OSHA Permissible Exposure Limits (PELs) are too Permissive.

by

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KEYWORDS

Permissible Exposure Limits; Exposure Limits; No-adverse-effects-exposure-limits; Minimal Risk Levels; NOAEL; OSHA; NIOSH; ACGIH; ATSDR
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The present monograph examines the differences (for selected toxic substances) between 1) the Federal legally enforceable occupational Permissible Exposure Limits (PELs) set by OSHA and 2) low-level exposures reported in the biomedical literature associated with serious adverse effects. In these selected cases, the PELs are orders of magnitude higher than what the premier biomedical literature would suggest is protective.

Our previous monograph [1] concluded that testing of single stressors (the main determinant of myriad types of Exposure Limits), rather than combinations of stressors, greatly under-estimates the toxicity of the stressors in real-world environments. When these 1) stressor combination conclusions are added to the 2) results from the present monograph, one can 3) seriously question whether present-day Exposure Limit regulations offer credible levels of occupational protection from ANY potentially toxic stressors.
INTRODUCTION

Background

A recent monograph [1] concluded there is little reason to believe that Exposure Limits on potentially toxic stimuli that have been set by ANY of the regulatory agencies are fully protective against serious adverse health effects. Existing Exposure Limits are based mainly on the results of single stressor experiments, whereas real-world exposures involve combinations of stressors. These combinations 1) typically lower the threshold constituent exposure levels associated with damage compared to 2) tests of combination constituents run in isolation. The enormous number of combinations possible for any stressor precludes testing more than a handful, and this under-representation of combination effects results in Exposure Limits that could greatly under-reflect the risks from any stressor.

Relation of Biomedical Literature Findings to Setting of Exposure Limits

In addition to uncertainties about Exposure Limits' adequacy based on minimal testing of stressor combinations, there are uncertainties stemming from how well

- existing data suffice for setting exposure limits
- existing data in the biomedical literature relevant to setting of Exposure Limits are fully taken into account, and
- existing data in the biomedical literature can be trusted for accuracy.

Consider the case of non-ionizing radiation, especially radiofrequency radiation (RFR). The Bioinitiative Report [2] and the recent Pall study [3] present copious examples of myriad types of adverse health impacts from athermal (non-heating) non-ionizing radiation, ranging from moderate to life-threatening. Yet, these athermal impacts are ignored completely by the FCC guidelines. For RFR, the FCC Exposure Limits (based on heating of tissue) are approximately six orders of magnitude above RFR exposures shown to cause adverse health effects from single and multi-stressor studies. For all practical purposes, the FCC guidelines are non-protective for athermal exposures!

Is the FCC's setting of RFR Exposure Limits many orders of magnitude above levels required for protection a unique case, or does it happen far more frequently than the public realizes? Answering that question, at least in part, will be the major focus of the present monograph.

Appendix 1 (taken from [1]) contains another example of experimental data not being taken into account for setting Exposure Limits, that of water fluoridation. According to the statements of a senior EPA toxicologist (who was fired for his remarks, then later re-instated), data from a single stressor experiment (which showed many instances of cancer associated with water fluoridation) were downgraded substantially from a cancer determination by the government research program manager.

References [4,5], from which some of the data for the Appendices were taken, provide other examples of data manipulation that could impact setting of Exposure Limits. There is little reason to believe that the above examples of discrepancies between 1) results from single and multi-stressor experiments already conducted and 2) Exposure Limits set eventually by regulatory agencies are rare, especially for substances that are commercially, militarily, or politically sensitive.
Types of Exposure Limits

There are myriad types of toxic substance Exposure Limits that have been published. Each country has its own lists of Exposure Limits, with at least one of the lists in each country being enforceable.

In the USA, the only legally enforceable occupational Exposure Limits at the Federal level are set by OSHA, and are called Permissible Exposure Limits (PELs) [6].

The National Institute for Occupational Safety and Health (NIOSH) has developed a set of Recommended Exposure Limits (RELs), and those RELs overlapping with PELs are listed in [6].

The American Conference of Governmental Industrial Hygienists (ACGIH) has developed sets of Exposure Limits called Threshold Limit Values (TLVs) and Biological Exposure Indices (BEIs), and those TLVs overlapping with PELs are listed in [6]. Most OSHA PELs were grandfathered in as initial OSHA PELs and came from the 1968 list of TLVs.

The Environmental Protection Agency (EPA) has developed sets of Exposure Limits called Inhalation Reference Concentrations (RfCs) and Oral Reference Doses (RfDs), and the Agency for Toxic Substances and Disease Registry (ATSDR) has developed Exposure Limits called Minimal Risk Levels (MRLs).

In addition, some of the States in the USA (e.g., CA) have set their own (usually more stringent than Federal) Exposure Limits.

The PELs and RELs are targeted for working adults, and exposures tend to focus on eight-hour day exposures. The RfCs, RfDs, and MRLs are targeted for the broader population, can include children as well as adults, and their most stringent cases are for 24 hour day exposures for seventy years. The differences among these myriad Exposure Limits are described in some detail in Appendix 4 (Definitions), and the above paragraphs are hyperlinked to the relevant sections of Appendix 4.

Structure of Monograph

The main focus of the remainder of this monograph is to examine selected cases of PELs, and relate them to exposures shown in the biomedical literature to result in serious adverse effects.

The next section (ANALYSIS) summarizes the methodology and selection strategy for the substances analyzed. The following section (RESULTS AND DISCUSSION) examines the results, and the subsequent section (CONCLUSIONS) draws conclusions from the study.

The REFERENCES follow, and are themselves followed by the APPENDICES. The monograph ends with an extensive BIBLIOGRAPHY of Medline records that show health effects from exposures to the OSHA chemicals analyzed.
ANALYSIS

Overview

The goal of this study is to determine the gap between 1) toxic substance *occupational* Exposure Limits set by regulatory agencies and 2) *occupational* Exposure Limits that would fully reflect research reported in the premier biomedical literature.

There are thousands of toxic substances (including radiation types) that have been reported in the biomedical literature. Reference [5] lists ~8,000 substances that are contributing factors to disease, with perhaps 800 of those being pervasive (contributing to at least some threshold number of diseases). It was acknowledged in [6] that *many* more contributing factors could have been identified.

Each of these potentially toxic substances is associated with many published papers, in some cases perhaps thousands of papers. Clearly, only a minute fraction of these total toxic substances can be examined to identify the gaps between 1) the regulatory Exposure Limits on these toxic substances and 2) what the biomedical literature suggests should be protective Exposure Limits.

Substance Selection Strategy

Therefore, a strategy for selecting substances for analysis in this study is required. This substance selection strategy starts by randomly selecting a few toxic substances on the OSHA list of PELs. Then, a survey is performed of the research reported in the biomedical literature related to exposures and adverse effects for each of the selected substances. A query of the form

(substance name) AND (*toxic* OR expos*)

was used to retrieve documents from Medline. Obviously, a more complex query could have retrieved more records. The retrieval was supplemented by references in, and citations to, the retrieved documents, where warranted.

To limit the number of substances evaluated, a dynamic sampling strategy was employed. Substances were selected randomly (as stated above), and results were generated for each substance. When a pattern of results for the substances analyzed began to emerge, the selection process was terminated.

Those substances for which

- the biomedical literature identified Exposure Limits at least an order of magnitude less than ANY of those listed for the substance in [6], and
- the papers presenting these low Exposure Limits were judged to be credible

are shown in Table 1.
<table>
<thead>
<tr>
<th></th>
<th>REGULATORY</th>
<th>RECOMMENDED</th>
<th>BIOMEDICAL LITERATURE</th>
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</thead>
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<td>CAL/NIOSH/ACGIH</td>
<td></td>
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Table 1 has nine columns. Column 1 shows the toxic substance being analyzed. Columns 2 and 3 contain the OSHA PELs, the legally enforceable Federal occupational Exposure Limits. Column 2 presents the PEL in parts per million (ppm), and Column 3 presents the PEL in mg/m^3. The PELs are typically 8-hour time weighted averages (TWAs).

Columns 4-6 contain recommended Exposure Limits by the three organizations shown alongside the OSHA list of PELs [6]. Column 4 contains the California Division of Occupational Safety and Health (CAL/OSHA) Permissible Exposure Limits (PELs), which are enforced in workplaces under its jurisdiction. Column 5 contains the National Institute for Occupational Safety and Health (NIOSH) Recommended Exposure Limits (RELs), which are for up to 10-hour time-weighted averages (TWAs) during a 40-hour work week unless otherwise indicated. Column 6 contains the ACGIH Threshold Limit Values (TLVs).

In Columns 4-6, three types of exposures are possible, although values for each type are not always provided. They are TWA (essentially the average Exposure Limit for a work day), ST (short-term exposure limits, essentially the average Exposure Limit over a fifteen-minute period), and C (essentially the ceiling, or maximum Exposure Limit over any period of time). The default entry is the TWA.

Columns 7-9 contain Exposure Limits suggested in papers from the premier biomedical literature. These limits were not always expressed in the units of the OSHA PELs, so they were converted to the OSHA PEL units where possible.
RESULTS AND DISCUSSION

Each of the substances listed in Table 1 will be discussed briefly, and the gap between the Federal legally enforceable occupational PELs and the Exposure Limits suggested by the biomedical literature will be emphasized.

R-1. Acetaldehyde

Acetaldehyde is a chemical compound found widely in foods and nature, and produced in industry. It is listed by IARC as a Group 1 carcinogen.

The OSHA PEL values shown in Table 1 are 200 ppm (360 mg/m$^3$), and the other agency/organization Exposure Limits listed in Columns 4-6 of Table 1 are about an order of magnitude less.

Three references that showed serious adverse effects at low exposures were extracted from the biomedical literature:

- Reference [7] showed that acetaldehyde exposure of 10.5 ppb led to formation of DNA adducts in the lungs and brains of rats, which could be an important factor in the mechanism of mutagenesis and carcinogenesis.
- Reference [8] used a model of acetaldehyde pharmacokinetics in the nose to derive an inhalation reference concentration (RfC) of 0.4 ppm based on animal experiments.
- Reference [9] started with the results of credible rat studies, then applied an interspecies factor of 1, a factor of 10 for inter-individual variability, and a factor of 2 to account for the higher respiratory rate of children compared to adults, to generate a health hazard guide value (RW II) of 1 mg acetaldehyde/m$^3$. The authors of [9] recommended a health precaution guide value (RW I) of 0.1 mg acetaldehyde/m$^3$.

Thus, the acetaldehyde Exposure Limits suggested by the premier biomedical literature are about three-four orders of magnitude more stringent than the OSHA PELs, and are two-three orders of magnitude more stringent than the other acetaldehyde Exposure Limits in Columns 4-6 of Table 1.

R-2. Trichloroethylene

Trichloroethylene is an industrial solvent of the halocarbon class that has been characterized by the EPA as a carcinogen. The OSHA PEL values shown in Table 1 are 100 ppm (537 mg/m$^3$), and the other Exposure Limits listed in Columns 4-6 of Table 1 are about an order of magnitude less.

Three references that showed serious adverse effects at low exposures were extracted from the biomedical literature:

- Metabolism investigation of trichloroethylene (TCE) in the early embryo showed strong upregulation of CYP2H1 transcripts in response to 8 ppb TCE exposure in the heart. Immunostaining for CYP2C subfamily expression confirmed protein expression and showed
localization in both myocardium and endothelium. TCE exposure increased protein expression in both tissues [10].

- Chick embryos exposed to 8 ppb TCE that survived to hatch exhibited a high incidence of muscular ventricular septal defects [11].
- "The Ad-hoc Working Group on Indoor Guidelines recommends 0.02mg trichloroethylene/m(3) (~4 ppb) as a risk-related guideline for indoor air" [12].

Thus, the trichloroethylene Exposure Limits suggested by the premier biomedical literature are about four orders of magnitude more stringent than the OSHA PELs, and are about three orders of magnitude more stringent than the other trichloroethylene Exposure Limits in Columns 4-6 of Table 1.

R-3. Naphthalene

Naphthalene is a polycyclic aromatic hydrocarbon that has been classified by the IARC as a possible carcinogen (Group 2B). The OSHA PEL values shown in Table 1 are 10 ppm (50 mg/m³); the CAL/OSHA value shown in Table 1 is two orders of magnitude less.

Three references that showed serious adverse effects at low exposures were extracted from the biomedical literature:

- "the Ad-hoc Working Group derived a chronic NAEC of 2.5 mg naphthalene/m(3). Time scaling was considered by a factor of 5.6 extrapolating from 6 to 24 h and 5 to 7 days, a factor of 2 applied for the use of F344 rats instead of the more sensitive Sprague-Dawley rats. Incorporating
  - an interspecies factor of 1,
  - an intraspecies factor of 10 and
  - a factor of 2 for insufficient data on the toxicity of naphthalene in children
resulted in a precautionary value of 0.01 mg naphthalene/m(3) and a hazard-based guide value of 0.03 mg naphthalene/m(3)" [13].
- "Severe effects in terms of inflammation were observed in almost all rats exposed to the lowest (but still relatively high) naphthalene dose of 53 mg/m3.....this can be taken as a LOAEL, even though it is related to severe effects. Taking this LOAEL as a starting point and adjusting for continuous exposure (dividing by a factor of 24/6 and 7/5), a value of about 10 mg/m3 is obtained. Further, incorporating
  - a factor of 10 for using a LOAEL rather than a NOAEL,
  - a factor of 10 for inter-species variation and
  - a factor of 10 for inter-individual variation,
a guideline value of 0.01 mg/m3 is established." [14]
- "Based on the consideration of this literature and based on our direct experience investigation health complaints due to workplace exposures to naphthalene OHCOW recommends that the OEL for naphthalene be lowered to a level below 0.5 ppm and preferably 0.01 ppm." [15].

Thus, the naphthalene Exposure Limits suggested by the premier biomedical literature are about two-three orders of magnitude more stringent than the OSHA PELs, and are about one order of magnitude more stringent than the CAL/OSHA Exposure Limit in Column 4 of Table 1.
R-4. Dimethylbenzene (xylene)

Dimethylbenzene is a volatile liquid obtained from coal tar, and xylene is one or more of the three isomers of dimethylbenzene. The OSHA PEL values shown in Table 1 are 100 ppm (435 mg/m³), identical to those of the three other organizations in Columns 4-6 of Table 1.

Three references that showed serious adverse effects at low exposures were extracted from the biomedical literature.

- The Agency for Toxic Substances and Disease Registry (ATSDR) provides MRLs (Minimum Risk Levels) for hazardous substances. An MRL is defined as "an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse non-cancer health effects over a specified duration of exposure." [16]. Reference [17] lists three inhalation MRLs for xylene based on three separate studies, ranging from 0.05-2 ppm.
- "Surveys were conducted in factories in China where workers were engaged in the production of rubber boots or plastic-coated wire or in printing work, and were exposed to xylene vapors....the sum of the three isomer concentrations was 14 ppm as a geometric mean and 21 ppm as an arithmetic mean....There was an increased prevalence of subjective symptoms in the exposed workers which were apparently related to the effects on the central nervous system and to the local effects on the eyes, the nose, and the throat" [18].
- "Conversion of experimental to continuous exposure leads to a LOAEC of 39 mg xylene/m(3) for the endpoint neurotoxicity. A health hazard guide value (RW II) of 0.8 mg xylene/m(3) is derived by applying
  - an interspecies factor of 2.5,
  - a factor of 10 for interindividual variability, and
  - a factor of 2 to account for the higher respiratory rate of children compared to adults.
A health precaution guide value of 0.1 mg xylene/m(3) is recommended." [19].

Thus, the dimethylbenzene Exposure Limits suggested by the premier biomedical literature are about three orders of magnitude more stringent than both the OSHA PELs and the recommendations of the other organizations in Columns 4-6 of Table 1.

R-5. Carbon Disulfide

Carbon disulfide is a highly toxic inorganic solvent. The OSHA PEL value shown in Table 1 is 20 ppm; the other organization values shown in Columns 4-6 of Table 1 are 1 ppm, an order of magnitude less than the OSHA PEL.

Three references that showed serious adverse effects at low exposures were extracted from the biomedical literature.

- "The most sensitive endpoint for CS2 was DNT [Developmental Neurotoxicity].....(lowest NOEL = 0.01 ppm)" [20].
- Based on a thorough literature review, the EPA assigned an inhalation Reference Concentration (RIC) of 0.7 mg/m³ (~0.2 ppm) to carbon disulfide. [21].
Based on a thorough literature review, the ASTDR assigned a Minimal Risk Level (MRL) of 0.3 ppm to carbon disulfide [22].

Thus, the carbon disulfide Exposure Limits suggested by the premier biomedical literature are about two-three orders of magnitude more stringent than the OSHA PELs, and are about one order of magnitude more stringent than the other organization Exposure Limits in Columns 4-6 of Table 1.

Interim Summary

A clear pattern of results emerged from the five substance evaluations above. The Exposure Limits suggested by the biomedical literature were approximately two to four orders of magnitude less than the OSHA PELs, and one to two orders of magnitude less than the limits recommended by the entities in Columns 4-6 of Table 1.

In addition, the lowest Exposure Limits suggested typically were from EPA (Reference concentration for inhalation exposure-RfC, or reference dose for oral exposure-RfD), ATSDR (Minimal Risk Levels-MRL), and the German Working Group on Indoor Guidelines of the Federal Environment Agency (Health Hazard Guide-RW II, Health Precaution Guide-RW I). These three organizations start with an NOAEL or LOAEL, and then divide this number by an Uncertainty Factor to arrive at the final Exposure Limit recommendation.

Thus, the widest gap between the OSHA occupational PELs and the Exposure Limits suggested by the biomedical literature could be identified (for all practical purposes) by considering the results from the latter three organizations alone.

Substances 6-10 on Table 1 are enumerated using recommended Exposure Limits from one or more of these three organizations (not all organizations examined each PEL substance). The pattern that emerged from the analysis of substances 1-5 was applicable to substances 6-10 as well. The results for substances 6-10 will now be summarized.

R-6. Ethyl Acetate

Ethyl Acetate is an organic solvent, and the ester of ethanol and acetic acid. The OSHA PEL values shown in Table 1 are 25 ppm (100 mg/m³), five times the CAL/OSHA and ACGIH amounts.

Two references that showed serious adverse effects at low exposures were extracted from the biomedical literature.

- “This study leads to a lowest observed adverse effect concentration (LOAEC) of 1280 mg ethyl acetate/m(3) indoor air, corresponding to a LAEC for continuous exposure of 230 mg ethyl acetate/m(3), for the endpoint nasal epithelium degeneration. By applying
  o an interspecies factor of 1,
  o a factor of 10 for interindividual variability and
  o a factor of 2 to account for the higher respiratory rate of children compared to adults,
  a health hazard guide value (RW II) of 6 mg ethyl acetate/m(3) is obtained. A health precaution guide value (RW I) of 0.6 mg ethyl acetate/m(3) indoor air is recommended” [23].
• Michigan's Department of Environmental Quality (MDEQ) converted from EPA’s Provisional Peer Reviewed Toxicity Values (PPRTV) to generate a chronic provisional Reference Concentration (p-RfC) of 0.7 mg/m\(^3\). The **Uncertainty Factor** used to derive this p-RfC is 3,000, based on 10 each for 1) interspecies variability, 2) use of a sub chronic study and 3) database deficiencies, and 3 for interspecies extrapolation. [24].

Thus, the ethyl acetate Exposure Limits suggested by the premier biomedical literature are about **two orders of magnitude** more stringent than the OSHA PELs, and are about **one order of magnitude** more stringent than the CAL/OSHA Exposure Limit in Column 4 of Table 1.

**R-7. Ethyl Benzene**

Ethyl Benzene is a flammable aromatic hydrocarbon whose main use is in the production of styrene. The OSHA PEL values shown in Table 1 are 100 ppm (435 mg/m\(^3\)), an **order of magnitude** greater than the CAL/OSHA amounts.

Two references that showed serious adverse effects at low exposures were extracted from the biomedical literature.

• Based on a thorough literature review, the ASTDR assigned a Minimal Risk Level (**MRL**) of 0.06 ppm (chronic exposure) to Ethyl Benzene [25].
• Based on a thorough literature review, the EPA assigned an inhalation Reference Concentration (**RfC**) of 1 mg/m\(^3\) (~0.2 ppm) to Ethyl Benzene. The **uncertainty factor** used (300) reflects a factor of
  o 10 to protect unusually sensitive individuals,
  o 3 to adjust for interspecies conversion and
  o 10 to adjust for the absence of multigenerational reproductive and chronic studies. [26]

Thus, the ethyl benzene Exposure Limits suggested by the premier biomedical literature are about **three orders of magnitude** more stringent than the OSHA PELs, and are about **two orders of magnitude** more stringent than those listed in Columns 4-6 of Table 1.

**R-8. Carbon Tetrachloride**

Carbon Tetrachloride is a manufactured chemical that has been used as a solvent, refrigerant, and cleaning fluid. The OSHA PEL values shown in Table 1 are 10 ppm (63 mg/m\(^3\)), two to five times the amounts shown in Columns 4-6 of Table 1.

Two references that showed serious adverse effects at low exposures were extracted from the biomedical literature.

• Based on a thorough literature review, the ASTDR assigned a Minimal Risk Level (**MRL**) of 0.03 ppm (chronic exposure) to carbon tetrachloride [27].
• Based on a thorough literature review, the EPA assigned an inhalation Reference Concentration (**RfC**) of 0.1 mg/m\(^3\) (~0.02 ppm) to carbon tetrachloride [28].
Thus, the carbon tetrachloride Exposure Limits suggested by the premier biomedical literature are about **two-three orders of magnitude** more stringent than the OSHA PELs, and are about **one order of magnitude** more stringent than the CAL/OSHA Exposure Limit in Column 4 of Table 1.

**R-9. Hydrogen Sulfide**

Hydrogen Sulfide is a foul-smelling gas 1) produced by the anaerobic digestion of organic matter and 2) used frequently in bioconversion to elemental sulfur. The OSHA PEL values shown in Table 1 are (C) 20 ppm (28 mg/m$^3$), an **order of magnitude** higher than the ACGIH amount.

Two references that showed serious adverse effects (e.g., respiratory failure, nasal inflammation) were extracted from the biomedical literature.

- Based on a thorough literature review, the ASTDR assigned a Minimal Risk Level (MRL) of 0.02 ppm (intermediate exposure) to hydrogen sulfide [29].
- Based on a thorough literature review, the EPA assigned an inhalation Reference Concentration (RfC) of 0.002 mg/m$^3$ (~0.0015 ppm) to hydrogen sulfide [30]

Thus, the hydrogen sulfide Exposure Limits suggested by the premier biomedical literature are about **three-four orders of magnitude** more stringent than the OSHA PELs, and are about **two orders of magnitude** more stringent than the ACGIH Exposure Limit in Column 6 of Table 1.

**R-10. Methyl Isobutyl Ketone**

Methyl isobutyl ketone is an organic compound, mainly used as a solvent. The OSHA PEL values shown in Table 1 are 25 ppm (~103 mg/m$^3$), two to five times the amounts shown in Columns 4-6 of Table 1.

Two references that showed serious adverse effects at low exposures were extracted from the biomedical literature.

- Based on a thorough literature review, the EPA assigned an inhalation Reference Concentration (RfC) of 3 mg/m$^3$ (~0.75 ppm) to methyl isobutyl ketone [31]
- "Using a benchmark approach the Working Group assessed a BMDL10 of 57 mg MIBK/m for continuous exposure for the endpoint nephrotoxicity. By applying
  - an interspecies factor of 2.5,
  - a factor of 10 for interindividual variability, and
  - a factor of 2 to account for the higher respiratory rate of children compared to adults,
- a health hazard guide value (RW II) of 1 mg MIBK/m3 indoor air is obtained. A precautionary guide value of 0.1 mg MIBK/m3 indoor air is recommended." [32]

Thus, the methyl isobutyl ketone Exposure Limits suggested by the premier biomedical literature are about **two-three orders of magnitude** more stringent than the OSHA PELs, and are about **one order of magnitude** more stringent than the recommended limits in Columns 4-6 of Table 1.
CONCLUSIONS

OSHA PELs vs other Organization Exposure Limit Recommendations

Ten chemicals were selected randomly from the list of OSHA PELs, and each chemical's PEL was compared with

- recommendations from three organizations (CAL/OSHA, NIOSH, ACGIH) for supposedly protective occupational Exposure Limits
- experiments from the biomedical literature showing adverse effects at low exposures and/or
- comprehensive evaluations of the exposure-adverse events literature by expert panels/organizational teams that resulted in minimal risk recommendations for Exposure Limits.

In general, the OSHA PELs were one-two orders of magnitude greater than the (CAL/OSHA, NIOSH, ACGIH) recommendations, and the OSHA PELs were three-four orders of magnitude greater than the recommendations of the expert panels/organizational teams. Essentially, a vast amount of data is being ignored by the OSHA when they formulate the legally enforceable PELS!

These conclusions apply only to chemicals to which PELs have been assigned. There are fewer than 500 such chemicals, while there are tens of thousands of chemicals to which workers are/could be exposed. Essentially, for those thousands of chemicals not assigned a PEL, the effective occupational Exposure Limits that are Federally enforceable are infinite!

Uncertainty Factors in estimating Exposure Limits

An important analytical reason for the large gaps between the PELs and the recommendations of the expert panels/organizational teams is application of substantial Uncertainty Factors to the experimental results. The Uncertainty Factor serves to (typically) translate laboratory experimental results on animals to relevant results for human beings. In all the records surveyed for the present study, the Uncertainty Factors ranged from about a few tens to a few thousands, depending on the level of data uncertainty and type of extrapolation desired.

What is the Uncertainty Factor, and why is it used? "The uncertainty factor concept is integrated into health risk assessments for all aspects of public health practice.....The use of uncertainty factors is predicated on the assumption that a sufficient reduction in exposure from those at the boundary for the onset of adverse effects will yield a safe exposure level for at least the great majority of the exposed population, including vulnerable subgroups.....Five key uncertainties that are often examined include interspecies variability in response when extrapolating from animal studies to humans, response variability in humans, uncertainty in estimating a no-effect level from a dose where effects were observed, extrapolation from shorter duration studies to a full life-time exposure, and other insufficiencies in the overall health effects database indicating that the most sensitive adverse effect may not have been evaluated. In addition, a modifying factor is used by some organizations to account for other remaining uncertainties—typically related to exposure scenarios or accounting for the interplay among the five areas noted above." [33]
Uncertainty Factor components ignored

While the magnitudes of the different Uncertainty Factor components can be debated, as well as the method of how they should be combined, there are two key components that are missing from most of the Uncertainty Factors examined for the present study. Both of these missing Uncertainty Factor components will 1) increase the magnitude of the Uncertainty Factor substantially and 2) further widen the gap between the PELs and the exposures at which adverse effects can be discerned.

The first of these two components was addressed in a recent monograph [1]. Existing Exposure Limits are based mainly on the results of single stressor experiments, whereas real-world exposures involve combinations of stressors. These combinations 1) typically lower the threshold constituent exposure levels associated with damage compared to 2) tests of combination constituents run in isolation. Thus, an Uncertainty Factor component would have to be included that accounts for the reduction in 'safe' Exposure Limits when extrapolating from single stressor studies to combined stressor studies.

The second of these two components involves uncertainty in the credibility of the data. For topics that are commercially, militarily, or politically sensitive, there are myriad incentives for sponsors and performers of research to enable predetermined agendas [4]. There are strong incentives to downplay and minimize the adverse effects of toxic materials. Appendices 1, 2, and 3 are examples of sponsor manipulation of research data to benefit both government and industry at the expense of the public. In all three of these examples, manipulation of the research results was not the result of a rogue Federal bureaucrat, but rather involved the complicity of high levels of management of the Federal Agency(s) involved.

If an Agency is willing to manipulate results for one toxic material in order to eventually benefit the corporate political donors, what would prevent the Agency from manipulating results for ANY toxic material under its purview?

Identifying data manipulation by either the sponsor and/or the performer is extremely difficult for anyone not involved directly in the research. Even the Gold Standard of research validation, replication by another organization, can be manipulated. If a sponsor funds multiple performers for a given topic, and the sponsor has a predetermined agenda, then there could be pressure on the performers to arrive at the desired results if they want long-term funding to continue.

In the three examples in the Appendices, the information about the data manipulation came from whistleblowers who were willing to sacrifice their careers to make the truth known. Except for these whistleblowers, the information about the data manipulation would never have seen the light of day. There is no reason to believe these three examples are isolated. Given the incentives to downplay adverse effects of toxic materials, and the strong disincentives for bringing the truth to the public, data manipulation may not be uncommon for substances that are commercially, militarily, or politically sensitive.
How do we incorporate these two additional sources of uncertainty (combination effects, data manipulation) into the methodology for extrapolating from the experimental data to real-world impacts on human beings? I don't know. I don't know how combination effects can be estimated without having run large numbers of combinations. I don't know how data manipulation can be estimated in the absence of insiders coming forth and risking their careers and finances to tell the truth.

**The Bottom Line**

The Federal legally enforceable OSHA PELs appear to exceed relatively safe exposure levels by ~3-4 orders of magnitude, depending on toxic substance. The CAL/OSHA, NIOSH, and ACGIH recommended Exposure Limits appear to exceed relatively safe exposure levels by ~1-3 orders of magnitude, depending on substance. And, these supposedly relatively 'safe' exposure levels are estimated 1) ignoring combination effects on initial appearance of adverse effects and 2) assuming that the data has not been manipulated by the sponsor or performer.

In the Introduction, the FCC's RFR Exposure Limits (based on tissue heating only) were estimated to be approximately **six orders of magnitude** above RFR exposures shown to cause adverse health effects from single and multi-stressor studies. It was concluded that for all practical purposes, the FCC guidelines are non-protective for athermal exposures!

We then asked whether the setting of legally enforceable RFR Exposure Limits **six orders of magnitude** above exposures shown to have adverse health effects was a unique case, or does it happen far more frequently than the public realizes? Based on the results of the present study, the RFR example is by no means unique. The difference between the RFR Exposure Limits set by the FCC and the RFR exposure levels where adverse effects start to appear may in fact mirror the discrepancies between the OSHA PELs and the toxic substance exposure levels where adverse effects begin to surface.

We examined ten toxic substances (randomly selected) for the present study. Should we have examined more before drawing the conclusions we did? Given the consistent discrepancies we found between PELs and (relatively) risk-free exposures, we can state: **ONE IS TOO MANY!**
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APPENDICES

Appendix 1 - EPA/NTP Study on Health Effects of Water Fluoridation

This appendix summarizes a single stressor study used as one component in the determination of water fluoridation safety. It derives from reference [4], where the example of EPA’s addressing safety limits of fluoridation (p.19) was presented. The issue was summarized as follows: "Dr. William Marcus was a toxicologist and Senior Science Advisor at EPA. He reported potential cover-up of cancers (by the National Toxicology Program) resulting from fluoride ingestion [34], and was fired in 1992. He challenged this decision in court, and was re-instated."

The cover-up (above) referred to re-stating the results of a study performed by an NTP contractor. Marcus wrote an internal memo describing an NTP contractor review meeting, where every one of the cancers reported by the contractor had been downgraded by the NTP [35].

At his 1995 interview [34], Dr. Marcus described the NTP actions at the review meeting thusly: "Now I’ve been in the toxicology business looking at studies of this nature for nearly 25 years and I’ve never seen that; never ever seen where every single endpoint that was a cancer endpoint had been downgraded.....I found that very suspicious and I went to see an investigator in the Congress at the suggestion of my friend Bob Carton. And this gentleman and his staff investigated very thoroughly and found out that the scientists at the NTP down at Research Triangle Park had been coerced to change their findings."

In Senate testimony that included comments on the NTP final report on the contractor study [36], Dr. William Hirzy stated: "In 1990, the results of the National Toxicology Program cancer bioassay on sodium fluoride were published, the initial findings of which would have ended fluoridation. But a special commission was hastily convened to review the findings, resulting in the salvation of fluoridation through systematic down-grading of the evidence of carcinogenicity. The final, published version of the NTP report says that there is, "equivocal evidence of carcinogenicity in male rats," changed from "clear evidence of carcinogenicity in male rats."

There is no reason to believe that EPA is the only government organization that would manipulate results to achieve a predetermined agenda, or fluorine is the only toxic stimulus for which this was done. In reference [4], similar distortions of results by other Federal regulatory agencies were listed, and many more examples could have been presented had space been available.
Appendix 2 - CDC Study on Health Effects of MMR Vaccine

Background

This example addresses alleged CDC cover-up of an in-house study that showed potential links between 1) increased risk for autism and 2) MMR vaccine timing. A series of allegations from an employee of the Federal agency responsible for monitoring vaccine safety (CDC) suggested there may be a potential link between the MMR vaccine and autism, and documentation of the link had been suppressed deliberately. On August 27, 2014, the following excerpted statement by Dr. William Thompson (a CDC Senior Researcher) appeared on the website of Morgan Verkamp, LLC, a legal organization representing Dr. Thompson [37].

“I regret that my coauthors and I omitted statistically significant information in our 2004 article published in the journal Pediatrics. The omitted data suggested that African American males who received the MMR vaccine before age 36 months were at increased risk for autism. Decisions were made regarding which findings to report after the data were collected, and I believe that the final study protocol was not followed…My concern has been the decision to omit relevant findings in a particular study for a particular sub group for a particular vaccine. There have always been recognized risks for vaccination and I believe it is the responsibility of the CDC to properly convey the risks associated with receipt of those vaccines.”

According to taped phone conversations between Dr. Brian Hooker and Dr. Thompson, not only were the African-American children who received the MMR vaccine at substantially greater risk for autism (as alleged by Dr. Thompson), but according to Dr. Hooker, Dr. Thompson mentioned that children of all races were shown to have an increased risk of ‘Isolated Autism’ (young children, regardless of race, who had (1) received the MMR vaccine on schedule, as recommended by the CDC, and (2) had no other factors sometimes observed to accompany autism, such as cerebral palsy, mental retardation, and birth defects.)

The MMR vaccine-autism study to which Dr. Thompson referred had been performed shortly after the turn of the new millennium, and the stated results (no link between MMR vaccine and autism) had been published in the journal Pediatrics [38]. As of mid-2018, the article has not been retracted by Pediatrics, despite the serious allegations of intentional omission of critical data by one of its co-authors.

Thus, according to Dr. Thompson’s allegations of August 27, 2014, CDC had known for at least a decade that these two groups of children were at increased risk for autism from the MMR vaccine, and did not disclose this information to the public. Internal CDC memos also showed the highest levels of CDC management had been informed of these problems with the MMR vaccine since the early 21st century [39].

Impact

The alleged intentional withholding of the link between the MMR vaccine and autism from the public (for over a decade) resulted in many children (from specific sub-groups) being placed at higher risk for autism. These children could have minimized this risk through either alternative vaccinations, modified vaccination schedules, or no vaccinations, had their parents been informed of the potential increased risk. Thus, there were potentially more children who suffered from autism as a result of the
alleged intentional withholding of this critical information, and there were resulting health and economic consequences that accompanied the additional potential cases of autism.

The following analysis summarizes the economic assessment performed of the costs of this alleged potential withholding of information [6]. The potential economic cost resulting from this alleged intentional withholding of critical information is the product of the number of children afflicted times the cost per victim.

For reasons enumerated in [6], the economic assessment started with an estimate of 100,000 African-American children who developed autism (between 2001 and 2015) due to the CDC's alleged intentional withholding of its findings of increased risk starting about 2001. The next question is: what are the economic costs associated with these 100,000 additional cases of autism? There are at least three main components of potential cost:

1) the cost of care for an autistic person;

2) the cost of medication and other medical treatments and services for an autistic person;

3) the wages lost over a lifetime by an autistic person.

For reasons shown in [6], a conservative estimate was used to arrive at a mean lifetime cost of $4M per capita for all USA autism afflicted. As will be shown in the next paragraph, both the $4M conservative lifetime cost per capita and the $2M extremely conservative lifetime cost per capita (shown in [6]) result in astronomical total costs.

For 100,000 African-American children in the USA over the period 2001-2015, using the approximate mean lifetime cost per capita of $4M from the above computations yields a total lifetime cost for the sub-group of roughly $400B! Using the approximate mean lifetime cost per capita of $2M would have yielded a total lifetime cost for the sub-group of roughly $200B. Either cost figure is astronomical, especially when the narrowness of the afflicted group is considered. Even if an undiscounted cost stream were used for the computations, total lifetime costs approaching $500B would have been obtained. The overall conclusions are essentially the same irrespective of which of these cost streams we assume; for purposes of further computation, the conservative assumption of $4M lifetime cost per capita will be used.

That $400B total lifetime cost estimate neglects the other sub-group identified at risk in the CDC study, children of all races who developed Isolated Autism from the MMR vaccine (according to Dr. Thompson's revelations to Dr. Hooker). This latter sub-group would be about seven or eight times as large as the African-American sub-group, since it includes all races, and the economic costs could be numerically larger than the African-American sub-group, depending on the level of increased risk for Isolated Autism.
As a side note, the NVICP (National Vaccine Injury Compensation Program), which is a special Court set up to compensate the vaccine injured, has paid out approximately $3.2B since 1988 for injuries from all vaccines. Approximately 9% of all compensated claims were for the MMR vaccine, for all groups, so the total MMR compensation would be somewhat less (perhaps substantially less) than $1B since 1988. Compare that with the ~$400B cost estimate above for 1) African-American children for the 2) 2001-2015 time period for the 3) USA for the 4) MMR vaccine only.

That's a small fraction of one percent of the total costs being compensated for this one group over one period. Other sub-groups potentially injured from the MMR vaccine, such as children with Isolated Autism, are not included in the $400B estimate!

So, if the lifetime autism cost for this one afflicted sub-group is ~$400B over its lifetime, and the Trust Fund compensation has been on the order of one-tenth of one percent of the lifetime autism cost (or less), then on the order of 99.9% (or more) of the total lifetime autism costs are being borne by sources other than the Trust Fund. Who are these sources? The main sources would appear to be the afflicted (and their families) and the taxpayers, through their support of myriad social services.

Status

According to Dr. Thompson’s statement above, thousands of documents have been turned over to Congress. On July 29, 2015, the Congressman to whom Dr. Thompson provided the documents, Rep. William Posey (R-FL), made a five minute speech on the Floor of the House [40], confirming and amplifying Dr. Thompson’s revelations.

The CDC study results, according to Dr. Thompson’s allegations, appear to contradict those of the large-scale open literature studies. It could be that the sample in the CDC study (metro Atlanta) was an anomaly relative to the samples in the large-scale studies. It could be there were errors in the data analysis in the CDC study. Or, as in the cases reported in Merchants of Doubt [41], it could be the large-scale studies were performed with the specific objective of showing vaccines were safe, particularly the MMR vaccine.

Given that a senior credible researcher, Dr. William Thompson (CDC), was willing to risk his reputation, career, and finances by coming forward with his allegations, his statements cannot be dismissed easily. If his allegations can be confirmed, with the implication that the CDC organization had the objective of proving the MMR vaccine safe, then the credibility of ANY CDC in-house or sponsored vaccine study has to be questioned.

As of mid-2018, there has been no change in the status of the MMR vaccine issue. There have been no Congressional Hearings on this issue, and Dr. Thompson has not testified in any legal proceeding. The mainstream media has ignored or downplayed Dr. Thompson's allegations. The paper in Pediatrics has not been retracted.
Appendix 3 - EPA Distortion of Biosludge Science

This section is excerpted from references [4] and [5]. In 1993, EPA generated a sludge rule that allows very toxic biosolids, or treated sewage sludge, to be used for farms, forests, playgrounds, and parks, etc. [42]. Dr. David Lewis (ex-EPA senior researcher) exposed how EPA, in coordination with other Federal Agencies, research institutions, and advisory groups, suppressed public knowledge of potential biosludge adverse health effects for years [43].

He did this exposure through
- published research articles,
- testimony and depositions before Congress,
- testimony and depositions before Department of Labor hearings, and
- lawsuits that revealed (under oath) the detailed participation of EPA officials and others in the science distortions.

Dr. Lewis was forced to retire and, in 2014, published a book (recounting his experiences) entitled Science for Sale [43]. He
- implied that EPA-sponsored research had to support EPA policy,
- implied that there was selective funding of scientists who supported EPA’s sludge rule,
- showed myriad ways the science was distorted to present biosludge as safe, and
- showed collusion among EPA and other agencies.


The data presented in the present study showing
- OSHA’s ultra-permissive Exposure Limits for toxic occupational substances,
- the EPA’s biosludge and Water Fluoridation examples shown in the appendices,
- the CDC’s alleged manipulation of MMR vaccine-autism data shown in Appendix 2,
- the FCC’s ultra-permissive Exposure Limits for wireless radiation that ignore the adverse effects reported in the literature,
and many other examples, provide strong evidence that what we have in practice (for these commercially, militarily, and politically sensitive topics) is a Government-Industrial-Media-Complex that effectively controls what the public knows and believes about these topics.
Appendix 4 - Definitions

4A. OSHA Permissible Exposure Limits (PELs)

The OSHA PELs derive from the OSH Act of 1970. Section 6(b)5 states [44]:

"The Secretary, in promulgating standards dealing with toxic materials or harmful physical agents under this subsection, shall set the standard which most adequately assures, to the extent feasible, on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capacity even if such employee has regular exposure to the hazard dealt with by such standard for the period of his working life. Development of standards under this subsection shall be based upon research, demonstrations, experiments, and such other information as may be appropriate. In addition to the attainment of the highest degree of health and safety protection for the employee, other considerations shall be the latest available scientific data in the field, the feasibility of the standards, and experience gained under this and other health and safety laws. Whenever practicable, the standard promulgated shall be expressed in terms of objective criteria and of the performance desired."

The main consideration in the Act is health and safety of the worker, although considerations of feasibility and scientific data are mentioned.

A 1997 article addressing new attempts to revise the PELs states, in part [45]:

"In 1970 OSHA created the first PELs by adopting the 1968 American Conference of Governmental Industrial Hygienists (ACGIH) threshold limit values (TLVs) as allowed by Congress, ... few PELs have been updated over the past 25 years, and the changes that were made often involved time-consuming, costly, and litigious fights among the regulated community, OSHA, and organized labor. For example, only 16 rules for about 20 chemicals have been promulgated since 1975."

"In 1988 OSHA proposed to change 420 of them.....The new standards, promulgated in 1989, were based on recent scientific information and all but one of the PELs was lowered.....However, legal challenges to the standard by numerous labor and industry groups were heard in the Eleventh Circuit Court of Appeals in 1992, and the court overturned the new PELs. The court indicated that OSHA needed to perform quantitative analyses of risk for noncancer endpoints.....offer more extensive discussion of the evidence for adverse effects for each substance, and prepare more careful feasibility analyses for each of the new PELs."

Thus, risk assessment/risk management/acceptable levels of risk and feasibility of implementation need to be considered along with worker health and safety.

4B. National Institute for Occupational Safety and Health (NIOSH) Recommended Exposure Limits (RELs).

OSHA describes the RELs as follows [46]:

"NIOSH RELs are authoritative Federal agency recommendations established according to the legislative mandate for NIOSH to recommend standards to OSHA. RELs are intended to limit exposure to hazardous substances in workplace air to protect worker health. In developing RELs and other recommendations to protect worker health, NIOSH evaluates all available medical, biological,
engineering, chemical, and trade information relevant to the hazard. NIOSH transmits its recommendations to OSHA for use in developing legally enforceable standards."

4C. American Conference of Government Industrial Hygienists (ACGIH®) Threshold Limit Values (TLVs®) and Biological Exposure Indices (BEIs®).

OSHA describes the TLVs and BEIs as follows [47]:

"ACGIH® is a private, not-for-profit, nongovernmental corporation. It is not a standards setting body. ACGIH® is a scientific association that develops recommendations or guidelines to assist in the control of occupational health hazards. TLVs® and BEIs® are health-based values and are not intended to be used as legal standards.

Threshold Limit Values (TLVs®) refer to airborne concentrations of chemical substances and represent conditions under which it is believed that nearly all workers may be repeatedly exposed, day after day, over a working lifetime, without adverse effects.

Biological Exposure Indices (BEIs®) are guidance values for assessing biological monitoring results – concentrations of chemicals in biological media (e.g., blood, urine). BEIs® represent the levels of determinants that are most likely to be observed in specimens collected from healthy workers who have been exposed to chemicals in the same extent as workers with inhalation exposure at the TLV®.

Since ACGIH® TLVs® and BEIs® are based solely on health factors, there is no consideration given to economic or technical feasibility. ACGIH® does not believe that TLVs® and BEIs® should be adopted as standards without an analysis of other factors necessary to make appropriate risk management decisions (e.g., control options, technical and economic factors, etc.)."

4D. Environmental Protection Agency (EPA) Integrated Risk Information System (IRIS)

4D1. Inhalation Reference Concentration (RFC)

EPA describes its RfCs as follows [48]:

"An estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious noncancer health effects during a lifetime. It can be derived from a NOAEL, LOAEL, or benchmark concentration, with uncertainty factors generally applied to reflect limitations of the data used."

"In the simplest terms, the RFC derivation begins with the identification of a no-observed-adverse-effect level (NOAEL) and a lowest-observed-adverse-effect level (LOAEL), which are determined for the specified adverse effect from the exposure levels of a given individual study on the various species tested. The NOAEL is the highest level tested at which the specified adverse effect is not produced and is therefore, by definition, a subthreshold level....This NOAEL/LOAEL approach, is also a function of the exposure levels used in the experimental design or is the function of designating a specified health effect measure....in the case of some alternative modeling approaches, and thus, does not necessarily reflect the "true" biological threshold."
"The RfC methodology requires conversion by dosimetric adjustment of the NOAELs and LOAELs observed in laboratory animal experiments or in human epidemiological or occupational studies to human equivalent concentrations (HECs) for ambient exposure conditions. These conditions are currently assumed to be 24 h/day for a lifetime of 70 years.

The dosimetric conversion to an HEC is necessary before the different adverse effects in the data array can be evaluated and compared."

4D1a. Lowest-Observed-Adverse-Effect Level (LOAEL)

"The lowest exposure level at which there are statistically and biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control group."

4D1b. No-Observed-Adverse-Effect Level (NOAEL)

"An exposure level at which there are no statistically and biologically significant increases in the frequency or severity of adverse effects between the exposed population and its appropriate control. Some effects may be produced at this level, but they are not considered as adverse, nor immediate precursors to specific adverse effects. In an experiment with several NOAELs, the assessment focus is primarily on the highest one for a given critical effect, leading to the common usage of the term NOAEL as the highest exposure without adverse effect."

4D1c. Uncertainty Factor (UF)

"One of several, generally 3- to 10-fold factors, used in operationally deriving the inhalation reference concentration (RfC) from experimental data. UFs are intended to account for (1) the variation in sensitivity among the members of the human population, (2) the uncertainty in extrapolating laboratory animal data to humans, (3) the uncertainty in extrapolating from data obtained in a study that is of less-than-lifetime exposure, (4) the uncertainty in using LOAEL data rather than NOAEL data, and (5) the inability of any single study to adequately address all possible adverse outcomes in humans. The RfC methods use 3 for the UF for interspecies extrapolation due to the incorporation of default dosimetric adjustments."

Also, reference [49] provides an early perspective on the purpose and methodology of Uncertainty Factors.

4D2. Oral Reference Dose (RfD)

"An estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from a NOAEL, LOAEL, or benchmark dose, with uncertainty factors generally applied to reflect limitations of the data used."

The derivations and caveats for an RfD are similar to those for the RfC above, with the difference being the RfC is based on inhalation, and the RfD is based on oral ingestion.
4E. Agency for Toxic Substances and Disease Registry (ATSDR)

ATSDR describes its MRLs as follows [50]:

"The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) [42 U.S.C. 9604 et seq.], as amended by the Superfund Amendments and Reauthorization Act (SARA) [Pub. L. 99-499], requires that the Agency for Toxic Substances and Disease Registry (ATSDR) develop jointly with the U.S. Environmental Protection Agency (EPA), in order of priority, a list of hazardous substances most commonly found at facilities on the CERCLA National Priorities List (NPL) (42 U.S.C. 9604(i)(2)); prepare toxicological profiles for each substance included on the priority list of hazardous substances, and to ascertain significant human exposure levels (SHELs) for hazardous substances in the environment, and the associated acute, subacute, and chronic health effects (42 U.S.C. 9604(i)(3)); and assure the initiation of a research program to fill identified data needs associated with the substances (42 U.S.C. 9604(i)(5)).

The ATSDR Minimal Risk Levels (MRLs) were developed as an initial response to the mandate. Following discussions with scientists within the Department of Health and Human Services (HHS) and the EPA, ATSDR chose to adopt a practice similar to that of the EPA's Reference Dose (RfD) and Reference Concentration (RfC) for deriving substance specific health guidance levels for non-neoplastic endpoints. An MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse non-cancer health effects over a specified duration of exposure. These substance specific estimates, which are intended to serve as screening levels, are used by ATSDR health assessors and other responders to identify contaminants and potential health effects that may be of concern at hazardous waste sites. It is important to note that MRLs are not intended to define clean up or action levels for ATSDR or other Agencies.

The toxicological profiles include an examination, summary, and interpretation of available toxicological information and epidemiologic evaluations of a hazardous substance. During the development of toxicological profiles, MRLs are derived when ATSDR determines that reliable and sufficient data exist to identify the target organ(s) of effect or the most sensitive health effect(s) for a specific duration for a given route of exposure to the substance. MRLs are based on non-cancer health effects only and are not based on a consideration of cancer effects. Inhalation MRLs are exposure concentrations expressed in units of parts per million (ppm) for gases and volatiles, or milligrams per cubic meter (mg/m³) for particles. Oral MRLs are expressed as daily human doses in units of milligrams per kilogram per day (mg/kg/day). Radiation MRLs are expressed as external exposures in units of millisieverts.

ATSDR uses the no observed adverse effect level/uncertainty factor (NOAEL/UF) approach to derive MRLs for hazardous substances. They are set below levels that, based on current information, might cause adverse health effects in the people most sensitive to such substance-induced effects. MRLs are derived for acute (1-14 days), intermediate (>14-364 days), and chronic (365 days and longer) exposure durations, and for the oral and inhalation routes of exposure. Currently MRLs for the dermal route of exposure are not derived because ATSDR has not yet identified a method suitable for this route of exposure. MRLs are generally based on the most sensitive substance-induced end point considered to be of relevance to humans. ATSDR does not use serious health effects (such as irreparable damage to the liver or kidneys, or birth defects) as a basis for establishing MRLs. Exposure to a level above the MRL does not mean that adverse health effects will occur."
4F. California Division of Occupational Safety and Health (Cal/OSHA) Permissible Exposure Limits (PELs).

OSHA describes the Cal/OSHA PELs as follows [51]:

Cal/OSHA has established an extensive list of PELs that are enforced in workplaces under its jurisdiction. Cal/OSHA PELs are promulgated under statutory requirements for risk and feasibility that are no less protective than the OSH Act.
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Special Mention Article

The following article deserves special mention. It is an outstanding enumeration of the role of business and other institutions in influencing government regulations. While it focuses on electromagnetic pollution, its main message is widely applicable to many other substances subjected to government regulation.

The conflicts of interest may even be worse than suggested by the authors. There are documented and undocumented conflicts. The authors focus on the documented conflicts. But, for some members of these advisory and regulatory committees, their time on the committee/panel serves as a ‘job interview’ for future funding and/or an eventual lucrative position in the industry impacted. The latter conflict is extremely difficult to identify a priori, but it would certainly impact the nature of their contribution.


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Presently, Research Affiliate at Georgia Institute of Technology.

Published over 200 peer-reviewed articles, served as Guest Editor of four journal Special Issues since 1994, and obtained two text mining system patents. Published on numerous medical topics in the peer-reviewed literature, including:

- potential treatments for Multiple Sclerosis, Parkinson's Disease, Raynaud's Phenomenon, Cataracts, SARS, Vitreous Restoration, and Chronic Kidney Disease;
- potential causes of Chronic Kidney Disease and Alzheimer's Disease;
- potential treatment protocol for prevention and reversal of Alzheimer's Disease;
- potential impact of toxin combinations on determining exposure limits;
- potential impacts of Electromagnetic Fields on health.

Listed in:

- Who's Who in America, 60th Edition (2006);
- Who's Who in Science and Engineering, 9th Edition (2006); and