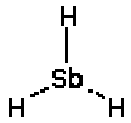
ACUTE EXPOSURE GUIDELINE LEVELS (AEGLs)
FOR
STIBINE (ANTIMONY HYDRIDE)
7803-52-3

SbH_3



INTERIM

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PREFACE

Under the authority of the Federal Advisory Committee Act (FACA) P. L. 92-463 of 1972, the National Advisory Committee for Acute Exposure Guideline Levels for Hazardous Substances (NAC/AEGL Committee) has been established to identify, review and interpret relevant toxicologic and other scientific data and develop AEGLs for high priority, acutely toxic chemicals.

AEGLs represent threshold exposure limits for the general public and are applicable to emergency exposure periods ranging from 10 minutes to 8 hours. Three levels — AEGL-1, AEGL-2 and AEGL-3 — are developed for each of five exposure periods (10 and 30 minutes, 1 hour, 4 hours, and 8 hours) and are distinguished by varying degrees of severity of toxic effects. The three AEGLs are defined as follows:

AEGL-1 is the airborne concentration (expressed as parts per million or milligrams per cubic meter [ppm or mg/m3]) of a substance above which it is predicted that the general population, including susceptible individuals, could experience notable discomfort, irritation, or certain asymptomatic, non-sensory effects. However, the effects are not disabling and are transient and reversible upon cessation of exposure.

AEGL-2 is the airborne concentration (expressed as ppm or mg/m3) of a substance above which it is predicted that the general population, including susceptible individuals, could experience irreversible or other serious, long-lasting adverse health effects or an impaired ability to escape.

AEGL-3 is the airborne concentration (expressed as ppm or mg/m3) of a substance above which it is predicted that the general population, including susceptible individuals, could experience life-threatening health effects or death.

Airborne concentrations below the AEGL-1 represent exposure levels that could produce mild and progressively increasing but transient and nondisabling odor, taste, and sensory irritation or certain asymptomatic, non-sensory effects. With increasing airborne concentrations above each AEGL, there is a progressive increase in the likelihood of occurrence and the severity of effects described for each corresponding AEGL. Although the AEGL values represent threshold levels for the general public, including susceptible subpopulations, such as infants, children, the elderly, persons with asthma, and those with other illnesses, it is recognized that individuals, subject to unique or idiosyncratic responses, could experience the effects described at concentrations below the corresponding AEGL

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EXECUTIVE SUMMARY

Stibine (SbH_3) is a colorless gas with a disagreeable odor produced when antimony-containing lead batteries are charged. Stibine is also produced when strong reducing agents react with acid solutions of antimony compounds. Nascent hydrogen is required for its production.

Stibine is an eye and respiratory irritant and can cause death from pulmonary congestion and edema in laboratory animals. Stibine is also a hemolytic poison. The relatively brief time to death following acute exposure is consistent with death as a consequence of pulmonary edema rather than death from renal failure subsequent to hemolysis. No human case reports are available, but stibine air samples from factories where stibine is produced provide data regarding human exposure. Eye and respiratory irritation complaints prompted one report, but these workers were also exposed to arsine and sulfuric acid in addition to stibine.

Exposure-response data from rat and guinea pig studies were used to derive acute exposure guideline level (AEGL) values for stibine due to lack of quantitative human data. The AEGL values for the periods of concern are scaled from the experimental duration using exponential scaling ($C^n \times t = k$, where C= exposure concentration, t= exposure duration, and k= a constant). Data are unavailable to empirically derive a scaling factor (n) for stibine. The concentration-exposure time relationship for many irritant and systemically acting vapors and gases may be described by $C^n \times t = k$, where the exponent n ranges from 0.8 to 3.5 (ten Berge et al. 1986). Temporal scaling was performed using n = 3, when extrapolating to shorter time points and n = 1 when extrapolating to longer time points for AEGL values (NRC 2001).

AEGL-1 values are not recommended because data are not available from human or animal studies consistent with AEGL-1 endpoints.

AEGL-2 values are based upon the highest experimental concentration (29.1 ppm) without an AEGL-2 effect. Rats and guinea pigs exposed for 30 minutes to 29.1 ppm stibine suffered no "untoward effects" (Price et al. 1979). At the next highest exposure, 191 ppm, eye and respiratory irritation were present, and the animals experienced initial hyperactivity followed by reduced physical activity. Renal tubular dilation and pulmonary edema were also noted at this concentration. A total uncertainty factor of 10 was applied to account for interspecies extrapolation and intraspecies variability. A factor of 3 was applied for interspecies variability. Price et al. (1979) reported similar mortalities from pulmonary edema for rats (70%) and guinea pigs (70%) at 333 ppm and similar lesions (renal tubular dilation, pulmonary edema) were reported in both species at 191 ppm. An uncertainty factor of 3 was applied for intraspecies variability. Although the mechanism of toxicity is unknown, the point of departure is based on contact irritation in the lung, and that respiratory irritant action is not expected to vary a great deal among individuals. The intraspecies factor of 3 is supported by the steep-concentration response shown by Price et al. (1979) in two species. No lethality was observed when both species were exposed to 191 ppm for 30 min, but a 1.7-fold increase in concentration (333 ppm) for the same duration resulted in 70 and 70% lethality in rats and guinea pigs, respectively.

The highest concentration with no treatment-related mortality (191 ppm, 30-min exposure in rats) was identified as the basis of the AEGL-3 derivation (Price et al. 1979). One guinea pig died exposed to 191 ppm for 30 min died and cortical necrosis of the cerebrum was found in that animal. The lesion and death were not considered by Price et al. (1979) to be treatment-related.

At the next highest concentration, 333 ppm, 7/10 rats and 7/10 guinea pigs died from fatal pulmonary edema. The identical uncertainty factors and rationale used for AEGL-2 values were applied to AEGL-3 calculations.

The calculated values are listed in Table 1.

TABLE 1. Summary of AEGL Values for Stibine

Classification	10-minute	30-minute	1-hour	4-hour	8-hour	Endpoint (Reference)
AEGL−1 (Notable discomfort)	NR	NR	NR	NR	NR	
AEGL−2 (Disabling)	4.2 ppm (21 mg/m^3)	2.9 ppm (15 mg/m^3)	1.5 ppm (7.7 mg/m^3)	0.36 ppm (1.8 mg/m^3)	0.18 ppm (0.92 mg/m^3)	No effect level for irreversible toxicity (Price et al. 1979)
AEGL−3 (Lethal)	28 ppm (140 mg/m^3)	19 ppm (97 mg/m^3)	9.6 ppm (49 mg/m^3)	2.4 ppm (12 mg/m^3)	1.2 ppm (6.1 mg/m^3)	Highest exposure with no mortality (191 ppm for 30 min) in rats and guinea pigs (Price et al. 1979)

NR-Not recommended. Numeric values for AEGL-1 values were derived due to insufficient human data and the limited animal data was used to derive AEGL-2 values. Absence of an AEGL-1 value does not imply that exposure below the AEGL-2 concentration is without adverse effects.

1. INTRODUCTION

Stibine is a thermally unstable, volatile colorless gas produced either when an acid reacts with antimony or is produced from antimony when lead-acid batteries are charged and overcharged. It can be created by dissolving zinc-antimony or magnesium-antimony in diluted hydrochloric acid. It is also produced during the formation of lead plates. Stibine is known to be generated in situ and used in infrared devices and solid-state lasers (Clark 2006). It has also been used as a fumigating agent and in the production of semiconductors. Chemical and physical properties for stibine are listed in Table 2.

TABLE 2. Chemical and Physical Properties

Parameter	Value	References
Synonyms	Antimony hydride, antimony trihydride, antimonwasswerstoffes, hydrogen antimonide, antymonowodor	O'Neil et al. 2001, RTECS 2006
Chemical formula	SbH_3	O'Neil et al. 2001
Molecular weight	124.78	O'Neil et al. 2001
CAS Reg. No.	7803-52-3	O'Neil et al. 2001
Physical state	Gas	O'Neil et al. 2001
Solubility in water	4.1 at 0ºC	HSBD 2006
Vapor pressure	> 1 atm at 20ºC	ACGIH 2001
Vapor density (air =1)	2.204 g/ml at boiling point	O'Neil et al. 2001
Liquid density (water =1)	2.26 at -25ºC	IPCS 2001
Melting point	-88ºC	O'Neil et al. 2001
Boiling point	-18.4ºC	O'Neil et al. 2001
Flammability limits	4	HSBD 2006
Conversion factors	1 ppm = 5.11 mg/m^3 1 mg/m^3 = 0.196 ppm	ACGIH 2001

2. HUMAN TOXICITY DATA

2.1. Acute Lethality

No reports of mortality from stibine exposure were found.

2.2. Nonlethal Toxicity

Human exposure to stibine is usually occupation-related. Several epidemiological studies included measured stibine concentrations in the air of battery manufacturing plants, particularly in the manufacturing areas where stibine was produced.

2.2.1. Odor Threshold/Odor Awareness

Stibine has a hydrogen sulfide-like odor, but no odor threshold data were located.

2.2.2. Epidemiologic Studies

A survey of a battery manufacturing plant by Young (1979) determined concentrations in the assembly area of 0.007 mg/m^3 (0.0014 ppm) stibine (SbH_3). Samplers consisted of charcoal tubes attached to sampling pumps, and area samples were collected for three hours.

Hartle (1980) collected six air samples in the battery shop of a locomotive and electric passenger car maintenance and repair shop during the work day. Stibine area concentrations ranged from 0.0002-0.0013 ppm (0.001-0.0066 mg/m^3). No health problems were recorded in the report, however, it was noted that employees indicated the potential for increased concentrations during the night shift (during times of minimal ventilation) could affect the morning shift workers. No information was given as to how the workers could be affected.

Costello (1980) measured stibine at a battery production plant. Breathing zone air samples were taken from immersion fillers, process attendants in the forming area, power spin operators, and acid levelers. Two different sampler pumps were used along with filters and charcoal tubes that collected personal samples for seven to eight hours. Concentrations of stibine (SbH_3) ranged from 0.05 mg/m^3 (0.01 ppm) to 0.3 mg/m^3 (0.06 ppm). No medical complaints associated with stibine exposure were mentioned in the report or investigated in these workers, but potential health effects of exposure to stibine and arsine, including hemolysis, were listed in the report.

Lucas and Cone (1982) evaluated stibine levels at a diesel engine and railroad locomotive plant following complaints of sore throat, respiratory irritation, and eye irritation from workers in the battery charging area. Area samplers consisted of charcoal tubes on portable pumps. The air samples, including those from the battery charging area and breathing zone samples, revealed concentrations up to 0.0023 ppm (0.012 mg/m^3). The authors acknowledged that equipment malfunction delayed evaluation of stibine air samples and that actual concentrations of stibine may have been higher than those reported. Workers within 100 feet of the battery charging area were at a greater risk of suffering from throat, respiratory, and eye irritation than workers farther away from the area. These workers, however, were also exposed to arsine and sulfuric acid.

Jones and Gamble (1984) sampled the workplace area air in five different lead acid storage battery manufacturing plants using charcoal tubes and low flow pumps. The air samplers were placed at different areas within each plant including case formation, element formation, and plate formation. Each sampler ran from four to five hours. The highest level detected was 2.5 mg/m^3 (0.5 ppm) in an element formation area. Breathing zone sampling for stibine was not conducted. No information on health effects was included in the report.

Singal et al. (1985) measured stibine (SbH_3) in the breathing zone of workers. Samplers consisted of sodium carbonate-impregnated filters and a charcoal tube attached to a portable pump and sample time ranged from five to eight hours. Concentrations of stibine ranged from less than 0.04 ug/m^3 (0.008 ppm) to 0.54 ug/m^3 (0.11 ppm). Stibine was most frequently detected in the breathing zone samples of the post-burn and battery forming workers; however, there were no stibine-related medical complaints associated with these levels in the workers.

2.3. Neurotoxicity

Hussain et al. (1998) studied stibine in relation to cholinesterase and acetylcholinesterase activity. The activity of human plasma, human red blood cells, and mouse neuronal cells was measured in the presence of stibine. Stibine failed to inhibit cholinesterase or acetyl-cholinesterase activity.

2.4. Developmental/Reproductive Toxicity

There are no studies on potential developmental or reproductive effects of stibine.

2.5. Genotoxicity

Genotoxicity data relevant to the derivation of AEGLs for stibine were not available.

2.6. Carcinogenicity

There are no reports on the potential carcinogenicity of stibine.

2.7. Summary

Area samples in battery operations reveal concentrations to which humans may be exposed during 3 to 8 hour work shifts ranged from 0.0013 to 0.5 ppm. Respiratory irritation was the main complaint of workers exposed to stibine. However, these workers were also exposed to arsine and sulfuric acid which also initiate irritation.

3. ANIMAL TOXICITY DATA

3.1. Acute Lethality

Several inhalation studies examined lethality of stibine in different species. Lethal concentrations for various species are listed in Table 3.

3.1.1. Dogs

Webster (1946) reported that death occurred within a few hours or was delayed up to a day in dogs exposed for 1 hour to 40-45 ppm. Hemoconcentration, pulmonary congestion and edema were cited as the cause of death. No additional details were given in the Webster (1946) report.

3.1.2. Rats

Price et al. (1979) exposed groups of five male and five female Sprague-Dawley rats to stibine concentrations of 29.1, 191, or 333 ppm stibine for 30 minutes on one day and observed the animals for 14 days. The stainless steel and glass inhalation chamber allowed whole-body animal exposure. Analytical concentrations were measured by spectrophotometry. At 333 ppm, 5/5 males and 2/5 females died and six of these animals died within four hours post-exposure. The rats displayed fatal pulmonary edema and intravascular congestion. Pulmonary fibrosis was also noted. There were no deaths in rats in the exposure groups.

3.1.3. Guinea Pigs

Dunn and Webster (1944) exposed 97 guinea pigs to stibine-air mixtures at concentrations from 44 to 293 ppm for one hour and then observed them. Eleven of 76 guinea pigs exposed to greater than 66 ppm died the first day and 40 died between days two and six. Twenty-three of the 40 guinea pigs dying between days two and six were found to have hemoglobin in the renal tubules in the form of crystals and casts formed as a result of chemical hemolysis. Hemoglobinuria was noted in the animals exposed to 133 ppm or greater concentrations. The remaining guinea pigs survived from seven days to almost three years. Very few details on exact concentrations administered and experimental design were available.

Webster (1946) noted that the lethal concentrations of stibine in guinea pigs were three- or four-fold higher than those required in cats and dogs.

Price et al. (1979) exposed groups of five male and five female Hartley guinea pigs to 29.1, 191, or 333 ppm stibine for 30 minutes. The stainless steel and glass inhalation chamber allowed whole-body animal exposure. Analytical concentrations were measured by spectrophotometry. One female guinea pig that inhaled 191 ppm died one day post-exposure. The animal had cortical necrosis of the cerebrum, but this was not considered compound-related. Four of five males and 3/5 females from the 333 ppm group died one to nine hours post-exposure. During exposure, the animals experienced generalized depressed activity, shallow, rapid breathing, and mild tremors 15 minutes into the exposure and labored breathing after 30 minutes of exposure. The depressed activity persisted for 4 hour after exposure ceased. Examination revealed pulmonary scarring, congestion, and edema in those animals.

3.1.3. Mice

Haring and Compton (1935) used overcharged three-cell batteries to expose one mouse each to varying (30, 43, 45, and 50 ppm) concentrations of stibine. An adapted iodo-starch colorimetric scheme was used to calculate the chamber air concentration within 5%. Collected stibine gas was passed into a cylindrical glass chamber into which a mouse had been placed. The lowest concentration caused death after 7 hours and 40 minutes. At 43 ppm, death occurred 5 hours and 15 minutes into the exposure. The mouse exposed to 45 ppm showed signs of distress after 15 minutes of exposure and died after 4 hours and 16 minutes of exposure. The mouse exposed to 50 ppm died after 3 hours and 55 minutes of exposure.

3.1.4 Cats

Webster (1946) reported 40-45 ppm as being dangerous to cats exposed for an hour. It was reported that death was rapid. No additional details were reported.

3.2. Nonlethal Toxicity

There are reports of nonlethal toxicity in laboratory animals exposed to stibine. Nonlethal inhalation concentrations are listed in Table 3.

3.2.1. Rats

Price et al. (1979) used male and female Hartley guinea pigs and Sprague-Dawley rats to investigate species variability of acute exposure to stibine. Rats were exposed for 30 minutes to 29.1, or 191 ppm and observed for 14 days. Effects found at the lowest dose level included increased relative liver and adrenal gland weights in male rats. Rats exposed to the higher concentration displayed initial hyperactivity followed by depressed activity during exposure. Rats developed eye irritation after 15 minutes of exposure and closure after 25 minutes at 191 ppm. These animals were judged to not have an impaired ability to escape by the authors. Renal tubular dilation and calcific debris in the renal pelvis were found in two rats. Exposed male rats were found to have increased relative heart weight and adrenal glands in the female rats weighed more than those of the control group.

3.2.2. Guinea Pigs

Dunn and Webster (1944) exposed 97 guinea pigs to a single one hour exposure to stibine-air mixture. Concentrations ranged from 44 to 293 ppm. Death rarely occurred at 44 ppm, but was frequent at 90 ppm and higher concentrations. Hemoglobinuria was present in animals exposed to 66 ppm or higher concentration. These animals also displayed hemoglobin casts and crystals in the renal tubules caused by hemolysis.

Webster (1946) noted that guinea pigs exposed to 65 ppm stibine for one hour displayed changes in erythrocyte morphology including the formation of red corpuscles with tiny spicules extending from the periphery. The morphological changes were irreversible and were followed by hemoglobinuria and anemia. These animals were less susceptible than dogs and cats and rarely died before 24 hours unless exposed to higher concentrations.

Price et al. (1979) used guinea pigs and rats to determine variability between species response to stibine. Ten guinea pigs (5/sex) were exposed to 29.1 or 191 ppm for 30 minutes and observed for 14 days. Granuloma was observed two animals at 29.1 ppm, but the authors noted that the lesions were probably not treatment-related. During exposure, guinea pigs exposed to 191 ppm experienced generalized depressed activity after 25 minutes. Renal tubular dilation, calcific debris in the renal pelvis, and pulmonary inflammation were observed at 191 ppm. Female guinea pigs exposed to 29.1 ppm group ovaries which weighed significantly more than those of the control group. In the 191 ppm group, male guinea pigs weighed less than males in the control group and had livers that weighed more than those of the control group. Females that inhaled 191 ppm developed increased relative renal weights.

TABLE 3. Inhalation Data for Laboratory Animals Exposed to Stibine

Species	Concentration (ppm)	Duration (min)	Effect	Reference
Cat	40-45	60	Pulmonary congestion and edema, death within few hours up to one day	Webster 1946
Dog	40-45	60	Pulmonary congestion and edema, death within few hours up to one day	Webster 1946
Rat	44-293	60	≥ 66 ppm-67% mortality; ≥ 133 ppm-hemoglobinuria	Dunn and Webster 1944
Rat	29.1	30	No untoward effects	Price et al. 1979
Rat	191	30	Eye irritation and closure, generalized depressed activity 15 min into exposure, renal tubular dilation	Price et al. 1979
Rat	333	30	Generalized depressed activity, dyspnea, pulmonary congestion and edema, 70% mortality (100% male, 40% female)	Price et al. 1979
Guinea pig	65	30	Irreversible erythrocyte morphology changes, hemoglobinuria, anemia	Webster 1946
Guinea Pig	29.1	30	No untoward effects	Price et al. 1979
Guinea Pig	191	30	Generalized depressed activity 25 min into exposure, renal tubular dilation, pulmonary inflammation	Price et al. 1979
Guinea Pig	333	30	Generalized depressed activity, tremors, pulmonary congestion and edema, 70% mortality (80% male, 60% female)	Price et al. 1979

3.3. Developmental/Reproductive Toxicity

There are no reports on the potential developmental or reproductive toxicity of stibine.

3.4. Genotoxicity

Andrewes et al. (2004) used pBR322 plasmid DNA to determine if stibine, produced from a reaction of sodium borohydride and potassium antimony tartrate, could damage DNA. The DNA was either suspended above a reaction producing stibine or within the reaction itself for 30 minutes. They found that DNA suspended above the reaction was damaged, but the DNA placed in the reaction was minimally damaged. The authors did not measure the concentration of stibine during the reaction, but estimated (through an unstated method) that 6000 mg/m^3 (1176 ppm) was the minimum amount of stibine gas required to damage naked DNA *in vitro*.

3.5. Carcinogenicity

There are no reports on the potential carcinogenicity of stibine.

3.6. Summary

The consequences of acute inhalation exposure to stibine are described in the above section and presented in Table 3. Respiratory irritation, depressed activity, renal changes, and death were caused by acute irritation resulting in pulmonary congestion and edema.

4. SPECIAL CONSIDERATIONS

4.1. Metabolism and Disposition

Kentner et al. (1995) found that exposure to stibine gas could be detected by measuring the antimony concentration in the blood and urine of humans. However, there is no way to distinguish between stibine exposure and exposure to other antimony compounds. They also reported complete elimination of antimony following a weekend of no workplace stibine exposure (not detected in urine samples).

4.2. Mechanism of Toxicity

Stibine is a respiratory irritant that induces pulmonary inflammation, edema, and congestion. Stibine is recognized as a hemolytic poison that causes the rapid breakdown of erythrocytes. Webster (1946) noted changes in guinea pig erythrocyte morphology that occurred within minutes after initial exposure. The presence of "spine cells", erythrocytes resembling crenated cells having spicules extending symmetrically around the periphery of the spherical cells and "Thornapple cells", similar to cells produced with hyper- and hypotonic salt solutions, were also present. The hemolytic phase immediately followed the morphologic change. The mechanism by which these changes occur is not understood.

4.3. Structure Activity Relationships

Stibine regulations are often based on analogy with arsine which has a similar acute mortality in rats.

4.4. Other Relevant Information

4.4.1. Species Variability

Webster (1946) reported stibine concentrations of 40-45 ppm as being "dangerous" to dogs and cats after a 1-hour exposure, and that guinea pigs could tolerate concentrations three to four times higher than dogs and cats. However, no experimental details were given. There were no significant differences in stibine-induced toxicity among Sprague-Dawley rats and Hartley guinea pigs exposed to 29.1, 191, or 333 ppm stibine for 30 minutes (Price et al. 1979). Effects were similar between the species and included depressed physical activity, pulmonary inflammation, pulmonary edema, and congestion.

4.4.2. Unique Physiochemical Properties

Nascent hydrogen is required for the formation of stibine.

4.4.3. Concurrent Exposure

Exposure to stibine occurs in occupational settings that often involve concurrent exposures to other metal vapors and solvents including arsine and sulfuric acid. It is assumed that concurrent exposure with other chemicals, especially arsine, could increase the severity of the effects of an exposure to stibine.

5. DATA ANALYSIS FOR AEGL-1

5.1. Summary of Human Data Relevant to AEGL-1

No human data are available for the derivation of AEGL-1 values for stibine.

5.2. Summary of Animal Data Relevant to AEGL-1

Data on laboratory animals consistent with the AEGL-1 definition were limited. No untoward effects were found in rats and guinea pigs exposed to 29.1 ppm (Price et al. 1979). Lesions observed in the animals at this concentration were not treatment-related.

5.3. Derivation of AEGL-1 Values

No AEGL-1 values were derived due to insufficient human and limited animal data.

TABLE 4. AEGL-1 Values for Stibine

10-minute	30-minute	1-hour	4-hour	8-hour
NR	NR	NR	NR	NR

NR-Not recommended. Absence of an AEGL-1 value does not imply that exposure below the AEGL-2 concentration is without adverse effects

6. DATA ANALYSIS FOR AEGL-2

6.1. Summary of Human Data Relevant to AEGL-2

No human data are available for the derivation of AEGL-2 values.

6.2. Summary of Animal Data Relevant to AEGL-2

Irreversible, nonlethal effects were reported by Price et al. (1979) in rats and guinea pigs exposed to 29.1 or 191 ppm for 30 min. Lesions induced in rodents after exposure at 29.1 ppm were thought not to be treatment related. At 191 ppm both species experienced minimal renal tubular calcification damage considered a precursor to irreversible lesions. The 29.1 ppm is the no effect level for irreversible renal effects as defined by Price et al. (1979).

6.3. Derivation of AEGL-2 Values

Data used for deriving AEGL-2 values are those from Price et al. (1979) that provide exposure response relationships for rats and guinea pigs exposed to stibine for 30 min at 29.1 to 333 ppm. As stated above, the 191 ppm exposure resulted in renal tubular dilation and calcification that would result in scarring. Pulmonary inflammation was also found in one guinea pig in this group. Eye irritation and closure were observed in rats. All animals experienced generalized depressed activity, but none were judged to have an impaired ability to escape. The lowest concentration, 29.1 ppm, was used as the point of departure as it resulted in the highest exposure without an AEGL-2 effect.

A total uncertainty factor of 10 was applied to account for interspecies extrapolation and intraspecies variability. A factor of 3 was applied for interspecies variability. Price et al. (1979) reported similar mortalities from pulmonary edema for rats (70%) and guinea pigs (70%) at 333 ppm and similar lesions (renal tubular dilation, pulmonary edema) were reported in both species at 191 ppm. An uncertainty factor of 3 was applied for intraspecies variability. Although the mechanism of toxicity is unknown, the point of departure is based on contact irritation in the lung, and that respiratory irritant action is not expected to vary a great deal among individuals. The intraspecies factor of 3 is supported by the steep-concentration response shown by Price et al. (1979) in two species. No lethality was observed when both species were exposed to 191 ppm for 30 min, but a 1.7-fold increase in concentration (333 ppm) for the same duration resulted in 70 and 70% lethality in rats and guinea pigs, respectively.

The concentration exposure time relationship for many irritant and systemically acting vapors and gases may be described by $C^n \times t = k$, where the exponent ranges from 0.8 to 3.5 (ten Berge et al. 1986). In lieu of a definitive data set allowing empirical derivation of the exponent n, temporal scaling was performed, using n = 3 when extrapolating to shorter time points and n = 1 when extrapolating to longer time points using the $C^n \times t = k$ equation (NRC 2001).

TABLE 5. AEGL-2 Values for Stibine

10-minute	30-minute	1-hour	4-hour	8-hour
4.2 ppm (21 mg/m^3)	2.9 ppm (15 mg/m^3)	1.5 ppm (7.7 mg/m^3)	0.36 ppm (1.8 mg/m^3)	0.18 ppm (0.92 mg/m^3)

7. DATA ANALYSIS FOR AEGL-3

7.1. Summary of Human Data Relevant to AEGL-3

No human data were available for the derivation of AEGL-3 values.

7.2. Summary of Animal Data Relevant to AEGL-3

Lethality data are available for two species, rats and guinea pigs (Price et al. 1979). Both species were exposed to 29.1, 191, or 333 ppm for 30 min. No treatment-related mortality was observed at the lower concentrations, but 70% of rats and 80% of guinea pigs died at 333 ppm. Death appeared to be caused by pulmonary edema.

7.3. Derivation of AEGL-3 Values

Data used for deriving AEGL-3 values are those from Price et al. (1979) for rats and guinea pigs exposed to stibine for 30 min at concentrations of 29.1, 191, or 333 ppm. Inhaling 333 ppm concentration resulted in 70% mortality in rats and 70% mortality in guinea pigs from pulmonary edema, most within 9 hours following exposure. One rat died four days post exposure.

The highest concentration with no treatment-related mortality (191 ppm, 30-min exposure in rats) was identified as the basis of the AEGL-3 derivation. One guinea pig died at 191 ppm. The irreversible lesion reported for the guinea pig that died was cortical necrosis of the cerebrum, and this lesion was found only in that animal. The lesion and death were not considered by Price et al. (1979) to be treatment-related. A total uncertainty factor of 10 was applied to account for

interspecies extrapolation and intraspecies variability. A factor of 3 was applied for interspecies variability. Price et al. (1979) reported similar mortalities from pulmonary edema for rats (70%) and guinea pigs (70%) at 333 ppm and similar lesions (renal tubular dilation, pulmonary edema) were reported in both species at 191 ppm. An uncertainty factor of 3 was applied for intraspecies variability. Although the mechanism of toxicity is unknown, the point of departure is based on contact irritation in the lung, and that respiratory irritant action is not expected to vary a great deal among individuals. The intraspecies factor of 3 is supported by the steep-concentration response shown by Price et al. (1979) in two species. No lethality was observed when both species were exposed to 191 ppm for 30 min, but a 1.7-fold increase in concentration (333 ppm) for the same duration resulted in 70 and 70% lethality in rats and guinea pigs, respectively.

The concentration exposure time relationship for many irritant and systemically acting vapors and gases may be described by $C^n \times t = k$, where the exponent ranges from 0.8 to 3.5 (ten Berge et al. 1986). In lieu of a definitive data set allowing empirical derivation of the exponent n, temporal scaling was performed, using n = 3 when extrapolating to shorter time points and n = 1 when extrapolating to longer time points using the $C^n \times t = k$ equation (NRC 2001).

TABLE 6. AEGL-3 Values for Stibine

10-minute	30-minute	1-hour	4-hour	8-hour
28 ppm (140 mg/m^3)	19 ppm (97 mg/m^3)	9.6 ppm (49 mg/m^3)	2.4 ppm (12 mg/m^3)	1.2 ppm (6.1 mg/m^3)

8. SUMMARY OF AEGLS

8.1. AEGL Values and Toxicity Endpoints

AEGL-1 values are not recommended due to lack of human data and limited animal data. AEGL-2 values are based on the highest experimental concentration not having AEGL-2 effects (29.1 ppm) for respiratory irritation and renal effects in rats and guinea pigs. AEGL-3 values are based on the highest concentration with no treatment-related mortality (191 ppm). AEGL values for stibine are summarized in Table 7.

TABLE 7. Summary of AEGL Values

Classification	Exposure Duration				
	10-minute	30-minute	1-hour	4-hour	8-hour
AEGL-1 (Notable discomfort)	NR	NR	NR	NR	NR
AEGL-2 (Disabling)	4.2 ppm (21 mg/m^3)	2.9 ppm (15 mg/m^3)	1.5 ppm (7.7 mg/m^3)	0.36 ppm (1.8 mg/m^3)	0.18 ppm (0.92 mg/m^3)
AEGL-3 (Lethal)	28 ppm (140 mg/m^3)	19 ppm (97 mg/m^3)	9.6 ppm (49 mg/m^3)	2.4 ppm (12 mg/m^3)	1.2 ppm (6.1 mg/m^3)

NR-Not recommended. Numeric values for AEGL-1 values not were derived due to insufficient human data,and the limited animal data was used to derive AEGL-2 values. Absence of an AEGL-1 value does not imply that exposure below the AEGL-2 concentration is without adverse effects.

8.2. Comparison with Other Standards and Guidelines

All currently available standards and guidelines are shown in Table 8. The American Industrial Hygiene Association (AIHA) emergency response planning guideline (ERPG) values, Occupational Safety and Health Administration (OSHA) time weighted average, National Institute of Occupational Safety and Health (NIOSH) immediately dangerous to life and health values, and time weighted average value, and Dutch maximum allowable concentration (MAC) values have been published. The American Conference of Governmental Industrial Hygienists, Threshold Limit Value (ACGIH) threshold limit value-time weighted average (TLV-TWA) is based on prevention of kidney and erythrocyte damage and prevention of respiratory tract irritation. No other standards or guidelines are available for stibine (antimony hydride). The AEGL values are consistent with currently established guidelines. The absence of AEGL-1 values is consistent with the AIHA ERPG decision not to recommend ERPG-1 values.

TABLE 8. Extant Standards and Guidelines for Stibine

Guideline	Exposure Duration				
	10 minute	30 minute	1 hour	4 hour	8 hour
AEGL-1	NR	NR	NR	NR	NR
AEGL-2	4.2 ppm	2.9 ppm	1.5 ppm	0.36 ppm	0.18 ppm
AEGL-3	28 ppm	19 ppm	9.6 ppm	2.4 ppm	1.2 ppm
ERPG-1 (AIHA)[a]			ID		
ERPG-2 (AIHA) [a]			0.5 ppm		
ERPG-3 (AIHA) [a]			1.5 ppm		
PEL-TWA(OSHA)[b]					0.1 ppm
IDLH (NIOSH)[c]		5 ppm			
REL-TWA (NIOSH)[d]					0.1 ppm
TLV-TWA (ACGIH)[e]					0.1 ppm
MAC (The Netherlands)[f]					0.1 ppm

NR-Not recommended. Numeric values for AEGL-1 values not were derived due to insufficient human data, and the limited animal data was used to derive AEGL-2 values. Absence of an AEGL-1 value does not imply that exposure below the AEGL-2 concentration is without adverse effects.
ID-insufficient data.

[a]ERPG (Emergency Response Planning Guidelines, American Industrial Hygiene Association (AIHA 2005)

- The ERPG-1 is the maximum airborne concentration below which it is believed nearly all individuals could be exposed for up to one hour without experiencing other than mild, transient adverse health effects or without perceiving a clearly defined objectionable odor. There was insufficient data to establish an ERPG-1 value.

- The ERPG-2 is the maximum airborne concentration below which it is believed nearly all individuals could be exposed for up to one hour without experiencing or developing irreversible or other serious health effects or symptoms that could impair an individual's ability to take protective action. The ERGP-2 is based on an analogy with the toxicity and the ERPG-3 level of arsine, set below the expected threshold for delayed disabling effects of intravascular hemolysis.

- The ERPG-3 is the maximum airborne concentration below which it is believed nearly all individuals could be exposed for up to one hour without experiencing or developing life-threatening health effects. The

ERPG-3 was based on an analogy with the toxicity and the ERPG-3 level of arsine, set below the expected threshold for life-threatening hemolysis.

[b]OSHA PEL-TWA (Occupational Safety and Health Administration, Permissible Exposure Limits - Time Weighted Average) (OSHA 2005) is defined analogous to the ACGIH-TLV-TWA, but is for exposures of no more than 10 hours/day, 40 hours/week.

[c]IDLH (Immediately Dangerous to Life and Health, National Institute of Occupational Safety and Health) (NIOSH 1996) represents the maximum concentration from which one could escape within 30 minutes without any escape-impairing symptoms, or any irreversible health effects.

[d]NIOSH REL-TWA (National Institute of Occupational Safety and Health, Recommended Exposure Limits - Time Weighted Average) (NIOSH 2005) is defined as the time-weighted average concentration for up to a 10-hour workday during a 40-hr workweek.

[e]ACGIH TLV-TWA (American Conference of Governmental Industrial Hygienists, Threshold Limit Value - Time Weighted Average) (ACGIH 2001) is the time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek, to which nearly all workers may be repeatedly exposed, day after day, without adverse effect.

[f] MAC (Maximaal Aanvaaarde Concentratie [Maximal Accepted Concentration]) Nationale MAC List (2000). (SDU Uitgevers [under the auspices of the Ministry of Social Affairs and Employment], The Hague, The Netherlands 2000 is defined analogous to the ACGIH-TLV-TWA.

8.3. Data Adequacy and Research Needs

The data reported for occupational exposures to stibine include actual concentration and /or duration parameters to which workers have been exposed. There are few reports of adverse effects at the levels measured; however, the complaints confirm the respiratory irritation of stibine exposure. The absence of human toxicity data is a key area where data are deficient, especially clinical data on the hemolytic potential associated with the hemolytic property of stibine. Quantitative animal data available from one study demonstrate a response similar to the reported human respiratory and eye irritation. The animal data are sufficient for showing lethality and non-incapacitating exposures. There were insufficient data for addressing exposure concentration-duration relationships as the studies exposed animals for only 30 minutes. Data on exposure durations would be useful in the development of more precise temporal extrapolation.

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3 **APPENDIX A: Derivation of AEGL Values**

Derivation of AEGL-1
No AEGL-1 values were derived due to insufficient human data, and the limited animal data was used to derive AEGL-2 and AEGL-3 values.

Key Study:

Toxicity endpoint:

Time scaling:

Uncertainty factors:

Modifying factor:

Calculations:

10-minute AEGL-1 = NR

30-minute AEGL-1 = NR

1-hour AEGL-1 = NR

4-hour AEGL-1 = NR

8-hour AEGL-1 = NR

Derivation of AEGL-2

Key Study: Price, N.H., W.G. Yates, S.D. Allen, and S.W. Waters. 1979. Toxicity evaluation for establishing IDLH values (Final Report) NTIS TR 1518-005. Utah Biomedical Test Laboratory, Salt Lake City, UT.

Toxicity endpoints: Highest experimental concentration (29.1 ppm) with no AEGL-2 effect.

Time scaling: $C^n \times t = k$, temporal scaling, using n = 3 when extrapolating to shorter time points and n = 1 when extrapolating to longer time points due to lack of data to derive a value of n (NRC 2001).

Uncertainty factors: A total uncertainty factor of 10 was applied to account for interspecies extrapolation and intraspecies variability. A factor of 3 was applied for interspecies variability. Price et al. (1979) reported similar mortalities from pulmonary edema for rats (70%) and guinea pigs (70%) at 333 ppm and similar lesions (renal tubular dilation, pulmonary edema) were reported in both species at 191 ppm. An uncertainty factor of 3 was applied for intraspecies variability. Although the mechanism of toxicity is unknown, the point of departure is based on contact irritation in the lung, and that respiratory irritant action is not expected to vary a great deal among individuals. The intraspecies factor of 3 is supported by the steep-concentration response shown by Price et al. (1979) in two species. No lethality was observed when both species were exposed to 191 ppm for 30 min, but a 1.7-fold increase in concentration (333 ppm) for the same duration resulted in 70 and 70% lethality in rats and guinea pigs, respectively.

Modifying factor: None

Calculations: 29.1 ppm/10 = 2.91 ppm

$$C^3 \times t = k$$
$$(2.91\ \text{ppm})^3 \times 30\ \text{min} = 739.265\ \text{ppm}^3\cdot\text{min}$$

$$C^1 \times t = k$$
$$2.91\ \text{ppm} \times 30\ \text{min} = 87.3\ \text{ppm min}$$

10-minute AEGL-2	$C^3 \times 10\ \text{min} = 739.265\ \text{ppm}^3\cdot\text{min}$
	$C = 4.2\ \text{ppm}$
30-minute AEGL-2	$C^1 \times 30\ \text{min} = 87.3\ \text{ppm}\cdot\text{min}$
	$C = 2.9\ \text{ppm}$
1-hour AEGL-2	$C^1 \times 60\ \text{min} = 87.3\ \text{ppm}\cdot\text{min}$
	$C = 1.5\ \text{ppm}$
4-hour AEGL-2	$C^1 \times 240\ \text{min} = 87.3\ \text{ppm}\cdot\text{min}$
	$C = 0.36\ \text{ppm}$
8-hour AEGL-2	$C^1 \times 480\ \text{min} = 87.3\ \text{ppm}\cdot\text{min}$
	$C = 0.18\ \text{ppm}$

Derivation of AEGL-3

Key Study: Price, N.H., W.G. Yates, S.D. Allen, and S.W. Waters. 1979. Toxicity evaluation for establishing IDLH values (Final Report) NTIS TR 1518-005. Utah Biomedical Test Laboratory, Salt Lake City, UT.

Toxicity endpoint: Highest experimental concentration (333 ppm) with no mortality from fatal pulmonary edema.

Time scaling: $C^n \times t = k$, temporal scaling, using n = 3 when extrapolating to shorter time points and n = 1 when extrapolating to longer time points due to lack of data to derive a value of n (NRC 2001).

Uncertainty factors: A total uncertainty factor of 10 was applied to account for interspecies extrapolation and intraspecies variability. A factor of 3 was applied for interspecies variability. Price et al. (1979) reported similar mortalities from pulmonary edema for rats (70%) and guinea pigs (70%) at 333 ppm and similar lesions (renal tubular dilation, pulmonary edema) were reported in both species at 191 ppm. An uncertainty factor of 3 was applied for intraspecies variability. Although the mechanism of toxicity is unknown, the point of departure is based on contact irritation in the lung, and that respiratory irritant action is not expected to vary a great deal among individuals. The intraspecies factor of 3 is supported by the steep-concentration response shown by Price et al. (1979) in two species. No lethality was observed when both species were exposed to 191 ppm for 30 min, but a 1.7-fold increase in concentration (333 ppm) for the same duration resulted in 70 and 70% lethality in rats and guinea pigs, respectively.

Modifying factor: None

Calculations: 191 ppm/10 = 19.1 ppm

$C^3 \times t = k$

$(19.1 \text{ ppm})^3 \times 30 \text{ min} = 209036.13 \text{ ppm}^3 \cdot \text{min}$

$C^1 \times t = k$

$19.1 \text{ ppm} \times 30 \text{ min} = 573 \text{ ppm min}$

10-minute AEGL-3	$C^3 \times 10 \text{ min} = 209036.13 \text{ ppm}^3 \cdot \text{min}$
	$C = 28 \text{ ppm}$
30-minute AEGL-3	$C^1 \times 30 \text{ min} = 573 \text{ ppm} \cdot \text{min}$
	$C = 19 \text{ ppm}$
1-hour AEGL-3	$C^1 \times 60 \text{ min} = 573 \text{ ppm} \cdot \text{min}$
	$C = 9.6 \text{ ppm}$
4-hour AEGL-3	$C^1 \times 240 \text{ min} = 573 \text{ ppm} \cdot \text{min}$
	$C = 2.4 \text{ ppm}$
8-hour AEGL-3	$C^1 \times 480 \text{ min} = 573 \text{ ppm} \cdot \text{min}$
	$C = 1.2 \text{ ppm}$

APPENDIX B: Time-Scaling Calculations

The concentration exposure time relationship for many irritant and systemically acting vapors and gases may be described by $C^n \times t = k$, where the exponent ranges from 0.8 to 3.5 (ten Berge et al. 1986). In lieu of a definitive data set allowing empirical derivation of the exponent n, temporal scaling was performed, using n = 3 when extrapolating to shorter time points and n = 1 when extrapolating to longer time points using the $C^n \times t = k$ equation (NRC 2001).

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2 **APPENDIX C: Derivation Summary for Stibine AEGLs**

ACUTE EXPOSURE GUIDELINE LEVELS FOR
STIBINE (CAS Reg. No. 7803-52-3)
DERIVATION SUMMARY

AEGL-1 VALUES

10-minute	30-minute	1-hour	4-hour	8-hour
NR	**NR**	**NR**	**NR**	**NR**
Key Reference: There were insufficient data to derive AEGL-1 values for stibine.				
Test Species/Strain/Number: Not Applicable				
Exposure Route/Concentrations/Durations: Not Applicable				
Effects: Not Applicable				
Endpoint/Concentration/Rationale: Not Applicable				
Uncertainty Factors/Rationale: Not Applicable				
Modifying Factor: Not Applicable				
Animal to Human Dosimetric Adjustment: Not Applicable				
Time Scaling: Not Applicable				
Data Adequacy: NR-Not recommended. Numeric values for AEGL-1 values were not derived due to insufficient human data, and the limited animal data was used to derive AEGL-2 and AEGL-3 values. Absence of an AEGL-1 value does not imply that exposure below the AEGL-2 concentration is without adverse effects.				

AEGL-2 VALUES

10-minute	30-minute	1-hour	4-hour	8-hour
4.2 ppm	**2.9 ppm**	**1.5 ppm**	**0.36 ppm**	**0.18 ppm**

Key Reference: Price, N.H., W.G. Yates, S.D. Allen, and S.W. Waters. 1979. Toxicity evaluation for establishing IDLH values (Final Report) TR 1518-005. Salt Lake City, UT.

Test Species/Strain/Number: Rat/Sprague-Dawley /5/sex/concentration, Guinea Pig/Hartley5/sex/concentration

Exposure Route/Concentrations/Durations: Inhalation (Whole Body): 0, 29.1, 191, or 333 ppm for 30 minutes

Effects: Irreversible Effects

29.1 ppm	No exposure-related adverse effects.
191 ppm	Generalized depressed activity persistent for 4 hours post exposure, renal tubule dilation, pulmonary inflammation, eye irritation and closure in rats; Rat 0% mortality, Guinea pig 10% mortality (not treatment-related)
333 ppm	Generalized depressed activity persistent for 4 hours post exposure, dyspnea, tremors, pulmonary congestion and edema; rat 70 % mortality; guinea pig 70% mortality

Endpoint/Concentration/Rationale: The highest concentration (29.1 ppm for 30 min) with no AEGL-2 effects

Uncertainty Factors/Rationale:
Interspecies 3: Price et al. (1979) reported similar mortalities from pulmonary edema for rats (70%) and guinea pigs (70%) at 333 ppm, and similar renal effects were noted for both species at 191 ppm.
Intraspecies 3: Although the mechanism of toxicity is unknown, the point of departure is based on contact irritation in the lung, and that respiratory irritant action is not expected to vary a great deal among individuals. The intraspecies factor of 3 is supported by the steep-concentration response shown by Price et al. (1979) in two species. No lethality was observed when both species were exposed to 191 ppm for 30 min, but a 1.7-fold increase in concentration (333 ppm) for the same duration resulted in 70 and 70% lethality in rats and guinea pigs, respectively.

Modifying Factor: None

Animal to Human Dosimetric Adjustment: None

Time Scaling: In the absence of an empirically derived exponent (n), and to obtain AEGL values, $C^n \times t = k$, temporal scaling, using n = 3 when extrapolating to shorter time points and n = 1 when extrapolating to longer time points (NRC 2001).

Data Adequacy: The study was considered adequate for AEGL-2 derivation. It was a well-designed and performed study, and an adequate numbers of animals were used.

AEGL-3 VALUES

10-minute	30-minute	1-hour	4-hour	8-hour
28 ppm	**19 ppm**	**9.6 ppm**	**2.4 ppm**	**1.2 ppm**

Key Reference: Price, N.H., W.G. Yates, S.D. Allen, and S.W. Waters. 1979. Toxicity evaluation for establishing IDLH values (Final Report) TR 1518-005. Salt Lake City, UT.

Test Species/Strain/Number: Rat/Sprague-Dawley /5/sex/concentration, Guinea Pig/Hartley5/sex/concentration

Exposure Route/Concentrations/Durations: Inhalation: 0, 29.1,191, or 333 ppm for 30 min.

Effects: Lethality
29.1 ppm No mortality
191 ppm Rat 0% mortality, Guinea Pig 10% mortality (not treatment-related)
333 ppm Rat 70 % mortality, Guinea Pig 70% mortality

Endpoint/Concentration/Rationale: The highest experimental concentration (191 ppm for 30 minutes) with no mortality.

Uncertainty Factors/Rationale:
Interspecies 3: Price et al. (1979) reported similar mortalities from pulmonary edema for rats (70%) and guinea pigs (70%) at 333 ppm, and similar renal effects were noted for both species at 191 ppm.
Intraspecies 3: Although the mechanism of toxicity is unknown, the point of departure is based on contact irritation in the lung, and that respiratory irritant action is not expected to vary a great deal among individuals. The intraspecies factor of 3 is supported by the steep-concentration response shown by Price et al. (1979) in two species. No lethality was observed when both species were exposed to 191 ppm for 30 min, but a 1.7-fold increase in concentration (333 ppm) for the same duration resulted in 70 and 70% lethality in rats and guinea pigs, respectively.

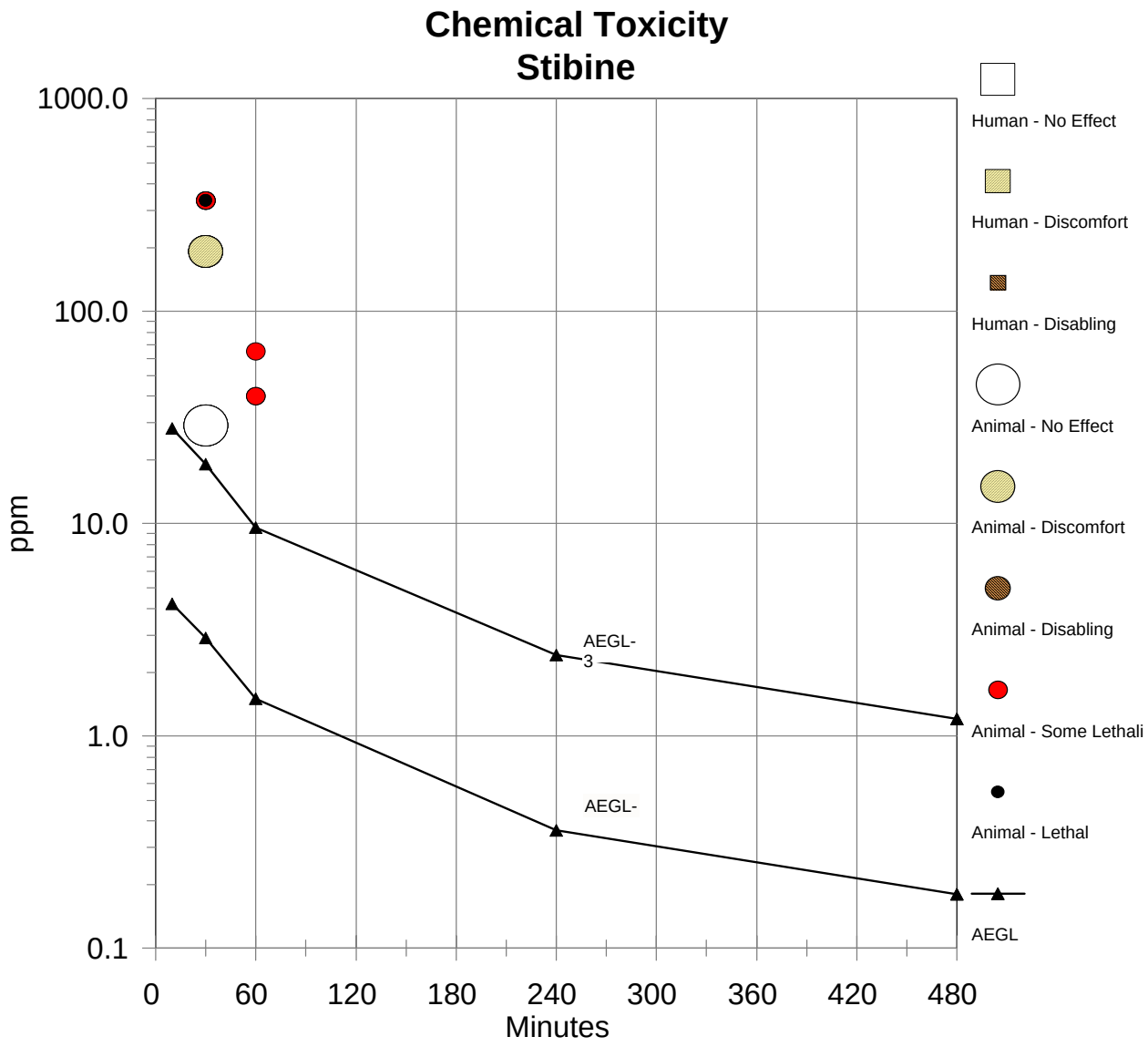
Modifying Factor: None

Animal to Human Dosimetric Adjustment: None

Time Scaling: In the absence of an empirically derived exponent (n), and to obtain AEGL values, $C^n \times t = k$, temporal scaling, using n = 3 when extrapolating to shorter time points and n = 1 when extrapolating to longer time points (NRC 2001).

Data Adequacy: The study was considered adequate for AEGL-3 derivation. It was a well-designed and performed study, adequate numbers of animals were used, and an endpoint consistent with AEGL-3 definition and toxicity of stibine was observed.

APPENDIX D: Category Plot for Stibine AEGLs


Chemical Toxicity
Stibine
1000.0
100.0
10.0
1.0
0.1
ppm
0
60
120
180
240
300
360
420
480
Minutes
AEGL-3
AEGL-
Human - No Effect
Human - Discomfort
Human - Disabling
Animal - No Effect
Animal - Discomfort
Animal - Disabling
Animal - Some Lethali
Animal - Lethal
AEGL

1

1 Category Plot Data

							For Category 0 = No effect, 1 = Discomfort, 2 = Disabling, SL = Some Lethality, 3 = Lethal
Source	Species	Sex	Exposures #	ppm	Time (min)	Category	Comments
NAC/AEGL-1				1	10	AEGL	
NAC/AEGL-1				1	30	AEGL	
NAC/AEGL-1				1	60	AEGL	
NAC/AEGL-1				1	240	AEGL	
NAC/AEGL-1				1	480	AEGL	
NAC/AEGL-2				4.2	10	AEGL	
NAC/AEGL-2				2.9	30	AEGL	
NAC/AEGL-2				1.5	60	AEGL	
NAC/AEGL-2				0.36	240	AEGL	
NAC/AEGL-2				0.18	480	AEGL	
NAC/AEGL-3				28	10	AEGL	
NAC/AEGL-3				19	30	AEGL	
NAC/AEGL-3				9.6	60	AEGL	
NAC/AEGL-3				2.4	240	AEGL	
NAC/AEGL-3				1.2	480	AEGL	
Webster 1946	dog		1	40	60	SL	Pulmonary edema
Webster 1946	guinea pig		1	65	60	SL	Changes in erythrocytes
Webster 1946	cat		1	40	60	SL	Pulmonary edema
Price et al. 1979	rat	M	1	29.1	30	0	No effects
Price et al. 1979	rat	F	1	29.1	30	0	No effects
Price et al. 1979	rat	M	1	191	30	1	Eye irritation and closure
Price et al. 1979	rat	F	1	191	30	1	Eye irritation and closure
Price et al. 1979	rat	M	1	333	30	3	100% mortality
Price et al. 1979	rat	F	1	333	30	SL	40% mortality
Price et al. 1979	guinea pig	M	1	29.1	30	0	No effects
Price et al. 1979	guinea pig	F	1	29.1	30	0	No effects
Price et al. 1979	guinea pig	M	1	191	30	1	Renal tubule dilation
Price et al. 1979	guinea pig	F	1	191	30	1	Renal tubule dilation
Price et al. 1979	guinea pig	M	1	333	30	SL	80% mortality
Price et al. 1979	guinea pig	F	1	333	30	SL	60% mortality

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