documentation questions the authors' (Coon et al., 1970) conclusion that: "specific chemically induced histopathological changes were not noted.". 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 TWA, based on an LOAEL of 5-10 ppm (CIIT, 1990, and, Coon et al., 1970) an equivalent TWA of 0.1-0.2 ppm (without uncertainty factors applied) can be derived. If one were to apply the uncertainty factors applied by the SCTWA then the TWA adjusted for a 5 fold uncertainty factor would be: 0.02-0.04 ppm.

The German MAK 1993 documentation follows the SCTWA 1991 lead in using the CIIT 1990 study to establish a 2 ppm MAK (essentially concurring with the SCTWA uncertainty factor of 5) but they note that the TWA "requires substantiation from experience of human exposures". In a similar vein, the ACGIH 2014 documentation states "It should be noted that at the TLV concentration, the odour of dimethylamine may be sufficiently unpleasant to make working under those conditions not possible." 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 NTWA of 0.5 ppm (which the 1986 ACGIH Documentation cites as the TWA 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 "not possible", we would recommend that the TWA be lowered from 5 ppm to at least 0.5 ppm. Furthermore, using the IRIS RfC methodology (but using the SCTWA uncertainty factor of 5), one would derive an equivalent occupational TWA of 0.02-0.04 ppm. We agree with the addition of the "DSEN" 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 "halo", or "foggy" vision disturbances.

1. ACGIH. (2014). Dimethylamine. Documentation of the Threshold Limit Values and Biological Exposure Indices. American Conference of Governmental Industrial Hygienists. Cincinnati. 2014.
2. Chemical Industry Institute of Toxicology (CIIT): Twenty Four Month Final Report, Inhalation Toxicity of Dimethylamine in F-344 Rats and B6C3F1 Mice and Third Party Audit Report Summary. Docket #11957. CIIT, Research Triangle Park, NC (1990).
3. Coon RA; Jones RA; Jenkins LJ Jr; et al.: Animal inhalation studies on ammonia, ethylene glycol, formaldehyde, dimethylamine, and ethanol. Toxicol Appl Pharmacol 16:646–665 (1970).
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Gasoline Exhaust (GE) TWA and Control Plan

GE is a complex mixture of variety of different constituents in the form of particulate matter and gases. Some of the constituents of GE such as CO, NO, PAHs, volatile organic compounds (benzene, toluene, xylene, ethylene), and formaldehyde are well known for their adverse health effects and also regulated in different jurisdictions. However, GE as a mixture is not regulated since there is no established occupational exposure limit in any of the jurisdictions. This is mainly due to scarcity of scientific literature on GE exposure and its adverse health effects on humans. The health effect which has been studied extensively is the carcinogenic effect by different organizations such as IARC and Health Canada. Both of these organizations concluded that the GE is not carcinogenic and IARC assigned a classification of 2B (limited evidence of carcinogenicity in humans and less than sufficient evidence of carcinogenicity in animals) to GE. Despite lack of studies with sufficient evidence of respiratory and cardiac effects there are few human studies and animal studies which suggest that the GE exposure can have an adverse respiratory and cardiac health effect as a mixture.

It should be noted that the GE mixture contains some of the known carcinogens such as benzene, PAHs, and formaldehyde which may act independently in the GE. Each contaminant has a specific occupational exposure limit to prevent its carcinogenic and adverse health effects. Although the concentration of each contaminant in the mixture is low the highly toxic nature of these chemicals warrant preventive measures.

OHCOW hygienists have learned from GE exposure assessments and discussing this issue with JHSC members from variety of different workplaces, that there is a common perception that there are no or negligible adverse health effects from GE exposure since it has been deemed as a less toxic alternate of diesel. Because of this rhetoric GE exposure is not usually controlled similar to diesel exhaust. We think that gasoline, in spite of being a less toxic source of fuel, its exhaust contains some of the known carcinogens, particulate matter (fine and ultrafine particles), and gases (CO and NO) which can have adversely affect the cardiopulmonary system. In the

light of individual constituents' toxicity, it would be prudent to prevent GE exposure through techniques such as anti-idling policy, engine maintenance program, ventilation etc. by developing a control program similar to diesel exhaust control program.

There are various chemicals in the GE which can be measured to estimate GE exposure, however, CO is considered as a better surrogate due to its presence as a major constituent of its gaseous phase and its ability to vary in concentration in relation to other constituents. However, exact co-relation between CO and other contaminants is not well established.

OHCOW recommends to prevent workers from GE exposure's adverse health effects by enforcing a **GE control program similar** to a diesel control program (OCRC has developed a diesel control program for mining as an example) since a control program is a more holistic approach in controlling a hazard in the absence of an occupational exposure limit in particular.

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