

NIST Technical Note 1644

**Compilation of Data on the
Sublethal Effects of Fire Effluent**

Erica D. Kuligowski

NIST Technical Note 1644

Compilation of Data on the Sublethal Effects of Fire Effluent

Erica D. Kuligowski
*Fire Research Division
Building and Fire Research Laboratory*

August 2009



U.S. Department of Commerce
Gary Locke, Secretary

National Institute of Standards and Technology
Patrick D. Gallagher, Acting Director

Certain commercial entities, equipment, or materials may be identified in this document in order to describe an experimental procedure or concept adequately. Such identification is not intended to imply recommendation or endorsement by the National Institute of Standards and Technology, nor is it intended to imply that the entities, materials, or equipment are necessarily the best available for the purpose.

National Institute of Standards and Technology Technical Note 1644
Natl. Inst. Stand. Technol. Tech. Note 1644, 47 pages (August 2009)
CODEN: NSPUE2

Abstract

The Nuclear Regulatory Commission (NRC) is developing guidance for performing quantitative human reliability analysis for post-fire mitigative human actions. In some of the scenarios, operators may be exposed to fire effluent as they perform critical tasks.

In this report, the National Institute of Standards and Technology (NIST) provides a review of the state-of-the-art on how fire effluent might affect people. The available scientific literature on the effects of narcotic and irritant gases, smoke obscuration, and heat on humans and animals were reviewed. The fire effluent data presented in this report are categorized by levels of effect on humans; specifically 1) minor physiological effects that are unlikely affect job performance or duties, 2) moderate to major physiological effects that may negatively influence job performance or duties, and 3) major physiological effects that may render an individual unable to perform his/her job duties. Where possible, NIST has identified groupings and/or contradictions for the compiled exposure data. With this information, one can estimate how exposure to various fire effluent might affect the operators' ability to perform critical procedures during a fire event.

Keywords

fire, fire research, smoke, heat, fire toxicity, smoke toxicity

Contents

1. Introduction.....	2
2. Data Collection	2
3. Data Grouping.....	4
4. Report Limitations	5
5. Data Tables and Summaries.....	6
5.1. Narcotic Gases – Tables and Summaries.....	7
5.1.1. Carbon monoxide (CO).....	8
5.1.2. Hydrogen Cyanide (HCN).....	11
5.2. Irritant Gases – Tables and Summaries	13
5.2.1. Hydrogen Chloride (HCl).....	14
5.2.2. Hydrogen Fluoride (HF).....	17
5.2.3. Hydrogen Bromide (HBr).....	19
5.2.4. Acrolein (C ₃ H ₄ O).....	21
5.2.5. Formaldehyde (CH ₂ O).....	22
5.2.6. Nitrogen Dioxide (NO ₂).....	24
5.3. Heat – Tables and Summaries.....	27
5.4. Smoke – Tables and Summaries.....	31
6. Conclusion	34
7. References.....	35

Tables

Table 1. Carbon Monoxide	9
Table 2. Summary of Data on Carbon Monoxide.....	10
Table 3. Hydrogen Cyanide	12
Table 4. Summary of Data on Hydrogen Cyanide.....	13
Table 5. Hydrogen Chloride	15
Table 6. Summary of Data on Hydrogen Chloride.....	16
Table 7. Hydrogen Fluoride.....	18
Table 8. Summary of Data on Hydrogen Fluoride	19
Table 9. Hydrogen Bromide	20
Table 10. Summary of Data on Hydrogen Bromide.....	20
Table 11. Acrolein	21
Table 12. Summary of Data on Acrolein.....	22
Table 13. Formaldehyde	23
Table 14. Summary of Data on Formaldehyde.....	24
Table 15. Nitrogen Dioxide	25
Table 16. Summary of Data on Nitrogen Dioxide.....	26
Table 17. Heat.....	29
Table 18. Summary of Data on Heat	31
Table 19. Smoke Density.....	32
Table 20. Summary of Data on Smoke Density	34

Acknowledgements

Support for the compilation of data on the sublethal effects of fire effluent has been provided by the Office of Nuclear Regulatory Research at the U.S. Nuclear Regulatory Commission. Thanks to Susan Cooper, Kendra Hill, Mark Salley, and Jason Dreisbach for their efforts. My appreciation to Richard Gann, Richard Peacock, Nathan Marsh, Jason Averill, and Nelson Bryner for providing detailed and insightful suggestions.

Compilation of Data on the Sublethal Effects of Fire Effluent

Erica D. Kuligowski
Fire Research Division; Building and Fire Research Laboratory

1. Introduction

The burning of materials produces toxic gases (narcotic and irritant gases), smoke aerosols, and heat. The quantity and type of materials burning and the ventilation available influences what types of fire products (effluent) are produced [1,2]. For example, nonflaming, ventilation-limited and/or large, post-flashover fires are the prime conditions for producing toxic products, specifically narcotic and irritant gases, and heavy, dark smoke.

Fires can spread smoke and hot gases throughout a building. An individual's location relative to the fire is a good predictor of whether he/she is likely to encounter harmful fire products and what types of products he/she is most likely to encounter first. This is known as the limited hazard [3]. For example, if an individual is located close to the fire source, he/she is more likely to encounter and be affected by the heat from the fire first, but if an individual is located farther away from the fire, he/she is more likely to initially encounter spreading smoke and gases.

If individuals are exposed to one or more fire products at sufficient concentrations over time, they can develop potentially serious physiological effects. Physiological effects from narcotic gases can include headaches, dizziness, and depression of the central nervous system; effects from irritant gases can include minor to severe irritation of the eyes, the upper respiratory tract (e.g., the nose, throat, and mouth), and/or lower respiratory tract (e.g., the trachea, bronchi, and lungs); and thermal effects can include hyperthermia, skin burns, and burns to the respiratory tract. These effects can lead to an inability to perform job duties, an inability to escape from a building, unconsciousness, and/or death. In addition to the effects during exposure, people can develop harmful symptoms after exposure that can lead to health problems and/or death [2].

The purpose of this report is to summarize data on the amount of fire effluent (narcotic gases, irritant gases, heat and smoke) necessary to produce sublethal effects to humans. Sublethal effects are important because they reduce the effectiveness of people performing critical tasks and also inhibits people's abilities to move to safety such that they may ultimately be exposed to lethal conditions.

2. Data Collection

Although fires can produce a variety of different harmful products, this report features the fire products most often identified as likely products that are toxic or irritant to humans (not limited

to nuclear power plants). This report focuses on effects to humans from exposures to heat; smoke; narcotic gases, specifically carbon monoxide (CO) and hydrogen cyanide (HCN); and irritant gases, specifically hydrogen chloride (HCl), hydrogen fluoride (HF), hydrogen bromide (HBr), acrolein (C₃H₄O), formaldehyde (CH₂O), and nitrogen dioxide (NO₂).

The bibliography of research studies compiled in this report was developed using the following major sources: the relevant National Fire Protection Association and Society of Fire Protection Engineers handbook chapters [1,2,4], Acute Exposure Guideline Levels (AEGL) documents [5] and the SFPE Engineering Guide Predicting 1st and 2nd Degree Skin Burns from Thermal Radiation [6]. These documents were seminal in identifying those studies where humans and animals developed sublethal effects to various gas, heat and smoke exposures. As much as possible, data found in the NFPA, SFPE, and AEGL resources were traced back to the original primary (or barring that, secondary) source.

The AEGL documents provided the majority of the references for the toxicological studies (narcotic and irritant gas studies) included in this report. The AEGL draft reports are developed by the Environmental Protection Agency (EPA)-sponsored* National Advisory Committee for Acute Exposure Guideline Levels for Hazardous Substances (NAC/AEGL), which are subsequently reviewed, modified, and published by the National Research Council's (NRC) Committee on Toxicology. The NAC/AEGL consists of members from the EPA, the Department of Defense (DOD), the Department of Energy (DOE), the Department of Transportation (DOT), other federal and state governments, the chemical industry, academia, and organizations from the private sector. In the AEGL reports, members of NAC/AEGL identified, reviewed, and interpreted data from relevant toxicological studies on humans and animals to develop acute exposure guideline levels (AEGLs) for the general public†. An AEGL document was available for each narcotic and irritant gas included in this report.

For narcotic fire effluent, toxicity (physiological effects) depends on the accumulated dose by the human or animal; namely the concentration of the effluent (in microliters per liter – $\mu\text{l/l}$)‡ and the time of exposure. Therefore, for each human or animal study, the concentration of the fire product, the time duration of the exposure (if applicable), and a description of the sublethal effect was noted as one data point. In some studies, more than one test was performed, which led to multiple data points from the same study. Data were collected from toxicological studies of animals exposures because data on humans, especially near-lethal effects, are understandably

* The EPA's Office of Pollution Prevention and Toxics (OPPT)

† AEGL exposures levels: The first level provides the threshold concentration (in parts per million – ppm) above which the general public, including susceptible populations (such as children, elderly, and people with pre-existing heart or respiratory diseases), could experience notable discomfort. The second level provides the threshold value above which the general public, including susceptible populations, could experience irreversible or other serious health effects and/or inability to escape. The third level provides the threshold value above which the general public, including susceptible populations, could experience life-threatening health effects or death.

‡ Historically, toxicological publications have used parts per million by volume (ppm) as the units for toxic gas concentrations. According to ISO [26:2], "the typical units for the concentration of a toxic gas are microliters per liter ($\mu\text{l/l}$)."

Therefore, this report uses $\mu\text{l/l}$. Please note that the units of $\mu\text{l/l}$ are numerically equivalent to ppm by volume [26]. Also, the term "concentration" will be used throughout the report to refer to the amount of a contaminant (gas) in the atmosphere per unit volume of the atmosphere ($\mu\text{l/l}$) rather than "volume fraction" to follow ISO 13571 [26].

limited. Animal studies have no one-to-one correspondence to human effects, but can provide some insight on human effects.

There were data found that are not included in this report. First, per request from the NRC, no data on susceptible populations are provided in this report. For example, susceptible populations can include fetuses, children, coronary artery disease patients, and asthmatics to narcotic or irritant gases. The AEGL threshold values† are also omitted from this report because they include data that account for susceptible populations in the threshold concentrations. Last, data for compounds that produce no effect on humans or animals and data for compounds that produce lethal effects on humans or animals are not included in this report. These two categories are outside of the scope of this report.

3. Data Grouping

The data points, consisting of the fire product concentration or dose and the description of the sublethal effect, were categorized into three main groupings. The following three categories, derived as part of this project, characterize the ability of the operator to perform work-related functions during exposure to fire products:

Category 1: Minor physiological effects that are unlikely to affect job performance or duties

Category 2: Moderate to major physiological effects that may negatively influence job performance or duties

Category 3: Major physiological effects that may render an individual unable to perform his/her job duties.

Category 1 groups all concentrations or doses that produce only minor physiological effects. These effects are unlikely to affect job performance or duties. For narcotic gases, these effects can include occasional or slight headaches or other mild central nervous system effects. For irritant gases, this can include mild irritation to the eyes or nose. In the case of heat, this can include mild discomfort to the individual. For smoke, this can include a slight decrease in an individual's walking speed or steadiness of movement. Concentrations or doses listed in Category 1 are likely to produce only slight discomfort that is unlikely to affect job performance or duties.

Category 2 groups all concentrations or doses that produce moderate to major physiological effects that may negatively influence job performance or duties. Effects from narcotic gases in Category 2 can include decided headaches, dizziness, and vomiting. The effects from irritant gases can include moderate to severe eye, nose, and throat irritation with possible developments of mild pulmonary effects. In the case of heat, this can include the initiation of pain to the skin. In the case of smoke, this can include a more significant decrease in walking speeds or visibility distance.

Category 3 groups all concentrations or doses that produce effects that render an individual unable to function and/or perform his/her job duties but are less than those concentrations or doses which may result in death. At this level, concentrations or doses of narcotic gases, irritant gases, heat, or smoke are so high that incapacitation potentially to loss of consciousness occurs. Irritant gases (except at doses likely to cause pulmonary effects), heat, and smoke are unlikely to

cause an individual to lose consciousness. For these fire products, incapacitation is defined as the inability of an individual to take constructive action to effect one's own escape. In this category, the irritant gases, heat, and smoke levels are such that the person is no longer able to see or move to effect his/her own escape. For example, heat effects at this level include the onset of pain and blistering to exposed skin and hyperthermia. In conclusion, Category 3 exposure levels represent significant danger to the individual exposed and a major threat to task completion.

In addition to the judgment of the author, two experts in the fields of 1) occupational medicine and internal medicine and 2) inhalation toxicology and fire science were consulted on the categorization of specific data points. For data points where experts did not agree, the data point was categorized using the more severe (or conservative) category chosen by one of the experts.

4. Report Limitations

There are several limitations regarding the potential use of this report and the data used within the report. First, this report is not a toxicological study, but rather an exercise of applied engineering judgment. As with most engineering judgment, legitimate differences of interpretation may exist given the uncertainty in the baseline of available data and the current state of scientific understanding of the toxicological effects of fire effluent. The data are presented in such a way that the categorization is transparent and may be re-categorized using other methods more appropriate to a particular engineering problem. Second, any assessment as to why one data point is higher or lower than another from the same study or different studies is outside of the scope of this report.

Third, this report provides data on each fire product as if it was the sole product in the fire scenario, although this is typically not the case in a real fire. The source data on exposures to gases, smoke, and heat are primarily based on studies that expose the subject to only a single fire product. Multiple toxic products may interact in three ways: 1) no interaction, in that the presence of other products has no effect on the symptoms caused by any particular product, 2) additive, in that each product contributes to the overall toxic level such that their contributions "add up" and are linear with the concentration of each product, and 3) synergistic, in that one particular product enhances the toxic hazard of one or more other products. There are studies that have found no measurable interaction between CO and HCN [7,8,9] and between carbon dioxide (CO₂) and CO [10,11]. Contradictory to this, studies have shown that there is at least some additive effect between CO and HCN [1,12,13,14,15,16] and CO and HCl [17]. Narcotic and/or irritant gases are additive when the fraction of the toxic dose of each gas (e.g., for incapacitation) adds up to unity [1,18][§]. In the case of synergism, some studies have found synergism between

[§] Fractional effective dose (FED) models can be used for predicting toxic hazards from toxicological and flammability data. A fractional effective dose for a fire product can be calculated by dividing the incremental exposure dose (concentration multiplied by time increment) by the total exposure dose required to produce a given toxicological effect (i.e., incapacitation or lethality). For irritants, the fractional effective concentration can be calculated by dividing the concentration of the irritant at a certain time by the concentration of irritant required to cause a specific effect (e.g., incapacitation). The fractional effective doses (or concentrations) for one or more toxic products are summed until a time is reached at which the sum become unity. At this time, the exposed subjects are expected to succumb to the effect (i.e., incapacitation or lethality). FED models and equations are not included in this report per request of the NRC and due to the fact that these models are used to predict very serious effects, such as incapacitation and lethality (lethality is not included in this report).

HCN and CO [1,19] and CO and CO₂ [16,20,21], with HCN and/or CO₂ increasing the respiration rate of an individual and in turn, producing a more rapid uptake of the toxicants present in the atmosphere. Purser [1] states that even though CO₂ is not toxic at concentrations below 5 %, at 3 % of CO₂, the respiratory minute volume (RMV) (or volume of air breathed) of an individual is approximately doubled and at 5 % CO₂, the RMV is approximately tripled. Overall, Purser [1] suggests the following: assume that CO and HCN are directly additive, assume that the rates of CO and HCN increase in proportion to any increase in ventilation caused by CO₂, and assume that irritancy is independent of asphyxia, however, the uptake of irritants is increased by CO₂.

Fourth, there are limited toxicological data on humans and even less data on more sensitive individuals. Not all humans exposed to the same amount of fire product for the same time period will experience similar effects. Some people are more sensitive than the general public and there are others that are more resistant. Those members of the subpopulations who are more sensitive to acute exposures of smoke gases include fetuses, children, the elderly, and individuals with pre-existing diseases that decrease the availability of oxygen to critical body tissues (e.g., coronary artery disease) for narcotic gases [5] and people with pre-existing respiratory diseases (e.g., asthma) for irritant gases [5]. Also, the activity level of the individual during exposure affects the uptake of the particular gas into the body, which influences the severity level of the effect(s) experienced by the person. Those individuals more susceptible to heat effects are people with higher skin temperatures (in the case of skin burns) and thinner skin thickness (in the case of skin blisters) [22]. An individual's initial skin temperature can vary significantly (between 27 to 38 °C) among people based on attributes such as age, sex, occupation, physical activity, and pregnancy [22]. Data on susceptible populations are outside of the scope of this report.

Finally, there are also uncertainties associated with the methods, data collection, and analysis of the human and animal studies collected for this report. In some cases, data are presented in secondary sources with no possibility of obtaining the original source. The reader should evaluate each value cited in this report to assess whether the data are appropriate to his/her analysis, taking into account the scientific measurement methods, experimental protocols, range of data, and purpose of the study. With these limitations in mind, readers of this report should use the provided data with caution and with complete understanding of the uncertainties associated with its collection and presentation.

5. Data Tables and Summaries

In the following sections of this report, data from a variety of different human and animal studies are categorized, including concentrations or dose and description of the sublethal effect. Data are provided first for narcotic gases, then irritant gases, and then for heat and smoke. In each section, a description of the fire product is provided, then all data points are presented in a compilation table, and finally data summaries are displayed based on the three severity categories described in the Data Grouping section of this report.

The compilation tables consist of all of the data points collected for each of the fire products that are categorized into the three main severity level categories. Data from studies performed on both humans and animals are included in each compilation table. Animal data are included in the compilation tables to provide additional information to support the more limited human data; however a larger uncertainty exists in their relation to human effects. In some cases, a factor of three difference is found between human and animal data. To the extent that animal studies provide data points that are the same or different from other data point/results, explanations for these results are outside of the scope of this report. For these kinds of interpretations, the reader would need to consult experts in human and animal physiology for further clarification.

Following the compilation table in each section, a data summary table is provided to summarize the data from the compilation tables. For each fire product, the range of concentration or dose data from the human and primate (if any) studies is provided for each of the severity categories, specifically identifying those concentrations or doses that caused minor, moderate, or severe effects on humans. Primate data are included because experts have found that primates are appropriate models for humans due to the similarity in both gross anatomy and respiration patterns [23,24]. In some circumstances, it would have been possible to extrapolate and/or interpolate from the compilation data to produce more complete data ranges for categories for which there were little or no data. However, due to uncertainty in the data on sublethal effects, the decision was made for the summary data tables to include only the data available in the literature. By using only the available data, data ranges for different categories sometimes overlap and other times, there are data gaps in between categories.

5.1. Narcotic Gases – Tables and Summaries

Narcotic gases produce effects on an individual by depressing the body's central nervous system. Normal body function is possible up to a certain concentration of narcotic gas for a period of time, then deterioration of the body functions are quick and severe. Effects can begin with slight headaches, dizziness, lethargy and then rapidly progress to incapacitation and death if the exposure continues.

The effects of narcotic gases on humans depend on the concentration of gas accumulated over a period of time. This is known as the accumulated dose. An individual can sustain a low concentration for a longer time or a high concentration for a shorter time and experience the same symptoms.

The principal narcotic gases produced in a typical fire are carbon monoxide (CO) and hydrogen cyanide (HCN). Although lower concentrations of oxygen (O₂) (less than 15 % from 21 %) and very high concentrations of CO₂ (greater than 5 %) can produce toxic effects, compiled and summarized data for these toxicants are not included in this report [1,25, 45]. At levels of O₂ and CO₂ where toxic effects can begin, the principal narcotic gases (CO and HCN) are likely to be present in doses that can cause harmful effects to humans. Therefore, this report focuses on those narcotic products most often identified as likely products that are toxic to humans (CO and HCN). In cases where additional information is required on the effects of O₂ depletion and CO₂ on humans, literature is available [1].

Data are presented on the accumulated doses of CO and HCN that produce three levels of severity effects in humans and animals. Each section describes the narcotic gas, presents the compiled data on the effects of the accumulated dose of the narcotic gas on humans and animals, and provides a data summary on human and/or primate data for each severity category.

5.1.1. Carbon monoxide (CO)

Carbon monoxide (CO) is a tasteless, nonirritating, odorless, and colorless gas. CO binds to hemoglobin in the blood forming carboxyhemoglobin (COHb), which renders hemoglobin molecules in the body less able to bind oxygen. By blocking the oxygen, CO lowers the blood's oxygen delivery capacity, thereby depriving the body's tissues, especially brain tissue, of oxygen [1,26,27]. The effects or symptoms produced by CO exposure are directly correlated to the percentage of blood hemoglobin that is converted to COHb during exposure.

For that reason many of the toxicological studies of CO on humans and animals report the percentage of COHb that causes specific physiological symptoms/effects on humans and animals. COHb is defined as the amount of CO (in ml) per ml of blood at a specific exposure time. COHb can be presented as a percentage by multiplying this concentration by 100 and then dividing by the concentration of oxyhemoglobin (OHb), i.e., the ml of oxygen per ml of blood under normal conditions [27]. COHb, as a percentage, can be converted to a specific concentration of CO ($\mu\text{l/l}$) over a time period (and vice versa) by using a mathematical model known as the Coburn Foster Kane equation (CFK) [27,28].

Table 1 includes data points from both human and animal exposures to CO categorized by the three main groupings. Note that only data on healthy adults and animals are included in this compilation table. In some studies or secondary sources, the accumulated dose is presented as a percentage of COHb, whereas in others, the dose is presented as a concentration of CO ($\mu\text{l/l}$) over time. Both types of data are presented in the compilation table for CO.

Table 1. Carbon Monoxide

Category 1: Minor effects unlikely to affect job performance					
$\mu\text{l/l}$	COHb (%)	Time (min)	Subject	Notes	References
	5	NA	Human	Subtle, nonadverse effects; decrements in neurobehavioral function begin at this percentage	[29,30,31]
	15 to 20	NA	Human	No effects observed during submaximal exercise in healthy individuals	[32]
	10	NA	Human	No appreciable effect	[31]
	20	NA	Human	Shortness of breath on moderate exertion, occasional headaches with throbbing temples	[31]
600		60	Human	2 out of 9 subjects reported slight headache	[33]
200		240	Human	Mild sinus headache in final hour (sedentary)	[34]
500		20, 60, 90	Human	Lightheadedness and frontal headache	[34]
524		10	Human	Lowest value to cause an effect	[35]
Category 2: Moderate to major effects that may negatively affect job performance					
	30		Human	Decided headache, irritable, easily fatigued, judgment disturbed, possible dizziness, dimness of vision	[31]
	20.7 \pm 7.0		Human	Majority complained of headaches, dizziness, weakness, nausea, trouble thinking, (6 % lost consciousness)	[36]
	21 \pm 0.7		Human	Majority complained of severe headaches, dizziness, weakness, nausea, chest pain/tightness, shortness of breath	[37]
5 000		11.5	Human	Increased breathing after running upstairs	[38]
3 600, 3 900		30	Human	On walking, throbbing in head, palpitations; on running, out of breath, slightly impaired vision	[38]
2 100		60	Human	Increased breathing more distinct; beginning to look pale/yellowish	[38]
1 200		120	Human	Increased breathing distinct, feeling uneasy; on running – weak in the legs, impaired vision and hearing	[38]
460		240	Human	Unusual shortness of breath, slight palpitations	[38]
	16 to 21		Monkey	Deficits in behavioral task performance started – momentary closure of eyes, yawning, shaking of head, occasional sitting down, less active	[39]
Category 3: Major effects that are likely to render an individual unable to complete job tasks					
	21		Human	Fullness of head and precordial pain, transient unconsciousness	[40]
	28 to 32		Human	Throbbing headaches, vomiting, vertigo; passed out several times	[41]
	40 to 50		Human	Headache, confusion, collapse, fainting on exertion	[31]
	27 \pm 12		Human	Loss of consciousness at higher percentages	[42]
800-900		60	Human	Decided frontal headaches, insomnia, irritability, marked loss of equilibrium	[33]
	30		Baboons	Incapacitation	[43]
2 700		10	Primates	Incapacitation	[1]
900		30	Primates	Incapacitation	[1]
2 738-2 968		10	Rats	Incapacitation	[12]

With the exception of one set of data [38], these data converge to create data ranges for each category for healthy adults. A summary data table (Table 2) was developed from the data on healthy adults. The summary data ranges were formed by examining the human data within each category and forming a range around which all or most of the data converge.

Most of the data on healthy adults can be placed within a range of COHb percentages, which can then be converted to concentrations of CO ($\mu\text{l/l}$) for specific time periods. For Category 1, the literature values converged between 5 % to 20 % of COHb by using the data from WHO [31] as the upper and lower bounds for the range. For Category 2, the literature values converged to form a range of 13 % to 30 % COHb by using Burney et al. [36] for the lower end of the range and WHO [31] to create the upper bound. Even though unconsciousness (6 %) was found in the Burney et al. [36] study, the lower bound of COHb from this study (13 %) is used as a lower bound for Category 2. For Category 3, data converged around a range of 21 % to 40 % COHb by using the Ebisuno et al. [40] study as the lower bound for incapacitating effects and the WHO [31] data as the upper bound. These data were even supported by the animal data presented in Table 1, however only human data were used to create the data ranges for each category. The Haldane study [38] was the only outlier in Table 1 that did not support this data range.

Table 2 shows the data ranges for each category from exposures to CO. The COHb percentages listed above were converted to concentrations of CO ($\mu\text{l/l}$) for each time interval using the CFK equation [27,28] with the Peterson and Stewart correction [44]**. Where values fell slightly outside of the COHb range but still converged to the data range, these were listed in the table (see [33,34,35] in Table 2). However, when they fell inside the data range, they were listed in the reference list (see a, b, and c in Table 2).

Table 2. Summary of Data on Carbon Monoxide

	10 min	30 min	60 min	120 min	240 min
Category 1 ($\mu\text{l/l}$)	550 to 2 500 ^a 524 ^[35]	195 to 880 ^a	105 to 475 ^a 500 ^[34] 600 ^[33]	60 to 275 ^a	42 to 190 ^a 200 ^[34]
Category 2^b ($\mu\text{l/l}$)	1 590 to 3 800	560 to 1 330	300 to 730	175 to 430	115 to 300
Category 3^c ($\mu\text{l/l}$)	2 630 to 5 110	920 to 1 800	500 to 1 000	290 to 600	200 to 440

^a The Category 1 concentrations were constructed based on the following studies that suggested minor effects from 5 % to 20 % COHb: WHO [31]; EPA [29]; Ely et al. [37]; Kimmerle [45]; Stewart et al. [34]

^b The Category 2 concentrations were constructed based on the following studies that suggested major effects from 13 % to 30 % COHb: WHO [31], Burney et al. [36]; Ely et al. [37]; Kimmerle [45]; Stewart et al. [34]. These values were supported by Purser and Berrill [39].

^c The Category 3 concentrations were constructed based on the following studies that suggested human incapacitation from 21 % to 40 % COHb: WHO [31]; Ebisuno et al. [40]; Grace and Platt [41]; Burney et al. [36]; Sokal and Kralkowska [42]; Henderson et al. [33]; Stewart [46]. These values were supported by Kaplan et al. [43]; Purser [1]; Crane et al. [12].

The data presented in Table 2 can be used to predict what effects are likely when/if an individual is exposed to a specific concentration of CO over a period of time. For some accumulated dose

** This calculation was done assuming a 70-kg man, a blood volume of 5 500 ml [28] and a daily inhalation volume (V_E) of 22 m³ (this assumes 8 hours resting and 16 hours light/non occupational activity), a respiration rate of 18-min⁻¹ and a dead space (V_D) of 2.2 ml/kg. These are the same assumptions used the CO AEGL document [27].

values (e.g., 2 500 $\mu\text{l/l}$ for 10 min), Table 2 shows that an individual can experience Category 1 effects or Category 2 effects. This overlap occurs because humans react to CO exposure in different ways. The concentration that may cause considerable effects in one person may cause almost no effects in another.

5.1.2. Hydrogen Cyanide (HCN)

Hydrogen cyanide (HCN) is a colorless, highly poisonous gas with an odor of bitter almonds [47,48]. The lethal dose is 25 times smaller than CO and it is a fast-acting narcotic. HCN prevents the utilization of oxygen by the cells of the body, which can lead to loss of consciousness, respiratory arrest, and death [26,47].

The compilation data for HCN are presented in Table 3, which includes both human and animal exposure studies. In each study or secondary source, the accumulated dose is presented as a concentration of HCN ($\mu\text{l/l}$) over time. Only data involving healthy subjects and animals were found for HCN, therefore people with health problems (e.g., asthma) or other susceptibilities are not represented in this data compilation.

Table 3. Hydrogen Cyanide

Category 1: Minor effects unlikely to affect job performance				
$\mu\text{l/l}$	Time (min)	Subject	Notes	References
8	60	Human	No more than mild central nervous system effects (e.g., mild headaches)	[49] by interpreting [50]
4 to 12	years	Human	Headache, weakness and objectionable changes in taste and smell	[50]
18 to 36	120+	Human	Slight headaches after several hours	[45]
Category 2: Moderate to major effects that may negatively affect job performance				
>15	Unknown	Human	Headache, dizziness, nausea or vomiting, loss of appetite	[51]
25 to 75	60	Human	Numbness, weakness, vertigo, nausea, rapid pulse, and flushing of the face	[52]
45 to 54	30 to 60	Human	Could be tolerated for ½ to 1 hour	[45]
500 to 625	1.5	Human	No immediate effects; postexposure - feelings of nausea, inability to concentrate in conversation	[53]
60	30	Monkey	Slight depressive effect on nervous system; changes in brain wave activity at end of exposure	[54]
55	30	Rat	No toxic signs, changes in lung dynamics	[55]
200	12.5	Rat	Possible changes in blood enzymes attributed to cardiac effects	[56]
63	30	Mouse	Respiratory depression by 50 %	[57]
Category 3: Major effects that are likely to render an individual unable to complete job tasks				
450	3	Human	Collapsed and unconscious	[58]
125	12	Monkey	Distinctly toxic	[59]
100, 102, 123, 140, 147, 156	19, 16, 15, 10, 8, 8	Monkey	Incapacitation	[60]
80 to 180	Up to 30	Monkey	Episode of hyperventilation with subsequent unconsciousness some time during 30-min period	[54]
94	30	Monkey	Incapacitation	[60]
99 to 119	10	Rats	Incapacitation	[12]
124, 74, 50, 42	5, 10, 20, 30	Mouse	Lack of movement for 5 min in a rotating cage after exercise	[61]
150	11	Mouse	Incapacitation	[57]

Although most of the human studies available on HCN exposure were limited to occupational reports and short-term exposures, data summaries can still be created from the data compilation. Table 4 shows the summary data for human and primate exposures to HCN. Data summary ranges are only provided for the exposure time on which data was available.

There are limited human data presented in Category 1. El Ghawabi et al. [50] found that individuals experienced headaches, weakness and changes in taste and smell after years of occupational exposure to 4 $\mu\text{l/l}$ to 12 $\mu\text{l/l}$ of HCN. From this data, the NRC Subcommittee on

Spacecraft Maximum Allowable Concentrations concluded that 8 µl/l of HCN would likely produce only mild central nervous system effects (e.g., slight headaches) after a 60-min exposure [49]. Therefore, the range chosen to produce only minor effects on humans after a 60-min exposure to HCN is 4 µl/l to 12 µl/l of HCN. Kimmerle [45] (secondary source) reports that individuals can be exposed to even higher concentrations for longer periods of time, which is considered to be an outlier for Category 1. These are the only data points found where the concentration produced only minor effects in healthy humans.

Table 4. Summary of Data on Hydrogen Cyanide

	10 min	Subject	30 min	Subject	60 min	Subject
Category 1 (µl/l)	--	--	--	--	4 to 12	Human [49,50]
Category 2 (µl/l)	--	--	45 to 54	Human [45,59,62] ^a	25 to 54	Human [45,52,62]
Category 3 (µl/l)	125 to 140	Monkey [59,60]	80 to 94	Monkey [54,60]	---	---

^a supported by Purser [54]

In Category 2, data on HCN concentrations are provided mainly for exposure times ranging from 30 min to 60 min. As a result, summary data for Category 2 are provided only for 30-min and 60-min exposure times (see Table 4). Kimmerle [45] and Dudley et al. [59], from Flury and Zernik [62], state that humans can tolerate 45 µl/l to 54 µl/l of HCN without too many difficulties for 30 min to 60 min. These data are supported by Purser [54] who found that monkeys experienced only moderate effects to brain wave activity at concentrations of 60 µl/l for 30 min. Therefore, Table 4 suggests that the 30-min dose of HCN that is likely to cause Category 2 effects in humans is 45 µl/l to 54 µl/l. For 60-min exposures, Parameter [52] found that humans experienced moderate to major effects between 25 µl/l and 75 µl/l and Kimmerle [45] suggests the same range of HCN (45 µl/l to 54 µl/l) as for 30-min exposures. In an attempt to conservatively mesh the two data ranges together, the 60-min exposure of HCN in Table 4 uses the lower bound of Parameter's [52] range and the upper bound of Kimmerle's [45] range. Therefore, a 60-min dose of HCN likely to cause Category 2 effects in humans is 25 µl/l to 54 µl/l.

For Category 3, the data summary was constructed of primate data since there was only one data point from a human study available (and this study provided a very short exposure time to HCN). At ten min, the primate data in Table 3 show that major effects can occur between 125 µl/l and 140 µl/l of HCN [59,60]. Additionally, primate data show that incapacitation can begin anywhere from 80 µl/l to 180 µl/l during a 30-min exposure to HCN [54] and another study notes the incapacitation of a monkey after 30 min of exposure to 94 µl/l of HCN [60]. Since there is an actual exposure time associated with 94 µl/l (and incapacitation can occur almost immediately at 180 µl/l), 94 µl/l was used as the upper limit for a 30-min exposure to HCN.

5.2. Irritant Gases – Tables and Summaries

Irritant gases cause physiological effects on people in two different ways: irritation to the upper respiratory tract (referred to as sensory irritation) and effects to the lungs. Irritant gases can affect

the upper respiratory tract and eyes of an individual almost instantaneously [1,2,26]. These sensory effects can range from eye irritation to pain in the respiratory tract. Although pain may be considered as functionally incapacitating, unconsciousness or even lethality is not likely to result from irritant gases, except at higher concentrations, where they are likely to penetrate the lungs and lead to pulmonary inflammation and edema (i.e., swelling or fluid accumulation in the lungs). Inflammation and edema can cause respiratory difficulties and ultimately lead to death hours after exposure. Whereas sensory effects are likely to occur immediately in response to certain concentrations of irritant gas, pulmonary effects depend on an accumulated dose.

Data are presented on the concentrations of irritant gases that produce three levels of severity effects in humans and animals. The irritant gases that are tabulated are hydrogen chloride (HCl), hydrogen fluoride (HF), hydrogen bromide (HBr), acrolein (C₃H₄O), formaldehyde (CH₂O), and nitrogen dioxide (NO₂). Each section describes the irritant gas, presents the compiled data on the effects of the concentration and/or accumulated dose of the irritant gas on humans (and animals), and provides a data summary on human and/or primate data for each severity category.

In the irritant gas data compilation tables presented in this report, many of the data points for humans include only the concentration rather than an accumulated dose. This is because much of the data available from human studies involve only sensory irritation, which is almost immediate, making the inclusion of a time period unnecessary. In the instances where an exposure time is listed for a human study, this is included in the summary table. It is more conservative to assume an immediate response for all sensory irritation effects. Also, when a concentration for incapacitation of humans is listed for each irritant gas, an immediate effect should also be assumed [26].

For all of the animal studies, a time of exposure is provided in the study in addition to the concentration of irritant gas for both sensory and pulmonary irritant effects. Therefore, the irritant gas compilation tables will always provide the exposure time for each concentration value or range of values. Again, with sensory irritation, an immediate effect should be assumed. For all cases, any evidence of concentrations that result in pulmonary effects (to both humans and animals) will be placed in the serious category (Category 3) unless the pulmonary effects are labeled by experts as mild or transient, in which case the concentrations are categorized as moderate to serious (Category 2).

5.2.1. Hydrogen Chloride (HCl)

Hydrogen chloride (HCl) is a colorless gas with a pungent, even suffocating odor, which is the product of the decomposition of any chloride-containing product, e.g., polyvinyl chloride. HCl is a sensory irritant as well as a pulmonary irritant [2,63].

The compilation data for HCl are presented in Table 5, which includes both human and animal exposure studies. From each study or secondary source, the concentration of HCl is presented and where known, the exposure time is also listed. Exposure times are only relevant when pulmonary effects are a consequence of the exposure. The data presented in the compilation data are taken from studies of healthy subjects and animals only.

Table 5. Hydrogen Chloride

Category 1: Minor effects unlikely to affect job performance				
$\mu\text{l/l}$	Time (min)	Subject	Notes	References
≥ 5 to 10	NA	Human	Immediately irritating; adaptation can occur	[64]
5 to 10	NA	Human	Mild irritants of the mucous membranes	[45]
107	30	Guinea pig	Exercising; mild irritation	[65]
Category 2: Moderate to major effects that may negatively affect job performance				
10 to 50	NA	Human	Irritation of the throat (35 $\mu\text{l/l}$); serious sensory irritation at end of range; can tolerate range for a few hours	[1,45,64,66]
500	15	Baboon	No impairment of pulmonary functions; however some moderate irritation effects	[67]
520	15	Guinea pig	Decrease in respiratory rate in moderate irritation range	[68]
120	10	Mouse	Mild to moderate nasal effects	[69]
200, 295	15	Rat	Decrease in respiratory rate in moderate irritation range	[17]
Category 3: Major effects that are likely to render an individual unable to complete job tasks				
50 to 100	NA	Human	Barely tolerable, severe sensory irritation	[1,45,66,70,71]
1 000	NA	Human	Incapacitation	[26]
5 000; 10 000	15	Baboon	Severe effects during exposure; (pulmonary hemorrhages)	[67]
11 400	5	Baboon	Able to perform escape task after 5 min exposure (permanent lung damage after exposure)	[43]
16 570; 17 290	5	Baboon	Able to perform task but died several weeks after exposure	[43]
140; 162	16.5; 1.3	Guinea pig	'E-Incapacitation' – during exercise, the trained guinea pig collapsed and could no longer run	[65]
3 940	15	Guinea pig	Deaths shortly after exposure (lung damage observed postexposure)	[68]
475	15	Mouse	Only moderate sensory irritation; however, 4/9 died postexposure (contradicts with other data)	[68]
2 550	15	Mouse	Died days after exposures (moderate lung edema found in one animal)	[68]
3 890	15	Rats	Moderate to severe sensory effects; no deaths postexposure	[68]

Due to the spread and difference in the data displayed in the compilation table (Table 5), it is imperative to summarize these data. Therefore, a data summary table (Table 6) is presented to provide ranges of healthy human (and primate) data for each severity category. Even though animal data are included in the compilation table, only human and primate data will be summarized due to the uncertainty in species differences between human/primates and other animals. Almost all of the human data from HCl exposures are collected from secondary sources.

Table 6. Summary of Data on Hydrogen Chloride

	Concentration ($\mu\text{l/l}$)	Time (min)	Subject
Category 1 ($\mu\text{l/l}$)	5 to 10	NA	Human
	---	---	Baboon
Category 2 ($\mu\text{l/l}$)	10 to 50	NA	Human
	500 (one data point)	15	Baboon
Category 3 ($\mu\text{l/l}$)	50 to 1 000	NA	Human
	5 000 to 17 290	5 to 15	Baboon

There are limited data presented on those effects that are unlikely to affect job performance (Category 1). Elkins [64] found that greater than and equal to 5 $\mu\text{l/l}$ of HCl is immediately irritating and at concentrations up to 10 $\mu\text{l/l}$ of HCl, workers developed some tolerance to the exposure. Therefore, a range of 5 $\mu\text{l/l}$ to 10 $\mu\text{l/l}$ is provided for Category 1. This range is also supported by data from Kimmerle [45].

Secondary sources have identified that 10 $\mu\text{l/l}$ to 50 $\mu\text{l/l}$ of HCl can negatively affect job performance (Category 2) [1,45,64,66]. While some of these sources mention that irritation can be immediate at these levels, especially throat irritation around 35 $\mu\text{l/l}$, Henderson and Haggard [66] state that 10 $\mu\text{l/l}$ to 50 $\mu\text{l/l}$ is maximum concentration tolerable for one hour. It is always more conservative to assume that effects begin immediately at these levels.

In Category 2, data are also provided for primates exposed to HCl. Kaplan et al. [67] found that baboons could withstand 500 $\mu\text{l/l}$ of HCl for 15 min with only moderate sensory impairments and no impairment to pulmonary functions. This concentration of 500 $\mu\text{l/l}$ is significantly higher than the suggested concentrations for humans that will produce the same physiological effects. While data from Kaplan et al. [67] are outliers in relation to the human data from secondary sources, it is important to report it since a baboon is considered a good model for human exposure to smoke gases.

Secondary sources also provide the range of HCl that can produce major effects in humans rendering them unable to perform job duties [1,45,66,70,71]. From 50 $\mu\text{l/l}$ to 100 $\mu\text{l/l}$ of HCl, it is suggested that humans encounter severe sensory irritation. Also, the data range for humans in Category 3 is bounded by the concentration likely to cause incapacitation in humans provided by the ISO [26]. At this concentration, humans are unable to take constructive action to effect their own escape. Therefore, this concentration (1 000 $\mu\text{l/l}$ for HCl) was placed as the upper bound for the third category.

Also listed in Category 3, are data from baboon studies [43,67]. Kaplan et al. [67] found that baboons could withstand up to 17 290 $\mu\text{l/l}$ of HCl and still perform the task assigned to them. Baboons exposed to 16 570 $\mu\text{l/l}$ and 17 290 $\mu\text{l/l}$ did perish after exposure. Baboons at other concentrations (5 000 $\mu\text{l/l}$, 10 000 $\mu\text{l/l}$, and 11 400 $\mu\text{l/l}$) survived at these concentration but experienced symptoms of pulmonary damage postexposure.

5.2.2. Hydrogen Fluoride (HF)

Hydrogen fluoride (HF) is a colorless, highly irritating and corrosive gas. HF is severely irritating to the eyes and nasal passages and at low concentrations, can be effectively scrubbed from the inhaled air, remaining in the anterior nasal passage. However, like other irritants, at higher concentrations, HF is likely to penetrate the lower respiratory tract and cause damage to the lungs [72]. In comparison with HCl, it is more likely that the body can effectively scrub a larger amount of HF in the nasal cavity, resulting in less penetration of HF into the lungs and less damage to the lower respiratory tract. Humans may experience effects to the lower respiratory tract especially during heavy exercise or physical exertion [72].

The compilation data for HF are presented in Table 7, which includes both human and animal exposure studies. From each study or secondary source, the concentration of HF is presented and where known, the exposure time is also listed. Exposure times are only relevant when pulmonary effects are a consequence of the exposure. The data presented in the compilation data are taken from studies of healthy subjects and animals only.

Table 7. Hydrogen Fluoride

Category 1: Minor effects unlikely to affect job performance				
$\mu\text{l/l}$	Time (min)	Subject	Notes	References
2.6 to 4.7	Days	Human	Slight irritation to skin, nose, and eyes	[73,74]
3 to 6.3	60	Human	Few experienced upper respiratory irritation; potential overreporting (1) of tightness in chest	[75]
32	3	Human	Mild irritation to eyes and nose; discomfort experienced	[76]; supported by [45]
460	15	Dog	Mild eye, nasal and respiratory irritation	[77]
157	60	Dog	Mild eye, nasal and respiratory irritation	[77]
61	300	Guinea pig Rabbit	Mild irritation to respiratory tract	[76]
103, 126	15	Rat	Occasional to mild eye, nasal irritation	[77]
307, 376	15	Rat	Slight to mild eye, nasal irritation	[77]
Category 2: Moderate to major effects that may negatively affect job performance				
61	1	Human	Eye and nasal irritation; irritation of breathing tracts	[76]; supported by [45]
666, 243	15, 60	Dog	Moderate eye, nasal and respiratory irritation, signs of discomfort	[77]
749, 590, 291	5, 15, 60	Rat	Moderate eye, nasal irritation	[77]
1 669	10	Rat	Inflammation, hemorrhage of the nasal area (moderate to severe ratings by experts)	[78,79]
100 to 1 000, 1 300	30, 30	Rat	Inflammation, hemorrhage of the nasal area; no damage to lungs; moderate irritation shown by respiratory rate	[80,81]
Category 3: Major effects that are likely to render an individual unable to complete job tasks				
50 to 100	NA	Human	Dangerous to life after a few minutes	[45,66]
120	NA	Human	Severe sensory	[1,70] supported by [45]
122	1	Human	Highest concentration that can be voluntarily tolerated for > 1 min	[76]
500	NA	Human	Incapacitation	[26]
18.5	6hr/days	Primate	Exposure tolerable; damage to kidneys	[82]
54	360	Guinea pig	Some liver and kidney damage	[76]
54	360	Rabbit	Some liver and kidney damage	[76]
854	15	Rabbit	Moderate sensory and lung hemorrhage	[77]
1247	15	Rabbit	Severe sensory and respiratory distress	[77]
1438	5	Rat	Severe sensory irritation only	[77]
2 432, 1 410, 1 377, 489	5, 15, 30, 60	Rat	Severe sensory irritation and respiratory distress	[77]
6 392	2	Rat	Moderate to severe damage to nasal passage; acute lung inflammation	[78,79]
7 014, 3 847, 1 224	10,10, 60	Rat	Moderate to severe sensory irritation; respiratory distress	[78,79]

A data summary table (Table 8) is presented to provide ranges of healthy human data for each severity level category. Even though most of the compilation table data are provided by animal studies, only data on human exposures will be summarized in the summary table. The human data on HF exposures are collected from experimental studies and secondary sources.

Table 8. Summary of Data on Hydrogen Fluoride

	Concentration (µl/l)	Time (min)	Subject
Category 1 (µl/l)	2.6 to 32	NA	Human
	---	---	Primate
Category 2 (µl/l)	61 (one data point)	1	Human
	---	---	Primate
Category 3 (µl/l)	50 to 500	NA	Human
	---	---	Primate

The data presented in Category 1 can be grouped into a HF range of 2.6 µl/l to 32 µl/l. Humans exposed to this range have experienced effects that are unlikely to affect job performance. Data from longer exposures of one hour or even over days show that humans exposed to 2.6 µl/l to 6.3 µl/l have experienced mild irritation to the upper respiratory tract [73,74,75]. More immediate effects have been experienced by humans exposed to approximately 32 µl/l of HF [76]. This range is supported by Kimmerle [45]. Therefore, a range of 2.6 µl/l to 32 µl/l is provided for a concentration range that can produce mild effects unlikely to affect job performance in healthy adults.

Only one data point from a human study could be found for a Category 2. Machle et al. [76] found that humans exposed to 61 µl/l immediately developed eye and nose irritation and irritation of the breathing tracts. Although this information is important, it is not enough to produce a summary range for Category 2.

The data in Category 3 can be grouped into a range of HF concentrations from 50 µl/l to 500 µl/l. First, secondary sources suggest that 50 µl/l to 100 µl/l of HF is dangerous to life after a few minutes and that 120 µl/l can produce severe sensory irritation in humans [1,45,66,70]. Supported by these data, Michle et al. [76] found that 122 µl/l was the highest concentration that could be voluntarily tolerated by humans for greater than one min. While these concentrations are at the lower end of the data range for Category 3, this range is bounded by the concentration likely to cause incapacitation in humans provided by the ISO [26]. At this concentration, humans are unable to take constructive action to effect their own escape. Therefore, this concentration (500 µl/l for HCl) was placed as the upper bound for the third category.

5.2.3. Hydrogen Bromide (HBr)

Hydrogen Bromide (HBr) is a colorless, corrosive gas that is severely irritating to the eyes, skin and nasal passages. Like many other irritants, at high concentrations, HBr may penetrate to the lower respiratory tract, possibly causing serious effects such as edema or hemorrhage [83].

The compilation data for HBr are presented in Table 9, which includes both human and animal exposure studies. From each study or secondary source, the concentration of HBr is presented and where known, the exposure time is also listed. Exposure times are only relevant when pulmonary effects are a consequence of the exposure. The data presented in the compilation data are taken from studies of healthy subjects and animals only. Also, since there are limited data available for human or animal exposure to HBr, data are provided in Table 9 for categories two and three only. No exposure data to HBr were found for Category 1.

Table 9. Hydrogen Bromide

Category 1: Minor effects unlikely to affect job performance				
$\mu\text{l/l}$	Time (min)	Subject	Notes	References
---	---	---	No data are provided for Category 1	---
Category 2: Moderate to major effects that may negatively affect job performance				
3 to 6	NA	Human	Nose and throat irritation (no eye irritation)	[84]
Category 3: Major effects that are likely to render an individual unable to complete job tasks				
100	NA	Human	Severe sensory irritation	[1,70]
1 000	NA	Human	Incapacitation	[26]
100 to 1 000	30	Rat	Moderate to severe nasal damage; no damage to the lungs; no deaths	[80,81]
1 300	30	Rat	Moderate to severe nasal damage; no damage to the lungs; 8 % mortality	[80]

A data summary table (Table 10) is presented to provide ranges of healthy human data for severity levels two and three. Only human data are reviewed in the summary table. The human data of HBr exposures are collected from experimental studies and secondary sources.

Table 10. Summary of Data on Hydrogen Bromide

	Concentration ($\mu\text{l/l}$)	Time (min)	Subject
Category 1 ($\mu\text{l/l}$)	---	---	Human
	---	---	Primate
Category 2 ($\mu\text{l/l}$)	3 to 6	NA	Human
	---	---	Primate
Category 3 ($\mu\text{l/l}$)	100 to 1 000	NA	Human
	---	---	Primate

Only one human study (with multiple data points) is provided for Category 2. Connecticut State Department of Health [84] found that humans exposed 3 $\mu\text{l/l}$ to 6 $\mu\text{l/l}$ of HBr experienced nose and throat irritation (without reports of eye irritation). This study neglected to mention the severity of the irritation; therefore, the reader should know that mild irritation is possible at these levels. As the data are unclear, the data were placed in Category 2 rather than Category 1.

The data in Category 3 are provided only by secondary sources. Secondary sources suggest that 100 µl/l of HF can produce severe sensory irritation in humans [1,70]. This concentration is used as the lower bound of the data summary for Category 3. The upper bound of this range is provided by the concentration likely to cause incapacitation in humans provided by the ISO [26]. At this concentration, humans are unable to take constructive action to effect their own escape. Therefore, the data summary provided for Category 3 is 100 µl/l to 1 000 µl/l.

5.2.4. Acrolein (C₃H₄O)

Acrolein (C₃H₄O) is a colorless or yellowish liquid at ambient temperature and pressure that has a strong, acrid, pungent odor. Acrolein is highly irritating to the upper respiratory tract and eyes and is a potent irritant at low concentrations and short exposure durations [85]. At higher concentrations and longer exposure times, pulmonary edema is possible.

The compilation data for acrolein are presented in Table 11, which includes both human and animal exposure studies. From each study or secondary source, the concentration of acrolein is presented and where known, the exposure time is also listed. Exposure times are only relevant when pulmonary effects are a consequence of the exposure. The data presented in the compilation data are taken from studies of healthy subjects and animals only.

Table 11. Acrolein

Category 1: Minor effects unlikely to affect job performance				
µl/l	Time (min)	Subject	Notes	References
0.1 to 0.3	NA	Human	Eye irritation, nose irritation, mild decrease in respiratory rate	[86]
Category 2: Moderate to major effects that may negatively affect job performance				
0.4 to 0.6	NA	Human	Throat irritation at 0.43 µl/l, moderate decrease in respiratory rate	[86]
0.8	NA	Human	Lacrimation, irritation of mucous membranes	[45,87]
<200	10	Dog	Mild pulmonary edema developed postexposure	[88]
Category 3: Major effects that are likely to render an individual unable to complete job tasks				
1.0 to 5.5	NA	Human	Severe sensory irritation	[1,45,66,70,71,89]
30	NA	Human	Incapacitation	[26]
1 025, 2 780	5	Baboon	Baboons can escape chamber; not incapacitating, however, pulmonary complications and some deaths after exposure (some survived)	[43]
200 to 300	10	Dog	Pulmonary edema consistently found postexposure	[88]

A data summary table (Table 12) is presented to provide ranges of healthy human (and primate) data for each severity level category. The human data on acrolein exposures are collected from experimental studies and secondary sources.

Table 12. Summary of Data on Acrolein

	Concentration ($\mu\text{l/l}$)	Time (min)	Subject
Category 1 ($\mu\text{l/l}$)	0.1 to 0.3	NA	Human
	---	---	Baboon
Category 2 ($\mu\text{l/l}$)	0.4 to 0.8	NA	Human
	---	---	Baboon
Category 3 ($\mu\text{l/l}$)	1 to 30	NA	Human
	1 025 to 2 780	5	Baboon

There are limited data presented on those effects that are unlikely to affect job performance (Category 1). Weber-Tschopp et al. [86] found that concentrations between 0.1 $\mu\text{l/l}$ and 0.3 $\mu\text{l/l}$ of acrolein caused healthy adults to experience mild eye and nose irritation and a small decrease in respiratory rate. With no other data available in this category, a range of 0.1 $\mu\text{l/l}$ to 0.3 $\mu\text{l/l}$ is provided. This range of data is provided from the same experimental study.

For Category 2, experimental studies on humans [86,87] supported by secondary sources [45] provide a range of 0.4 $\mu\text{l/l}$ to 0.8 $\mu\text{l/l}$ of acrolein. In this range, healthy adults experienced throat irritation (0.43 $\mu\text{l/l}$), a decrease in respiratory rate that signifies moderate sensory irritation, and lacrimation (or tearing of the eyes). Therefore, between 0.4 $\mu\text{l/l}$ and 0.8 $\mu\text{l/l}$ of acrolein, humans experienced effects that may negatively influence their job performance.

An experimental study [89] and secondary sources [1,45,66,70,71] also provide the range of acrolein that can produce major effects in humans rendering them unable to perform job duties. From 1.0 $\mu\text{l/l}$ to 5.5 $\mu\text{l/l}$ of acrolein, it is suggested that humans encounter severe sensory irritation. Also, the data range for humans in Category 3 is bounded by the concentration likely to cause incapacitation in humans provided by the ISO [26]. At this concentration, humans are unable to take constructive action to effect their own escape. Therefore, this concentration (30 $\mu\text{l/l}$ for acrolein) was placed as the upper bound for the third category.

However, also listed in Category 3, are data from a study on the effects of acrolein on baboons [43]. Kaplan et al. [43] found that baboons could still perform an “escape task” without succumbing to incapacitation at acrolein concentrations of 1 025 $\mu\text{l/l}$ and 2 780 $\mu\text{l/l}$. Baboons exposed to these concentrations did experience pulmonary complications postexposure and some died.

5.2.5. Formaldehyde (CH_2O)

Formaldehyde (CH_2O) is a colorless, flammable gas with a pungent, even suffocating odor [90]. At low concentrations, formaldehyde primarily affects the upper respiratory tract. Due to its solubility in water, formaldehyde becomes scrubbed by the nasal passages of humans and

rodents preventing it from reaching the lower respiratory tract. At higher concentrations formaldehyde becomes extremely irritating, affecting both the upper and lower respiratory tract [90].

The compilation data for formaldehyde are presented in Table 13, which includes both human and animal exposure studies. From each study or secondary source, the concentration of formaldehyde is presented and where known, the exposure time is also listed. Exposure times are only relevant when pulmonary effects are a consequence of the exposure. The data presented in the compilation data are taken from studies of healthy subjects and animals only.

Table 13. Formaldehyde

Category 1: Minor effects unlikely to affect job performance				
$\mu\text{l/l}$	Time (min)	Subject	Notes	References
>0.3	NA	Human	Eye irritation	[91,92]
0.9 to 1	NA	Human	Slight irritation, no pulmonary effects	[92,93,94]
0.1 to 1.6	NA	Human	Slight irritation of eyes and nose; no effect on performance of math tests or number transfer tasks	[45,95,96]
2.0 to 2.1	NA	Human	Mild nose and throat irritation during exercise	[96,97,98]
6; 15	360	Rat	Mild decrease in respiratory rate	[99,100]
20; 30	360	Rat	Mild effects	[101]
Category 2: Moderate to major effects that may negatively affect job performance				
2	40	Human	Moderate sensory irritant effects (at rest and exercising subjects)	[102,103,104,105]
3 ^a	60 to 180	Human	Moderate sensory irritation; small, transient decrements in pulmonary function in exercising healthy subjects	[97,98,106,107,108]
13.8	30	Human	Eye irritation with mild lacrimation, then adaptation	[87]; supported by [109]
6	360	Monkey	Eye irritation, mild lacrimation	[110]
Category 3: Major effects that are likely to render an individual unable to complete job tasks				
5	5	Human	Severe eye irritation	[111]
5 to 10	NA	Human	Severe sensory irritation	[1,45,70,71]
20	NA	Human	Lacrimation within seconds; eye, nose and throat irritation intolerable	[112]
250	NA	Human	Incapacitation	[26]

^a Without exercise, no decrease in pulmonary functions

A data summary table (Table 14) is presented to provide ranges of healthy human (and primate) data for each severity level category. The human data on formaldehyde exposures are collected from experimental studies and secondary sources.

Table 14. Summary of Data on Formaldehyde

	Concentration (µl/l)	Time (min)	Subject
Category 1 (µl/l)	0.3 to 2.1	NA	Human
	---	---	Monkey
Category 2 (µl/l)	2 to 13.8	+ 30 (to 180) ^a	Human
	6 (one data point)	360	Monkey
Category 3 (µl/l)	5 to 250	NA	Human
	---	---	Monkey

^a 2 µl/l for 40 min, 3 µl/l for 60 to 180 min, and 13.8 µl/l for 30 min

Formaldehyde is the one gas presented in this report that has had a great deal of human-subject clinical studies performed. The studies presented in the compilation table (Table 13) conform to a range of 0.3 µl/l to 2.1 µl/l of formaldehyde for Category 1. Clinical studies have shown that concentrations of formaldehyde in this range have produced only mild effects in humans, including slight sensory irritation [45,91,92,93,94, 95,96], and mild nose and throat irritation during exercise [96,97,98]. One study demonstrated that 1.6 µl/l of formaldehyde did not affect performance of math tests or number transfer tasks [95]. Therefore, this range of concentrations has produced minor effects in healthy adults that are unlikely to affect job performance.

Clinical studies were used to create a formaldehyde range of 2 µl/l to 13.8 µl/l for Category 2. At the lower level of this range, exposures of 2 µl/l or 3 µl/l of formaldehyde caused moderate sensory irritation and even slight decrements in pulmonary function in exercising healthy adults [97,98,102,103,104,105,106,107,108]. At the upper bound of this range Sims and Pattle [87] and Douglas [109] found that 13.8 µl/l of formaldehyde led to eye irritation and tearing of the eyes. Therefore, the range of 2 µl/l to 13.8 µl/l of formaldehyde has produced major effects that may negatively influence job performance.

Clinical studies [111,112] and secondary sources [1,45,66,70,71] provide evidence for the range of formaldehyde that can produce major effects in humans rendering them unable to perform job duties. From 5 µl/l to 10 µl/l of formaldehyde, it is suggested that humans encounter severe sensory irritation. Also, Barnes and Speicher [112] found that humans experienced intolerable eye, nose and throat irritation at 20 µl/l of formaldehyde. The data range for humans in Category 3 is bounded by the concentration likely to cause incapacitation in humans provided by the ISO [26]. At this concentration, humans are unable to take constructive action to effect their own escape. Therefore, this concentration (250 µl/l for formaldehyde) was placed as the upper bound for the third category.

5.2.6. Nitrogen Dioxide (NO₂)

Nitrogen dioxide (NO₂) is a reddish-brown gas with a sweet odor. NO₂ irritates the mucous membranes and with more severe exposures, can cause pulmonary edema represented by signs of chest pain, cough, dyspnea, and other serious symptoms [113].

The compilation data for NO₂ are presented in Table 15, which includes both human and animal exposure studies. From each study or secondary source, the concentration of NO₂ is presented and where known, the exposure time is also listed. Exposure times are only relevant when pulmonary effects are a consequence of the exposure. The data presented in the compilation data are taken from studies of healthy subjects and animals only.

Table 15. Nitrogen Dioxide

Category 1: Minor effects unlikely to affect job performance				
$\mu\text{l/l}$	Time (min)	Subject	Notes	References
10 to 20	NA	Human	Mild irritation to eyes, nose and upper respiratory tract	[45]
125, 52, 39	5, 15, 60	Dog	Mild sensory effects	[114]
20	24 hours	Dog, Rabbit, Guinea pig	Minimal signs of irritation	[115]
Category 2: Moderate to major effects that may negatively affect job performance				
5	120	Human	Moderate effects on airway and O ₂ partial pressure	[116]
10, 15	120	Monkey	Only mild respiratory effects; mild changes to the lung	[117]
164, 85, 53	5, 15, 60	Dog	Mild sensory effects, but some respiratory distress	[114]
9, 13	120	Guinea pig	Moderate respiratory effects	[118]
5.2, 6.5	240	Guinea pig	Moderate respiratory effects	[118]
Category 3: Major effects that are likely to render an individual unable to complete job tasks				
30	120	Human	Mild effects after 30 min; Burning sensations in chest, severe cough after 70 min; barely tolerable at 120 min	[119]
50	NA	Human	Distinct irritation	[45]
80	NA	Human	Severe sensory irritation	[1,70,71]
80	3 to 5	Human	Tightness of chest	[45]
250	NA	Human	Incapacitation	[26]
35	120	Monkey	Portion of lung collapsed, bronchi inflamed	[117]

Aside from what is presented in the compilation table (Table 15), there are other data available that provide some context for the data points in the table. The AEGl document for NO₂ suggests that there is a threshold level for NO₂ before which pulmonary functions are found to affect healthy adults [113].

Data show that no changes in pulmonary functions were found at concentrations of 1 $\mu\text{l/l}$ (2 hours), 2 $\mu\text{l/l}$ (3 hours), 2 $\mu\text{l/l}$ (4 hours), 3 $\mu\text{l/l}$ (2 hours), or 2.3 $\mu\text{l/l}$ (5 hours) for healthy subjects [120,121,122,123]. Additionally, no pulmonary effects were found in healthy adults engaging in intermittent light or heavy exercise at 4 $\mu\text{l/l}$ (1 hour) [124]. However, moderate irritation to the respiratory tract were found at 5 $\mu\text{l/l}$ (2 hours) [116], which is listed in Table 15.

Table 15 also does not show any data on exposures of rats and mice to NO₂. Many of these studies discussed here performed experiments on rats to discover those concentrations of NO₂ over certain time durations at which serious pulmonary effects would occur (e.g., pulmonary edema, increased lung wet weights, etc.). These pulmonary effects can be more serious than the three categories listed in Table 15. Therefore, instead of listing data from rat and mice studies in Table 15, a list of concentrations of NO₂ (with time durations) that did not produce serious pulmonary effects on rats and mice are provided here:

10 µl/l (30 min); 25 µl/l to 50 µl/l (up to 15 min) for rats [125]
 74 µl/l (5 min); 33 µl/l (15 min) for rats [114]
 20 µl/l (up to 24 hours) for rats and mice [115]

A data summary table (Table 16) is presented to provide ranges of healthy human and primate concentration data for each severity level category. The human data on NO₂ exposures are collected from experimental studies and secondary sources.

Table 16. Summary of Data on Nitrogen Dioxide

	Concentration (µl/l)	Time (min)	Subject
Category 1 (µl/l)	10 to 20	NA	Human
	---	---	Monkey
Category 2 (µl/l)	5 (one data point)	120	Human
	10 to 15	120	Monkey
Category 3 (µl/l)	30 to 250	NA	Human
	35 (one data point)	120	Monkey

For Category 1, a secondary source [45] suggests a concentration range for healthy adults that is likely only to produce mild irritation to the upper respiratory tract. Unfortunately, these are the only data available for Category 1. Therefore, 10 µl/l to 20 µl/l of NO₂ is listed as causing only minor effects in healthy adults that are unlikely to affect job performance.

Only one human data point is provided in Category 2 (5 µl/l for 120 min). At this concentration, von Neiding et al. [116] found that exposure to NO₂ produced moderate irritation to the respiratory tract. However, this concentration actually falls below the range of data provided in Category 1. This is likely the result of the long exposure time because breathing NO₂ for long periods of time at lower concentrations will cause a buildup of nitric acid throughout the respiratory system (primarily in the upper respiratory tract).

On the other hand, a study performed by Henry et al. [117] found that monkeys experienced mild respiratory effects and mild changes to the lung (which is why this is grouped in Category 2) after 120 min of 10-15 µl/l of NO₂. This provides support for the concentration data range listed in Category 1 as well as provides another viable data point for the range presented in Category 2.

Both experimental and secondary data provide evidence for the data range presented for Category 3 (30 µl/l to 250 µl/l of NO₂). Henschler et al. [119] exposed humans to 30 µl/l of NO₂ for 120 min and found that while only mild effects were experienced 30 min into the exposure, subjects eventually found the concentration barely tolerable after 120 min. Also, secondary

sources suggest that humans experience distinct irritation at 50 $\mu\text{l/l}$ [45] and severe sensory irritation and even tightness in the chest area at 80 $\mu\text{l/l}$ [1,45,70,71]. Therefore, these values provide the lower bounds for the Category 3 range of NO_2 concentrations. This data range is also bounded by the concentration likely to cause incapacitation in humans provided by the ISO [26]. At this concentration, humans are unable to take constructive action to effect their own escape. Therefore, this concentration (250 $\mu\text{l/l}$ for NO_2) was placed as the upper bound for the third category.

5.3. Heat – Tables and Summaries

During a fire, individuals can be exposed to heat in the form of conductive heat (the temperature of the hot object in $^{\circ}\text{C}$), convective heating (hot gases in $^{\circ}\text{C}$) and radiative heating (heat flux in kW/m^2). Exposure to conductive, convective and radiative heat can cause very serious effects on humans. Harmful effects on people due to heat depend on both the level of the heat (temperature or heat flux) and the period of time over which exposure to heat has taken place.

The three ways in which people can develop serious effects due to heat are hyperthermia (i.e., heat stroke), body surface burns, and burns of the respiratory tract. Hyperthermia occurs when people are exposed to heated environments at temperatures too low to cause burns, for an extended period of time (15 min or more) [1]. Exposure to these conditions gradually increases the individual's core body temperature to unhealthy levels [126], which can lead to unconsciousness or even death.

Skin burns can occur due to conducted, convective, and radiant heat [1,26]. Skin burns occur from a rise in skin temperature to a point where damage to the skin occurs [6]. Skin burns from conducted heat can occur anytime an individual comes in contact with a hot surface. Unless an individual is near to the fire and actually comes in contact with hot materials or the fire itself, these types of burns are unlikely to occur. Skin burns can also occur from convective heat, where any surface of the person comes in contact with hot gases from the fire. Last, radiant energy from a fire can travel from the source of the heat and be absorbed by any surface that it encounters, including human beings.

The last effect of heat on people is thermal damage or burns to the respiratory tract. If hot gases (at certain levels and humidity) are inhaled by an individual, burns to the larynx are possible, which may result in edema of the larynx and even damage to the lungs if not treated. However, thermal burns to the respiratory tract will not occur unless the temperatures and/or humidity are such that facial burns are likely to occur first [1]. Therefore, this report will focus on heat levels likely to cause skin burns, since these are likely to happen before any thermal damage to the respiratory tract.

The compilation data for heat are presented in Table 17. Data from studies of human exposure (exposed skin) to both radiant heat and convective heat are included in the compilation table. The table organizes data from heat studies into those levels of heat (heat flux and temperature) that produce only minor effects, moderate to major effects the may negatively interfere with job

performance, and major effects likely to render an individual unable to complete job tasks. When using temperatures listed in either the compilation data or the summary table for convective heat, the reader should know that the effects depend not only on the temperature of the air but also the amount of humidity or volume of water vapor present in the air. Hot air at lower temperatures with higher humidity can cause the same kinds of effects as higher temperature with lower humidity.

Table 17. Heat

Category 1: Minor effects unlikely to affect job performance					
kW/m ²	°C	Time (min)	Subject	Notes	References
<2.5		30 min	Humans	Can be tolerated without significant consequences	[1,26,127]
<1.7		Any	Humans	No pain experienced by any test subject below this incident heat flux	[128]
<1.1		Any		Threshold below which individuals can tolerate for a time without pain	[129]
	+27		Humans	If higher temperatures, or higher humidity (higher than 35 % to 60 % at 27 °C), people are uncomfortable	[130]
Category 2: Moderate to major effects that may negatively affect job performance					
---	---	---	---	No data are provided for Category 2	---
Category 3: Major effects that are likely to render an individual unable to complete job tasks					
16.7 12.6 8.4 6.2 5.2 4.1		2 s 2.5 s 5.5 s 8 s 10 s 13 s	Humans	Onset of pain to exposed skin	[131]
23.5 10.5 2.5		1.6 s 5 s 40 s	Humans	Onset of pain to exposed skin	[132]
16.7 12.6 8.4 6.2 4.1		6 s 8 s 13 s 21 s 34 s	Humans	Onset of blistering to exposed skin	[131,133]
21 12.5 8.4 3.3 2		2 s 5 s 10 s 30 s 50 s	Humans	Unbearable pain	[129]
	146	NA	Humans	Without moisture in air, this is the maximum survivable breathing air temperature	[26]
	120 dry 80 sat. ^a	15+ min	Humans	Below this threshold, hyperthermia possible over longer exposures (15+ min)	[1]
	100 dry 120 140 160 180	12 min 7 min 4 min 2 min 1 min	Humans	Above this, onset of considerable pain in minutes along with production of burns	[1,26,126]

^a Saturated air

As the Table 17 shows, Category 1 included those levels of heat that could be tolerated for a specific amount of time without significant consequences. There are little data on this level;

therefore, the author also included information on temperatures that cause discomfort in humans (Category 1).

No data are provided for Category 2. Research studies on the effects of heat consistently report heat levels likely to cause pain, burns, and blisters to exposed skin. Heat levels high enough to cause the onset of pain, which could be included Category 2, occur within seconds and are quickly followed (again in seconds) by burns, blistering, and severe pain. Due to the small time frame within which these effects occur, all data on pain and burns are included in Category 3.

Category 3 includes those levels of heat that initiate considerable pain, burning and blistering of the skin, and the threshold temperature for hyperthermia to occur. There is no specific listing for incapacitation for this category, although the levels of heat included in this category are very painful and could be considered incapacitating.

In order to make sense of the data included in the compilation table, a summary table for radiant heat and convective heat is included below as Table 18. If the level of heat is known for certain spaces of the building where people are expected to be, these data can assist with assessments made on how long an individual can withstand the effects of heat until minor or even major effects are likely to occur.

The data summary for radiant heat provides the range of heat flux (kW/m^2) that, if sustained for less than 30 min, is likely to produce only minor effects (if any at all) (Category 1) and the threshold for heat flux above which considerable pain can begin in a matter of seconds (Category 3). There are not enough data provided on radiant heat to establish a range of data for Category 2 with any confidence. The research on radiant heat can be summarized in the following way:

Category 1: Humans can be exposed to radiant heat between 1.1 and 2.5 kW/m^2 for up to 30 min and experience minor effects which are unlikely to affect job performance [1,26,127,129].

Category 3: Humans exposed to radiant heat at or above 2.5 kW/m^2 are likely to experience burning of the skin and blistering within 30 s or less [1,129,131,132,133].

Therefore, a radiant heat flux at or greater than 2.5 kW/m^2 can cause very serious effects to individuals that may render them unlikely to complete job tasks (Category 3).

Summary data are also provided for convective heat. The data summary provides a threshold value ($^{\circ}\text{C}$) for Category 1 that, if exceeded, is likely to produce only minor effects and discomfort to the individual. Also a threshold value ($^{\circ}\text{C}$) is provided for Category 3 to predict considerable pain and hyperthermia. There are not enough data provided on convective heat to establish a range of data for Category 2 with any confidence. The research on convective heat can be summarized in the following way:

Category 1: Temperatures above 27 $^{\circ}\text{C}$ (35 % to 60 % humidity) or higher humidity at 27 $^{\circ}\text{C}$ can cause humans to experience discomfort [130].

Category 3: Humans exposed to dry air temperatures above 120 °C are likely to experience the onset of considerable pain along with the production of burns within minutes [1,26,126].

Category 3: Humans exposed to dry air temperatures below 120 °C (80 °C in saturated air) for longer than 15 min can develop hyperthermia [1].

Therefore, convective heat greater than 120 °C (within minutes) and below 120 °C (for a prolonged exposure greater than 15 min) can cause very serious effects to individuals that may render them unlikely to complete job tasks (Category 3).

Table 18. Summary of Data on Heat

	Categories	Heat	Time (min)
Category 1	Radiant Heat (kW/m ²)	1.1 to 2.5	Up to 30 min
	Convective Heat (°C)	+ 27 (lower limit)	NA
Category 2	Radiant Heat (kW/m ²)	---	---
	Convective Heat (°C)	---	---
Category 3	Radiant Heat (kW/m ²)	+ 2.5	NA
	Convective Heat (°C)	+ 120 - 120	Within minutes For greater than 15 min

Clothing has been shown to provide some protection against skin pain and blistering. Its effect, however, is difficult to quantify due to the range of clothing available, its thickness, its construction and materials, and how tight it fits against the skin [1,133,134,135,136]. While clothing does slow the process of skin burns, any exposed skin is susceptible to skin burns.

5.4. Smoke – Tables and Summaries

From the burning of materials, smoke contains fire gases as well as particulate matter and aerosols (suspended liquid droplets). Due to the fact that the wavelength of visible light is similar to the average size of the smoke particulates and aerosols, the passage of light through the smoke is obscured, also obscuring an individual’s vision through the smoke [2]. Smoke obscuration (or optical density of smoke) is related to its concentration and usually expressed as optical density per meter (m⁻¹) [4].

A high smoke obscuration is likely to affect an individual’s safety in a building. Exposures to thick, dense smoke can negatively affect an individual’s ability to see their surrounding environment, and in turn, affect their speed of movement throughout a smoke-filled space and their concentration on a job task [1,137]. The density of the smoke itself affects visibility as well as the irritancy of the smoke. In some cases, irritants can be so potent that individuals cannot open their eyes to see. For people wearing smoke masks, the density or thickness of the smoke is the main problem.

Table 19 is the compilation data table for research on the effects of smoke density. There are data from two main research studies presented in the table and the threshold values for safe escape are presented from other research in the field. The research results have been grouped into the three main categories by relating job performance to impairments to visibility, walking speed and steadiness and concentration.

Table 19. Smoke Density

Category 1: Minor effects unlikely to affect job performance				
Optical density (m ⁻¹)	Visibility (m)	Subject	Notes	References
0.087 to 0.13	6.7 to 10	Human	Walking speeds begin to be negatively affected at these densities	[4,137,138]
0.04 to 0.17	5 to 20	Human	Steadiness of subject (steadiness tester) begins to degrade - irritant smoke	[4,137,139]
Category 2: Moderate to major effects that may negatively affect job performance				
0.04	20	Human	Threshold for safe escape	[140]
0.065 0.22	13 4	Human	Threshold for safe escape – unfamiliar Threshold for safe escape – familiar	[4]
0.08 0.2	10 4.3	Human	Threshold for safe escape – unfamiliar Threshold for safe escape – familiar	[1]
0.087	10	Human	Threshold for safe escape	[141,142]
0.19	4.5	Human	Threshold for safe escape	[143]
0.72	1.2	Human	Threshold for safe escape	[144]
0.17 to 0.22	4 to 5	Human	Walking speeds are decreased by 50 % in irritant smoke	[4,137,138]
0.22 to 0.41	2.1 to 4	Human	Walking speeds are decreased by 50 % in nonirritant smoke	[4,137,138]
0.15 to 0.24	3.6 to 5.7	Human	Steadiness of the expert researcher (steadiness test) begins to degrade – irritant smoke	[4,137,139]
Category 3: Major effects that are likely to render an individual unable to complete job tasks				
0.22	4	Human	Walking speeds are decreased as if individual is walking in the dark – irritant smoke	[4,137,138]
0.43	2	Human	Walking speeds are decreased as if individual is walking in the dark – nonirritant smoke	[4,137,138]
1.7	0.5	Human	Incapacitation	[26]

Research has shown that smoke optical density has an effect on how far people can see through the smoke (visibility), how fast people can walk or move (walking speed), and how steady an individual can remain during a steadiness test^{††}. Results from these studies can be categorized into the three main categories using the following assumptions: 1) visibility can affect both an individual’s ability to escape a building and his/her ability to perform work function or tasks; 2) a decrease in walking speed can affect both an individual’s ability to escape and his/her ability to

^{††} Subjects, one at a time, sat at a table and manipulated a steadiness tester in a room filled with smoke. The subject was told to place a metal stylus into holes in a specific order without touching the hole edges with the stylus. The holes ranged in size from large to small, taking more concentration to place the stylus in the smaller holes without contact.

move around a building to perform a work task or tasks; and 3) a decrease in steadiness and concentration can affect an individual's ability to perform certain work tasks efficiently and correctly. Each research result has been categorized with this logic to identify the smoke optical density ranges that are likely to produce minor, moderate, and serious effects on an individual's job performance.

The data grouped into Category 1 show that smoke optical densities of 0.087 m^{-1} to 0.13 m^{-1} (6.7 m to 10 m visibility) begin to slow walking speeds of individuals and smoke optical densities of 0.04 m^{-1} to 0.17 m^{-1} in irritant smoke (5 m to 20 m visibility) begin to affect one's ability to concentrate on a steadiness task [137]. These values were grouped into Category 1 because at these smoke optical densities, the effects of smoke on functioning (speed and steadiness) were just beginning. It is assumed that at this level, smoke density will only produce minor effects on individuals that are unlikely to affect job performance.

The data grouped in Category 2 include threshold smoke density levels for safe escape for familiar and unfamiliar occupants, smoke optical densities that are reported to reduce walking speeds by 50 %, and smoke optical densities that are reported to reduce the steadiness or concentration of expert researchers of the steadiness tester study. The thresholds for safe escape were grouped into Category 2 because these densities specify the limit at which people can still safely escape a building. At these levels, it is assumed that people can still function enough to escape a building, however their escape (and therefore, their job performance) may be negatively affected. The data presented in Table 19 show a large spread of threshold values ranging from 0.04 m^{-1} (20 m visibility) to 0.72 m^{-1} (1.2 m visibility). There is no indication from any of the research which value(s) are more appropriate than the others. Other data presented in Category 2 show that smoke optical densities of 0.17 m^{-1} to 0.22 m^{-1} (4 m to 5 m visibility) for irritant smoke and 0.22 m^{-1} to 0.41 m^{-1} (2.1 m to 4 m visibility) for nonirritant smoke slow walking speeds of individuals by 50 % and smoke optical densities of 0.15 m^{-1} to 0.24 m^{-1} in irritant smoke (3.6 m to 5.7 m visibility) begin to affect an expert's ability to concentrate on a steadiness task [137]. At these lower visibility conditions (2.1 m to 5.7 m visibility), job performance may be negatively affected.

The data grouped in Category 3 include smoke optical densities that significantly decrease speeds and the value provided by ISO [26] for incapacitation due to smoke density. At specific values of smoke optical density, namely 0.22 m^{-1} (4 m visibility) for irritant smoke and 0.43 m^{-1} (2 m visibility) for nonirritant smoke, individuals' walking speeds are decreased to a value (0.3 m/s) as if they were walking in the dark [137]. At these levels of smoke density, individuals did not walk in a straight line and guided their hands along the wall for support during movement, therefore individuals cannot be relied upon to complete work tasks. This category is bounded by the ISO level of smoke optical density likely to produce incapacitation. Incapacitation for smoke density is defined as the inability of an individual to effect his/her own escape. At an optical density of 1.7 m^{-1} (i.e., a visibility of 0.5 m), an individual is no longer able to see one's hand in front of one's face. Therefore, an individual subjected to smoke at an optical density of 1.7 m^{-1} is considered unlikely to be able to complete job tasks.

Data from the smoke density compilation table are summarized in Table 20. Ranges of smoke optical densities (m^{-1}) and corresponding visibility distances (m) likely to produce Category 1, 2,

and 3 effects are found in the table for both nonirritant and irritant smoke. For all three categories, data ranges are taken directly out of the compilation table and listed in summary form in Table 20. The data that are not included in the summary table are the thresholds for safe escape due to the large spread in data with little indication of the value of one set of data over the other. Table 20 shows that lower smoke density values (higher visibility distances) are listed for irritant smoke due to the fact that the irritants negatively affect the eyes before the thickness of the smoke can influence visibility.

Table 20. Summary of Data on Smoke Density

	Categories	Optical Density (m⁻¹)	Visibility (m)
Category 1	Nonirritant smoke	0.087 to 0.13	6.7 to 10
	Irritant smoke	0.04 to 0.17	5 to 20
Category 2	Nonirritant smoke	0.22 to 0.41	2.1 to 4
	Irritant smoke	0.15 to 0.24	3.6 to 5.7
Category 3	Nonirritant smoke	1.7 to 0.43	0.5 to 2
	Irritant smoke	1.7 to 0.22	0.5 to 4

6. Conclusion

The purpose of this report is to summarize data on the amount of fire effluent (namely narcotic gases, irritant gases, heat and smoke) necessary to produce sublethal effects to humans. This study first identified and collected research studies that documented sublethal effects to humans and animals from exposures to the more common fire products. Once research studies were collected, each was reviewed and data on concentrations of fire product (gas, heat, or smoke) that produced sublethal effects on humans or animals were tabulated. Then, the data were categorized into three main severity levels. For each fire product, consistencies in the human and primate data were identified as well as any data that were inconsistent with the rest. To resolve any of these inconsistencies would require a more in-depth physiological analysis, which is outside of the scope of this report.

7. References

- 1 Purser, D.A.. 2008. "Toxicity assessment of combustion products," in *The SFPE Handbook of Fire Protection Engineering, 3rd edition*. National Fire Protection Association, Quincy, MA.
- 2 Gann, R.G. and N.P. Bryner. 2008. "Combustion products and their effects on life safety," in *The Fire Protection Handbook, 20th edition*, National Fire Protection Association, Quincy, MA.
- 3 Gann, R.G., V. Babrauskas, R.D. Peacock, and J.R. Hall. 1994. "Fire conditions for smoke toxicity measurement." *Fire and Materials* 18:193-199.
- 4 Jin, T. 2002. "Visibility and human behavior in fire smoke," in *The SFPE Handbook of Fire Protection Engineering, 3rd edition*. National Fire Protection Association, Quincy, MA.
- 5 U.S. Environmental Protection Agency Acute Exposure Guideline Levels. 2008. "Chemicals," <http://www.epa.gov/oppt/aegl/pubs/chemlist.htm>
- 6 *Engineering Guide: Predicting 1st and 2nd Degree Skin Burns from Thermal Radiation*, Society of Fire Protection Engineers, Bethesda, MD: 2000.
- 7 Anderson, R.A., K.N. Willetts, K.N. Cheng, and W.A. Harland. 1983. "Fire deaths in the United Kingdom 1976-82." *Fire Mater.* 7:67-72.
- 8 Yamamoto, D. 1976. "Acute combined effects of HCN and CO with the use of the combustion products from PAN (polyacrylonitrile)-gauze mixtures." *Z. Rechtsmed.* 78:303-311.
- 9 Higgins, E.A., V. Fiorca, A.A. Thomas, and H.V. Davis. 1972. "Acute toxicity of brief exposures to HF, HCl, NO₂, and HCN with and without CO." *Fire Technology* 8:120-130.
- 10 Crane, C.R. 1985. "Are the combined toxicities of CO and CO₂ synergistic?" *Journal of Fire Sciences* 3:143-144.
- 11 Edginton, J.A.G. and R.D. Lynch. 1975. *The Acute Inhalation Toxicity of CO from Burning Wood*. Fire Research Note NO. 1040, Fire Research Station, Salisbury, Wiltshire, England.
- 12 Crane, C.R., D.C. Sanders, and B.R. Endecott. 1989. "Inhalation Toxicology: IX. Times-to incapacitation for rats exposed to carbon monoxide alone, to hydrogen cyanide alone, and to mixtures of carbon monoxide and hydrogen cyanide." Technical Report DOT/FAA/AM-89/4.
- 13 Hartzell, G.E., H.W. Stacy, W.G. Switzer, D.N. Priest, and S.C. Packham. 1985. "Modeling of toxicological effects of fire gases: V. Mathematical modeling of intoxication of rats by combined carbon monoxide and hydrogen cyanide atmospheres." *Journal of Fire Sciences* 3:330-342.
- 14 Purser, D.A. and P. Grimshaw. 1984. "The incapacitative effects of exposure to thermal decomposition products of polyurethane foams." *Fire and Materials* 8:10-16..
- 15 Pitt, B.R., E.P Radford, G.H. Gurtner, and R.J. Traystman. 1979. "Interaction of carbon monoxide and cyanide on cerebral circulation and metabolism." *Arch. Environ. Health* 24: 354-359.
- 16 Levin, B.C., M. Paabo, J.L. Gurman, and S.E. Harris. 1987. "Effects of exposure to single or multiple combinations of the predominant toxic gases and low oxygen atmospheres produced in fires." *Fundamental and Applied Toxicity* 9:236-250.
- 17 Hartzell, G.E., H.W. Stacy, W.G. Switzer, D.N. Priest, and S.C. Packham. 1985. "Modeling of toxicological effects of fire gases: IV. Intoxication of rats by carbon monoxide in the presence of an irritant." *Journal of Fire Sciences* 3:263-279.
- 18 Hartzell, G.E. and H.W. Emmons. 1988. "The fractional effective dose model for assessment of toxic hazards in fires." *Journal of Fire Sciences* 6:356-362.
- 19 Moss, R.H., C.F. Jackson, and J. Seiberlich. 1951. "Toxicity of carbon monoxide and hydrogen cyanide gas mixtures." *Arch. Ind. Hyg. Occup. Med.* 4:53-64.
- 20 Rodkey, F.L. and H.A. Collison. 1979. "Effects of oxygen and carbon dioxide on carbon monoxide toxicity." *Journal of Combustion Toxicology* 1:191-235.
- 21 Nelson, G.L., E.J. Hixon, and E.P. Denine. 1978. "Combustion product toxicity studies of engineering plastics." *Journal of Combustion Toxicity* 5:222-238.
- 22 Wiczorek, C.J. and N.A. Dembsey. 2001. "Human variability correction factors for use with simplified engineering tools for predicting pain and second degree skin burns," *Journal of Fire Protection Engineering* 11:88-111.
- 23 Barrow, C.S. 1986. *Toxicology of the Nasal Passages*. New York: Hemisphere Publishing Corporation.
- 24 Jones, T.C., D.L. Dungworth, and U. Mohr, eds. 1996. *Monographs on Pathology of Laboratory Animals: Respiratory System*. New York:Springer Verlag.

- 25 Haldane, J.S. 1922. *Respiration*. New Haven, CT: Yale University Press.
- 26 ISO 13571. 2007. Life-Threatening Components of Fire—Guidelines for the Estimation of Time Available for Escape Using Fire Data, International Organization for Standardization, Geneva, Switzerland.
- 27 AEGl. 2005. "Interim acute exposure guideline levels: Carbon monoxide." For NAS/COT Subcommittee for AEGls. <http://www.epa.gov/oppt/aegl/pubs/tsd306.pdf>
- 28 Coburn, R.F., R.E. Forster and P.B. Kane. 1965. "Considerations of the physiological variables that determine the blood carboxyhemoglobin concentration in man." *Journal of Clinical Investigation* 44:1899-1910.
- 29 EPA, U. S. Environmental Protection Agency, 2000. Air quality criteria for carbon monoxide. EPA 600/P-99/001F, U.S. Environmental Protection Agency, Office of Research and Development, Washington, D.C.
- 30 Hazucha, M.J. 2000. "Effect of carbon monoxide on work and exercise capacity in humans." Chapter 5 in: Carbon Monoxide Toxicity. D.G. Penney (Ed.). CRC Press, Boca Raton, London, New York, Washington D.C., pp. 101-134.
- 31 WHO, World Health Organization. 1999. Environmental Health Criteria 213, Carbon Monoxide (Second Edition), IPCS, International Programme on Chemical Safety; World Health Organization, Geneva, Switzerland.
- 32 Kizakevich, P.N., M.L. McCartney, M.J. Hazucha, L.H. Sleet, W.J. Jochem, A.C. Hackney and K. Bolick. 2000. "Noninvasive ambulatory assessment of cardiac function in healthy men exposed to carbon monoxide during upper and lower body exercise." *Eur. J. Appl. Physiol.* 83:7-16.
- 33 Henderson, Y., H.W. Haggard, M.C. Teague, A.L. Prince and R.M. Wunderlich. 1921. "Physiological effects of automobile exhaust gas and standards of ventilation for brief exposures." *Journal of Industrial Hygiene* 3: 79-92.
- 34 Stewart, R.D., J.E. Peterson, E.D. Baretta, R.T. Bachand, M.J. Hosko and A.A. Herrmann. 1970. "Experimental human exposure to carbon monoxide." *Archives of Environmental Health* 21:154-164.
- 35 Gigiena Truda i Professional'nye Zabolevaniia. 1987. Labor Hygiene and Occupational Diseases. 31.
- 36 Burney, R.E., Wu, S.C., Nemiroff, M.J. 1982. "Mass carbon monoxide poisoning: Clinical effects and results of the treatment of 184 victims." *Ann Emerg Med* 11:394-399.
- 37 Ely, E.W., B. Moorehead, and E.F. Haponik. 1995. "Warehouse workers' headache: emergency evaluation and management of 30 patients with carbon monoxide poisoning." *Am. J. Med* 98:145-155.
- 38 Haldane, J. 1895. "The action of carbonic acid on man." *J. Physiol.* 18:430-462.
- 39 Purser, D.A. and K.R. Berrill. 1983. "Effects of carbon monoxide on behavior in monkeys in relation to human fire hazard." *Arch. Environ. Health* 38:308-315.
- 40 Ebisuno, S., M. Yasuno, Y. Yamada, Y. Nishino, M. Hori, M. Inoue and T. Kamada. 1972. "Myocardial infarction after acute carbon monoxide poisoning: Case report." *Angiology* 37:621-624.
- 41 Grace, T.W. and F.W. Platt. 1981. "Subacute carbon monoxide poisoning." *J. Am. Med. Assoc.* 246:1698-1700.
- 42 Sokal, J.A. and E. Kralkowska. 1985. "The relationship between exposure duration, carboxyhemoglobin, blood glucose, pyruvate and lactate and the severity of intoxication in 39 cases of acute carbon monoxide poisoning in man." *Arch. Toxicol.* 57:196-199.
- 43 Kaplan, H.L., Grand, A.F., Switzer, W.G., Mitchell, D.S., Rogers, W. R., and Hartzell, G.E. 1985. "Effects of combustion gases on escape performance of the baboon and the rat." *Journal of Fire Sciences* 3:228-244.
- 44 Peterson, J.E. and R.D. Stewart. 1975. "Predicting the carboxyhemoglobin levels resulting from carbon monoxide exposures." *J. Appl. Physiol.* 39:633-638.
- 45 Kimmerle, G. 1974. "Aspects and methodology for the evaluation of toxicological parameters during fire exposure." *Combust. Toxicol.* 1:4-51.
- 46 R.D. Stewart. 1974. "The effects of carbon monoxide on man." *JFF/ Combustion Toxicology* 1:167.
- 47 AEGl - Subcommittee on Acute Exposure Guideline Levels Committee on Toxicology. 2002. "Acute exposure guideline levels for selected airborne chemicals, Volume 2: Appendix 5. Hydrogen cyanide acute exposure guideline levels." National Research Council of the National Academies. National Academies Press, Washington, DC. <http://www.epa.gov/oppt/aegl/pubs/tsd6.pdf>
- 48 Hartung, R. 1994. *Cyanides and nitriles*. In *Patty's Industrial Hygiene and Toxicology, 4th ed., vol. II, Part D*. John Wiley & Sons, pp. 3119-3172.
- 49 NRC (National Research Council). 2000. *Hydrogen cyanide. In Spacecraft Maximum Allowable Concentrations for Selected Airborne Contaminants, Volume 4*. Washington, DC; National Academy Press, pp. 330-365.

- 50 El Ghawabi, S.H., M.A. Gaafar, A.A. El-Saharti, S.H. Ahmed, K.K. Malash and R. Fares. 1975. "Chronic cyanide exposure: A clinical, radioisotope, and laboratory study." *Brit. J. Ind. Med.* 32:215-219.
- 51 Blanc, P., M. Hogan and M. Mallin. 1985. "Cyanide intoxication among silver-reclaiming workers." *J. Amer. Med. Assoc.* 253:367-371.
- 52 Parmenter, D.C. 1926. "Observations on mild cyanide poisoning: Report of a case." *J. Indust. Hyg.* 8:280-282.
- 53 Barcroft, J. 1931. "The toxicity of atmospheres containing hydrocyanic acid gas." *J. Hyg.* 31:1-34.
- 54 Purser, D.A. 1984. "A bioassay model for testing the incapacitating effects of exposure to combustion product atmospheres using cynomolgus monkeys." *Journal of Fire Sciences* 2:20-36.
- 55 Bhattacharya, R., P. Kumar, and A.S. Sachan. 1994. "Cyanide induced changes in dynamic pulmonary mechanics in rats." *Indian J. Pharmacol.* 38:281-284.
- 56 O'Flaherty, E.J. and W.C. Thomas. 1982. "The cardiotoxicity of hydrogen cyanide as a component of polymer pyrolysis smokes." *Toxicol. Appl. Pharmacol.* 63:373-381.
- 57 Matijak-Schaper, M., and Y. Alarie. 1982. "Toxicity of carbon monoxide, hydrogen cyanide and low oxygen." *Combust. Technol.* 9:21-61.
- 58 Bonsall, J.L. 1984. "Survival without sequelae following exposure to 500 mg/m³ of hydrogen cyanide." *Human Toxicol.* 3:57-60.
- 59 Dudley, H.C., T.R. Sweeney, and J.W. Miller. 1942. "Toxicology of acrylonitrile (vinyl cyanide). II. Studies of effects of daily inhalation." *J. Ind. Hyg. Toxicol.* 24:255-258.
- 60 Purser, D.A., P. Grimshaw, and K.R. Berrill. 1984. "Intoxication by cyanide in fires: A study in monkeys using polyacrylonitrile." *Arch. Environ. Health* 39:394-400.
- 61 Sakurai, T. 1989. "Toxic gas tests with several pure and mixed gases using mice." *J. Fire Sci.* 7:22-77.
- 62 Flury F. and F. Zernik. 1931. *Schädliche gase dämpfe, nebel, rauch und staubarten*. Berlin, Germany: Verlag von Julius Springer, p. 337 (in German).
- 63 AEGl - Subcommittee on Acute Exposure Guideline Levels Committee on Toxicology. 2004. "Acute exposure guideline levels for selected airborne chemicals, Volume 4: Appendix 2. Hydrogen chloride acute exposure guideline levels." National Research Council of the National Academies. National Academies Press, Washington, DC. <http://www.epa.gov/oppt/aegl/pubs/tsd52.pdf>
- 64 Elkins, H.B. 1959. *The Chemistry of Industrial Toxicology, 2nd Ed.* New York, NY: John Wiley & Sons. Pp. 79-80.
- 65 Malek, D.E., and Y. Alarie. 1989. "Ergometer within a whole-body plethysmograph to evaluate performance of guinea pigs under toxic atmospheres." *Toxicol. Appl. Pharmacol.* 101:340-355.
- 66 Henderson, Y. and H. W. Hagar. 1943. *Noxious Gases*. New York: Reinhold Publishing Corp. Pp. 126.
- 67 Kaplan, H.L., A. Anzeuto, W.G. Switzer, and R.K. Hinderer. 1988. "Effects of hydrogen chloride on respiratory response and pulmonary function of the baboon." *J. Toxicol. Environ. Health* 23:473-493.
- 68 Kaplan, H.L., W.G. Switzer, R.K. Hinderer, and A. Anzeuto. 1993. "Studies of the effects of hydrogen chloride and polyvinyl chloride (PVC) smoke in rodents." *J. Fire Sci.* 11:512-552.
- 69 Barrow, C.S., H. Lucia, and Y.C. Alarie. 1979. "A comparison of the acute inhalation toxicity of hydrogen chloride versus the thermal decomposition products of polyvinylchloride." *J. Combust. Toxicol.* 6:3-12.
- 70 "Documentation of the threshold limit values for substances in workroom air," *American Conference of Governmental Industrial Hygienists*, Cincinnati (1980).
- 71 Kane, L.E., C.S. Barrow, and Y. Alarie. 1979. "A short-term test to predict acceptable levels of exposure to airborne sensory irritants." *Am. Ind. Hyg. Assoc. J.*, 40: 207-229.
- 72 AEGl - Subcommittee on Acute Exposure Guideline Levels Committee on Toxicology. 2004. "Acute exposure guideline levels for selected airborne chemicals, Volume 4: Appendix 3. Hydrogen fluoride acute exposure guideline levels." National Research Council of the National Academies. National Academies Press, Washington, DC. <http://www.epa.gov/oppt/aegl/pubs/tsd53.pdf>
- 73 Largent, E.J. 1961. *Fluorosis: The Health Aspects of Fluorine Compounds*. Columbus, OH: Ohio State University Press. Pp. 34-39, 43-48.
- 74 Largent, E.J. 1960. "The metabolism of fluorides in man." *AMA Arch. Ind. Health* 21:318-323.
- 75 Lund, K., J. Ekstrand, J. Boe, P. Sostrand, and J. Kongerud. 1997. "Exposure to hydrogen fluoride: An experimental study in humans of concentrations of fluoride in plasma, symptoms, and lung function." *Occup. Environ. Med.* 54:32-37.

- 76 Machle, W., F. Thamann, K. Kitzmiller, and J. Cholak. 1934. "The effects of the inhalation of hydrogen fluoride. I. The response following exposure to high concentrations." *J. Ind. Hyg.* 16:129-145.
- 77 Rosenholtz, M.J., T.R. Carson, M.H. Weeks, F. Wilinski, D.F. Ford, and F.W. Oberst. 1963. "A toxicopathologic study in animals after brief single exposures to hydrogen fluoride." *Amer. Ind. Hyg. Assoc. J.* 24:253-261.
- 78 Dalbey, W., B. Dunn, R. Bannister, W. Daughtrey, C. Kirwin, F. Reitman, M. Wells, and J. Bruce. 1998. "Short-term exposures of rats to airborne hydrogen fluoride." *Toxicol. Environ. Health* 55(Part A):241-275.
- 79 Dalbey, W. 1996. *Evaluation of the Toxicity of Hydrogen Fluoride at Short Exposure Times*. Petroleum Environmental Research Forum Project 92-09, performed at Stonybrook Laboratories Inc., Pennington, NJ.
- 80 Stavert, D.M., D.C. Archuleta, M.J. Behr, and B.E. Lehnert. 1991. "Relative acute toxicities of hydrogen fluoride, hydrogen chloride, and hydrogen bromide in nose- and pseudo-mouth-breathing rats." *Fundam. Appl. Toxicol.* 16:636-655.
- 81 Kusewitt, D.F., D.M. Stavert, G. Ripple, T. Mundie, and B.E. Lehnert. 1989. "Relative acute toxicities in the respiratory tract of inhaled hydrogen fluoride, hydrogen bromide, and hydrogen chloride." *Toxicologist* 9:36.
- 82 MacEwen, J.D., and E.H. Vernot. 1970. *Toxic Hazards Research Unit Annual Technical Report: 1970*. AMRL-TR-70-77, AD 714694. Aerospace Medical Research Laboratory, Wright-Patterson AFB, Ohio.
- 83 A EGL. 2005. "Proposed acute exposure guideline levels A EGLs for hydrogen bromide / hydrogen iodide." For NAS/COT Subcommittee for A EGLs.
- 84 Connecticut State Department of Health. 1955. Unpublished data. Occupational Health Section, Connecticut State Department of Health, Hartford, CT.
- 85 A EGL. 2005. "Interim acute exposure guideline levels: Acrolein." For NAS/COT Subcommittee for A EGLs. <http://www.epa.gov/oppt/aegl/pubs/tsd303.pdf>
- 86 Weber-Tschopp, A., Fischer, T., Gierer, R., et al. 1977. "Experimentally induced irritating effects of acrolein on men." *Int. Arch. Occup. Environ. Health* 40:117-130.
- 87 Sim, V.M. and Pattle, R.E. 1957. "Effect of possible smog irritants on human subjects." *JAMA* 165:1908-1913.
- 88 Hales, C. A., Barkin, P. W., Jung, W., et al. 1988. "Synthetic smoke with acrolein but not HCl produces pulmonary edema." *J. Appl. Physiol.* 64:1121-1133.
- 89 Darley, E.F., Middleton, J.T., Garber, M.J. 1960. "Plant damage and eye irritation from ozone hydrocarbon reactions." *J. Agric. Food. Chem.* 8:483-485.
- 90 A EGL. 2007. "Interim acute exposure guideline levels for formaldehyde." For NAS/COT Subcommittee for A EGLs. http://www.epa.gov/oppt/aegl/pubs/formaldehydeinterimornl_oct2007c.pdf
- 91 Ritchie, I.M. and R.G. Lehnen. 1987. "Formaldehyde-related health complaints of residents living in mobile and conventional homes." *Am. J. Public Health* 77:323-328.
- 92 Bender, J.R., L.S. Mullin, G.J. Grapel, and W.E. Wilson. 1983. "Eye irritation response of humans to formaldehyde." *Am. Ind. Hyg. Assoc. J.* 44:463-465.
- 93 Paustenbach, D., Y. Alarie, T. Kulle, N. Schacter, R. Smith, J. Swenberg, H. Witchi, and S.B. Horowitz. 1997. "A recommended occupational exposure limit for formaldehyde based on irritation." *J. Toxicol. Environ. Health* 50:217-263.
- 94 Day, J.H., R.E.M. Lees, R.H. Clark, and P.L. Pattee. 1984. "Respiratory response to formaldehyde and off-gas of urea formaldehyde foam insulation." *Can. Med. Assoc. J.* 131:1061-1065.
- 95 Andersen, I. and L. Molhave. 1983. "Controlled human studies with formaldehyde." pp. 154-165 in Gibson, J.E., ed., *Formaldehyde Toxicity*. Washington, DC: Hemisphere Publishing Corporation.
- 96 Weber-Tschopp, A., T. Fischer, and E. Grandjean. 1977. "Irritating effects of formaldehyde on men." *Int. Arch. Occup. Environ. Health* 39:207-218.
- 97 Kulle, T.J. 1993. "Acute odor and irritation response in healthy nonsmokers with formaldehyde exposure." *Inhal. Toxicol.* 5:323-332.
- 98 Kulle, T.J., L.R. Sauder, J.R. Hebel, and D.J. Green. 1987. "Formaldehyde dose-response in healthy nonsmokers." *J. Air Pollut. Control Assoc.* 37:919-924.
- 99 Morgan, K.T., D.L. Patterson and E.A. Gross. 1986. "Responses of the nasal mucociliary apparatus of F-344 rats to formaldehyde gas." *Toxicol. Appl. Pharmacol.* 82:1-13.
- 100 Chang, J.C.F., E.A. Gross, J.A. Swenberg, and C.S. Barrow. 1983. "Nasal cavity deposition, histopathology, and cell proliferation after single or repeated formaldehyde exposures in B6C3F1 mice and F-344 rats." *Toxicol. Appl. Pharmacol.* 68:161-176.

- 101 Tobe, M., K. T. Kaneko, Y. Uchida, et al. 1985. *Studies of the inhalation toxicity of formaldehyde*. U.S. Environmental Protection Agency translation, TR-85-0236. Washington, DC: U.S. EPA.
- 102 Witek, T.J., E.N. Schacter, T. Tosun, G.J. Beck, and B.P. Leaderer. 1987. "An evaluation of respiratory effects following exposure to 2.0 ppm formaldehyde in asthmatics: lung function, symptoms, and airway reactivity." *Arch. Environ. Health* 42:230-237.
- 103 Schachter, E.N., T.J. Witek, T. Tosun, B.P. Leaderer, and G.J. Beck. 1986. "A study of respiratory effects from exposure to 2.0 ppm formaldehyde in healthy subjects." *Arch. Environ. Health* 41:229-239.
- 104 Witek, T.J., E.N. Schacter, T. Tosun, B.P. Leaderer, and G.J. Beck. 1986. "Controlled human studies on the pulmonary effects of indoor air pollution: Experiences with sulfur dioxide and formaldehyde." *Environ. Intl.* 12:129-135.
- 105 Schachter, E.N., T.J. Witek, T. Tosun, G.J. Beck and B.P. Leaderer. 1985. "Respiratory effects of exposure to 2.0 ppm of formaldehyde in asthmatic subjects." *Am. Rev. Respir. Dis.* 131:A170.
- 106 Green, D.J., R. Bascom, E.M. Healey, J.R. Hebel, L.R. Sauder, and T.J. Kulle. 1989. "Acute pulmonary response in healthy, nonsmoking adults to inhalation of formaldehyde and carbon." *J. Toxicol. Environ. Health* 28:261-275.
- 107 Green, D.J., L.R. Sauder, T.J. Kulle, and R. Bascom. 1987. "Acute response to 3.0 ppm formaldehyde in exercising healthy nonsmokers and asthmatics." *Am. Rev. Respir. Dis.* 135:1261-1266.
- 108 Sauder, L.R., C.D. Chatham, D.J. Green, and T.J. Kulle. 1986. "Acute pulmonary response to formaldehyde exposure in healthy nonsmokers." *J. Occup. Med.* 28:420-424.
- 109 Douglas, R.B. 1974. "Human reflex bronchoconstriction as an adjunct to conjunctival sensitivity in defining the threshold limit values of irritant gases and vapours." Thesis, TUC Centenary Institute of Occupational Health, London School of Hygiene and Tropical Medicine.
- 110 Monticello, T.M., K.T. Morgan, J.I. Everitt, and J.A. Popp. 1989. "Effects of formaldehyde gas on the respiratory tract of rhesus monkeys." *Amer. J. pathol.* 134:515-527.
- 111 Stephens, E.R., E.R. Darley, O.C. Taylor, and W.E. Scott. 1961. "Photochemical reaction products in air pollution." *Int. J. Air Water Pollut.* 4:79-100.
- 112 Barnes, E.C. and H.W. Speicher. 1942. "The determination of formaldehyde in air." *J. Ind. Hyg. Toxicol.* 24:10-17.
- 113 AEGLE. 2006. "Interim Acute Exposure Guideline Levels for Nitrogen Dioxide." For NAS/COT Subcommittee for AEGLEs. <http://www.epa.gov/oppt/aegl/pubs/tsd308.pdf>
- 114 Carson, T.R., M.S. Rosenholtz, F.T. Wilinski, and M.H. Weeks. 1962. "The responses of animals inhaling nitrogen dioxide for single, short-term exposures." *Am. Ind. Hyg. Assoc. J.* 23:457-462.
- 115 Hine, C.H., F.H. Meyers, and R.W. Wright. 1970. "Pulmonary changes in animals exposed to nitrogen dioxide, effects of acute exposures." *Toxicol. Appl. Pharmacol.* 16:201-213.
- 116 von Nieding, G., H.M. Wagner, H. Krekeler, H. Löllgen, W. Fries, and A. Beuthan. 1979. "Controlled studies of human exposure to single and combined action of NO₂, O₃, and SO₂." *Int. Arch. Occup. Environ. Health* 43:195-210.
- 117 Henry, M.C., R. Ehrlich, and W.H. Blair. 1969. "Effect of nitrogen dioxide on resistance of squirrel monkeys to *Klebsiella pneumoniae* infection." *Arch. Environ. Health* 18:580-587.
- 118 Murphy, S.D., C.E. Ulrich, S.H. Frankowitz, and C. Xintaras. 1964. "Altered function in animals inhaling low concentrations of ozone and nitrogen dioxide." *Am. Ind. Hyg. Assoc. J.* 25:246-253.
- 119 Henschler, D., A. Stier, H. Beck, and W. Neuman. 1960. "Odor threshold of a few important irritant gases (sulfur dioxide, ozone, nitrogen dioxide) and observations in humans exposed to low concentrations." *Archiv für Gewerbepathologie und Gewerbehygiene* 17:547-570. (translated from German).
- 120 Devlin, R., D. Horstman, S. Becker, T. Gerrity, M. Madden, and H. Koren. 1992. "Inflammatory response in humans exposed to 2.0 ppm NO₂." *Am. Rev. Respir. Dis.* 145:A456.
- 121 Rasmussen, T.R., S.K. Kjaergaard, U. Tarp, and O.F. Pedersen. 1992. "Delayed effects of NO₂ exposure on alveolar permeability and glutathione peroxidase in healthy humans." *Am. Rev. Respir. Dis.* 146:654-659.
- 122 Goings, S.A.J., T.J. Kulle, R. Bascom, L.R. Sauder, D.J. Green, J.R. Hebel, M.L. Clements. 1989. "Effect of nitrogen dioxide exposure on susceptibility to influenza A virus infection in healthy adults." *Am. Rev. Respir. Dis.* 139:1075-1081.

- 123 Hackney, J.D., F.C. Thiede, W.S. Linn, E.E. Pedersen, C.E. Spier, D.C. Law, and D.A. Fischer. 1978. "Experimental studies on human health effects of air pollutants. IV. Short-term physiological and clinical effects of nitrogen dioxide exposure." *Arch. Environ. Health* 33:176-181.
- 124 Linn, W.S. and J.D. Hackney. 1983. Short-term human respiratory effects of nitrogen dioxide: Determination of quantitative dose-response profiles. Phase 1. Exposure of healthy volunteers to 4 ppm NO₂. NTIS order no. PB84-132299. 30 pp.
- 125 Stavert, D.M. and B.E. Lehnert. 1990. "Nitric oxide and nitrogen dioxide as inducers of acute pulmonary injury when inhaled at relatively high concentrations for brief periods." *Inhal. Toxicol.* 2:53-67.
- 126 Blockley, W.V. 1973. *Biology Data Book*, Federation of American Societies for Experimental Biology, Bethesda, MD.
- 127 Babrauskas, V., 1979. *Full-Scale Burning Behavior of Upholstered Chairs (Technical Note 1103)*, National Bureau of Standards, Washington, DC.
- 128 Mudan, K.S. and P.A. Croce. 1995. "Fire hazard calculations for large open hydrocarbon fires," in *The SFPE Handbook of Fire Protection Engineering, 2nd edition*. National Fire Protection Association, Quincy, MA.
- 129 D.L. Simms and P.L. Hinkley. 1963. *Fire Research Special Report No. 3*, Her Majesty's Stationary Office, London, UK.
- 130 Canadian Centre for Occupational Health and Safety (CCOHS).
http://www.ccohs.ca/oshanswers/phys_agents/heat_health.html.
- 131 Stoll, A.M. and L.C. Greene. 1959. "Relationship between pain and tissue damage due to thermal radiation," *Journal of Applied Physiology*, 14:373-383.
- 132 Buettner, K. 1951. "Effects of extreme heat and cold on human skin. II. Surface temperature, pain and heat conductivity in experiments with radiant heat." *J. Appl. Physiol.*, 3: 703-713.
- 133 Veghte, J.H. 1982. "Designing protective clothing, Part 2." *F. Service Today*, 49:16-18.
- 134 Veghte, J.H. 1973. "Human exposure to high radiant environments." *Aerospace Medicine*, October:1147-1151.
- 135 Stoll, A.M. and M.A. Chianta. 1971. "Heat transfer through fabrics as related to thermal injury." *Transactions of the New York Academy of Sciences* 33:649-670.
- 136 Stoll, A.M. and M.A. Chianta. 1969. "Method of rating system for evaluation of thermal protection." *Aerospace Medicine* November: 1232-1237.
- 137 Jin, T. 1997. "Studies on human behavior and tenability in fire smoke." *Fire Safety Science – Proceedings of the Fifth International Symposium*, Hasemi, Y., ed. London, England: Interscience Communications Ltd: 3-21.
- 138 Jin, T. 1976. "Visibility through fire smoke (Part 5: Allowable smoke density for escape from fire)," Report of Fire Research Institute of Japan, 12-18.
- 139 Jin, T. 1979. "Human behavior in fire smoke – Experiments on emotional instability under increasing smoke density – Preliminary report." In U.S./Japan Government Cooperative Program on Natural Resources (UJNR). Fire Research and Safety. 4th Joint Panel Meeting. Tokyo, Japan, 323-339 pp.
- 140 Kawagoe, K. and F. Saito. 1967. "Measures to deal with smoke problems caused by fire." *Journal of Japanese Society for Safety Engineering*, 6:108-114.
- 141 Rasbash, D.J. 1975. "Sensitivity criteria for detectors used to protect life." 7th *International Seminar on Automatic Fire Detection*, Aachen, Germany.
- 142 Shern, J.H. 1966. "Smoke contribution factor in fire hazards classification of building materials." *Sixty-ninth Annual Meeting of the American Society for Testing and Materials*, American Society for Testing and Materials: Philadelphia, PA:93-105.
- 143 Rasbash, D.J. 1966. "Smoke and toxic gas." *Fire*, 59:175-179.
- 144 Kingman, F.E.T. 1953. "The products of combustion in burning buildings." *Journal of Applied Chem*, 3: 463-468.