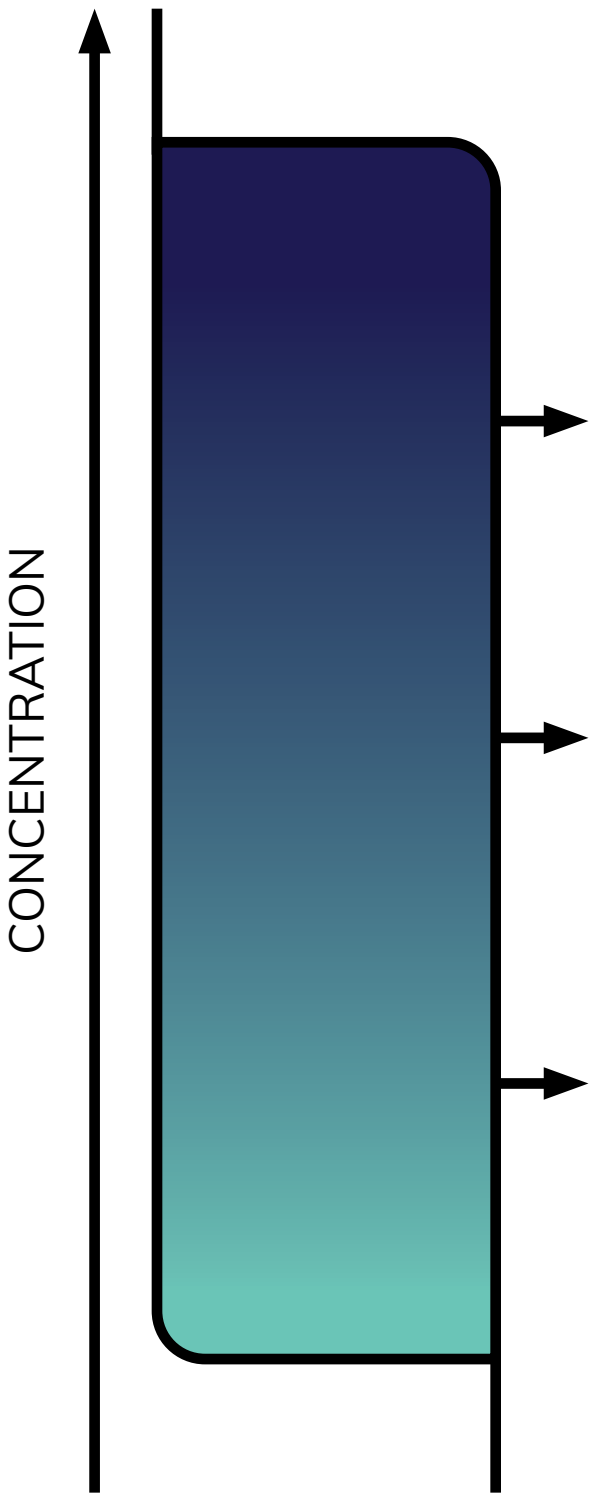
Emergency Response Planning Committee
GUIDELINE FOUNDATION

Emergency Response Planning Guideline Technical Supporting Documentation

Hydrogen Cyanide



ERPG-3: 25 ppm (27.75 mg/m^3)

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

ERPG-2: 10 ppm (11.1 mg/m^3)

The maximum airborne concentration below which 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.

ERPG-1: Not Applicable

The maximum airborne concentration below which nearly all individuals could be exposed to for up to one hour without experiencing other than mild, transient adverse health effects or without perceiving a clearly defined objectionable odor.

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EMERGENCY RESPONSE PLANNING GUIDELINE
TECHNICAL SUPPORTING DOCUMENTATION

HYDROGEN CYANIDE (2023)

ERPG–3: 25 ppm (27.75 mg/m^3)

ERPG–2: 10 ppm (11.1 mg/m^3)

ERPG–1: Not Applicable

I. IDENTIFICATION (AIHA 1982)(Baskin and Brewer 1997)(NIOSH 1976)(NIOSH 2004)(NIOSH and OSHA 1978)

Chemical Name:	Hydrogen cyanide
Synonyms:	Hydrocyanic acid; prussic acid; formonitrile; AC (NATO designation)
CAS Number:	74-90-8
DOT Number:	UN 1051
Molecular Formula:	HCN Structural Formula: HC ≡ N

II. CHEMICAL AND PHYSICAL PROPERTIES (PubChem 2022)

Physical State and Appearance:	At room temperature, hydrogen cyanide is a colorless gas or colorless to bluish volatile liquid.
Odor Description:	Bitter almonds, peach kernels, sweetish
Molecular Weight:	27.03
Conversion Factors:	1 mg/m^3 = 0.9 ppm 1 ppm = 1.11 mg/m^3
Melting Point:	-13.4°C (7.9°F)
Boiling Point:	25.6°C (78.1°F)
Vapor Pressure:	630 -742 mmHg 81.3 kPa (20°C)
Saturated Vapor Concentration:	96.5% at 25°C (77°F) and 760 mmHg 0.9 106 mg/m^3 (20°C)
Vapor Density (Air = 1):	0.94 at 20°C (68°F)
Flash Point:	-17.78°C (0°F) closed cup
Flammability Limits:	Lower explosive limit – 5.6% Upper explosive limit – 40.0%
Autoignition Temperature:	538°C (1000°F)
Reactivity and Incompatibilities:	When not absolutely pure or stabilized, can polymerize spontaneously with explosive violence. Presence of alkali favors explosive polymerization. Liquid will attack some forms of plastics, rubber, and coatings. Water solutions cause stress-cracking of carbon steels. Water solutions containing sulfuric acid as stabilizer severely corrode steel above 40 deg C and stainless steels above 80 deg C.
Solubility:	Miscible in water, ethanol, ether

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III. ANIMAL TOXICITY DATA

A. Acute Toxicity and Irritancy

1. Oral Toxicity

In a study with female rabbits (minimum of 4 dose levels, 6 – 10 animals/dose) the oral LD_{50} of HCN in aqueous solution was 0.092 - 0.156 m mol/kg (3.4 – 5.8 mg/kg) (Ballantyne 1983a)

2. Eye Irritation/Toxicity

In a study with female rabbits (minimum of 4 dose levels, 6 – 10 animals/dose) the LD_{50} of HCN was 0.39 m mol/kg (1.44 mg/kg) after administration of HCN in an aqueous solution into the inferior conjunctival sac (Ballantyne 1983b)

3. Skin Toxicity

3a. Skin Absorption

The dermal LD_{50} in female rabbits (minimum of 4 dose levels, 6 – 10 animals/dose) was 0.26 m mol/kg (9.6 mg/kg). (BAL1983a)

Hair was clipped from the abdomens of 5 guinea pigs that were then exposed to HCN vapor concentrations of about 312,000 to 455,000 ppm. Animals were restrained, and a spring device was used to maintain contact of mouth of the flask with HCN vapor to the abdomen. The exposed skin area (1.23 in^2) was estimated to be 2% of the body surface. Onset of labored breathing, collapse, convulsions, and coma varied from 15 to 55 minutes. Four guinea pigs died after 45 to 97 minutes. The fifth collapsed and was removed from exposure after 15 min; it recovered completely after 45 minutes. (Fairley 1934)

3b. Skin Irritation

No information available.

3c. Skin Sensitization

No information available.

4 Inhalation Toxicity

Lethality Studies

Lethality data for rats, mice and rabbits is given in Table I. Concentrations are analytical unless otherwise noted. Methods of analysis included Fourier Transform Infrared Spectrometer (FT-IR), IR validated by gas chromatography, and a cyanide-specific electrode calibrated against a standard.

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Table 1. Lethality Data in Rats, Mice, and Rabbits and Swine (concentrations are analytical unless otherwise noted)

Species	Concentration (ppm)	Duration (min)	Value	References
Rat	383	5	Highest non-lethal (0/10)	Sweeney et al 2014
Rat	198	15	Highest non-lethal (0/10)	Sweeney et al 2014
Rat	141	30	Highest non-lethal (0/10)	Sweeney et al 2014
Rabbit	128	35	Highest non-lethal (0/3)	Ballantyne 1994
Rat	86	60	Highest non-lethal (0/10)	Ballantyne 1994
Mouse	100 (nominal)	60	Highest non-lethal (0/4)	Matijak-Schaper and Alarie 1982
Rat	590	5	Lowest-lethal (2/10)	Sweeney et al 2014
Rat	272	15	Lowest-lethal (2/10)	Sweeney et al 2014
Rat	261	30	Lowest-lethal (1/10)	Sweeney et al 2014
Rabbit	138	35	Lowest-lethal (2/3)	Ballantyne 1994
Rat	121	60	Lowest-lethal (5/10)	Ballantyne 1994
Mouse	150 (nominal)	60	Lowest-lethal (2/4)	Matijak-Schaper and Alarie 1982
Rat	48.8 (nominal)	30	LC10	E.I. DuPont de Nemours 1986
Swine	5331*	1	LC50	Staugler et al 2018
Rat	774*	5	LC50	Sweeney et al 2014
Rat	423*	15	LC50	Sweeney et al 2014
Rat	261*	30	LC50	Sweeney et al 2014
Rat	152 (nominal)	30	LC50	E.I. DuPont de Nemours 1986
Rat	196	15	LC50	E.I. du Pont de Nemours 1981
Rat	163**	15	LC50	E.I. du Pont de Nemours 1981
Rat	173	30	LC50	E.I. du Pont de Nemours 1981
Rat	85**	30	LC50	E.I. du Pont de Nemours 1981
Rat	173 (nominal)	30	LC50	Ballantyne 1983a
Rat	160**	30	LC50	Levin et al. 1987
Rat	158	60	LC50	Ballantyne 1983a
Rat	143	60	LC50	Ballantyne 1994
Rat	139	60	LC50	E.I. du Pont de Nemours 1981
Rat	63**	60	LC50	E.I. du Pont de Nemours 1981
Mouse	166 (nominal)	30	LC50	Matijak-Schaper and Alarie 1982
Rabbit	208 (nominal)	35	LC50	Ballantyne 1983
Rabbit	188	35	LC50	Ballantyne 1994
Mouse	750 (nominal)	1-2	LC100	Matijak-Schaper and Alarie 1982
Rat	373 (nominal)	30	LC90	E.I. DuPont de Nemours 1986
Mouse	500 (nominal)	9-16	LC100	Matijak-Schaper and Alarie 1982
Mouse	330 (nominal)	14-30	LC100	Matijak-Schaper and Alarie 1982

** Nose only exposure*
***Animals were restrained (The additional stress lowers the LC50)*

The 10-min RD50 in rats and mice was reported as 125 ppm and 63 ppm, respectively. (E.I. du Pont de Nemours 1981) (Matijak-Schaper & Alarie, 1982)

As part of a study designed to test whether the toxic load model was valid when applied to intermittent acute exposures, male Sprague-Dawley rats were exposed nose-only to constant concentrations of HCN for 5, 15, or 30 minutes (Sweeney et al 2014). Concentrations ranged from 141 ppm to 966 ppm (Ct ranged from 1915 ppm-min to 9090 ppm-min). No concentration resulted in 100% mortality. Clinical signs in survivors included lethargy, labored breathing, tremors and impaired movement, and bloody nose and mouth. All clinical signs in survivors reversed within minutes upon cessation of exposure.

In a study of dynamic pulmonary mechanics, 30-minute exposures to HCN at a static mean concentration of HCN of 55 ppm were well-tolerated by rats with no deaths (Bhattacharya 1994). Air flow rate, transthoracic pressure, and tidal volume were measured at 10, 20, and 30 minutes of exposure; all parameters were significantly increased.

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Compliance, respiratory rate, and minute volume declined progressively through the exposure. No change in airway resistance was observed.

Incapacitation Studies

Four cynomolgus monkeys (*Macaca fasicularis*) were exposed to 60 ppm (nominal) HCN administered via a face mask. (Purser 1984a) Respiratory parameters (rate, tidal volume, and minute volume), EKG, EEG, auditory evoked response, and peripheral nerve conduction velocity were monitored in 30-minute pre-exposure, exposure, and post-exposure periods. Because all measurements could not be made simultaneously, each monkey was exposed three times, but there is no indication of the time interval between successive exposures or of the order in which the physiological parameters were measured. Parameters under examination returned to pre-exposure levels prior to subsequent exposures. (The animals were also exposed hypoxic (15% and 10% O2) and hypercapnic (5% CO_2) atmospheres. There is no indication of the time interval between these exposures and the HCN exposures.) Effects of 60 ppm HCN included a 39% increase in minute volume and 4.5% increase in nerve conduction time.. In two animals for which data is reported, there was a 15% and 19% change in the distribution of EEG power over 1-30 Hz. While the latency of the EP was unaffected, the amplitude of the late response was decreased by 26%. No effects were seen on cardiovascular parameters.

Over the range of 80-180 ppm marked hyperventilation was followed by incapacitation during the 30 minute exposure. Above 180 ppm severe hyperventilation occurred within 1-2 minutes, followed by loss of consciousness within 5 minutes.

Subsequently, cynomolgus monkeys were exposed via face mask to 100–156 ppm HCN (1 monkey per concentration) and times to “incapacitation” were recorded (Purser et al 1984b). Each test session consisted of 3 consecutive 30-minute periods: Pre-exposure period to establish baseline parameters; exposure period; recovery period. Time to incapacitation is shown in Table III. Severity of clinical signs required termination of 3 of the 5 exposures before the full 30- minute exposure was completed. Prior to incapacitation, effects included increased activity and marked hyperventilation, with an increase in the minute volume of up to four-fold. This was followed within 1–5 minutes by semiconsciousness, with a loss of muscle tone and limb reflexes, reduced heart rate with arrhythmia, slowed respiration, and reduced minute volume with a pause at the end of each expiration, and with EEG evidence of severe central nervous system depression.

Table II. Cynomolgus monkeys — Time to Incapacitation (Purser et al 1984b)

Concentration (ppm HCN)	Time to Incapacitation (minutes)
156	8
147*	8
123	15
102	16
100	19

** the same monkey was previously exposed to polyacrylonitrile and its thermal decomposition product HCN; HCN concentration was 124 ppm and time to incapacitation was 19 min..*

Incapacitation studies have been conducted in rats as well. Time to incapacitation was 5 minutes for rats exposed to 184 ppm HCN and 35 minutes for rats expose to 64 ppm (Chaturvedi et al 1995). The time to incapacitation was significantly reduced in when rats were co-exposed to HCN and CO. Further, the blood parameters were also significantly altered in the combined exposures.

Male rats (n=30) were exposed individually to mean concentrations of 75 – 273 ppm HCN (Crane et al 1989). Time to incapacitation was determined by shock avoidance, and ranged from 2.8 min at the highest concentrations to 22.6 min at the lower concentrations.

In a test of the toxic load model, rats were exposed to HCN in pulsed exposures at non-constant concentrations and with time profiles that did or did not include a time gap between the pulsed exposures.(Sweeney et al 2014) In 6 out of 8 exposures, the exposure results conformed to the toxic load model. But in 2 of the 8 exposures, the results were different from what the toxic load model would predict. The exposure scenarios that did not match the toxic load model were very short exposures (5 minutes) to very high concentrations.

The studies by Sweeney et al were used to determine whether time to incapacitation as a point of departure could be calculated from the LC01 for HCN or CO. (Pauluhn 2016). The LCt01 divided by a factor of 3 was considered to be most suitable and robust to estimate the human equivalent threshold (POD) of impairment of escape. In the absence of a more robust data set, this appears to provide a simple way of providing a conservative prediction of the time to incapacitation in a one hour exposure.

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B. Subchronic Toxicity

1. *Oral Toxicity*

No information available for HCN.

Subchronic studies of KCN, NaCN, and other cyanide salts have been reviewed. (ICPS-CICAD 2004). Effects include reduced weight gain and histological changes to the kidney, liver, and thyroid. There are no qualitative differences in acute poisoning between cyanide compounds, since the cyanide ion is the common agent that primarily inhibits tissue cytochrome oxidase activity in rats, mice, and rabbits, with resulting anoxia (Way 1984)(US EPA 1988) (as cited in ICPS 2004).

2. *Inhalation Toxicity*

Male rats received nose-only dynamic exposures to wood or polyurethane smoke with CO, HCN, or both added. There were five 12½-minute exposures, with 4 days between each exposure. Independent of CO, HCN at 200–230 ppm induced mild cardiotoxicity as determined by plasma levels of cardiac-specific creatine phosphokinase and norepinephrine-induced ectopic heart beats. (O'Flahrety 1982)

No statistically significant increase in the incidence of histopathological changes in the lungs or cardiovascular tissues (e.g., myocardial ultrastructure) compared with the controls was noted in male rabbits continuously exposed to 0.45 ppm hydrogen cyanide for 4 weeks. (Hugod 1981)

Dogs exposed to 45 ppm hydrogen cyanide for 30 min every third day for 28 days exhibited extensive vascular and cellular central nervous system lesions, including vasodilation and hemorrhages (Valade 1952 as cited in ICPS-CICAD 2004)

C. Chronic Toxicity and Carcinogenicity

In a 2-year dietary study, rats (10/sex/group) were administered food fumigated with HCN (stored in special containers to limit volatilization) (Howard and Hanzal 1955). Nominal concentrations were 100 and 300 ppm HCN; a later analysis estimated the average concentrations to be 0, 73, and 183 mg/kg (US EPA 2010). No treatment-related effects on survival or growth rate, signs of toxicity, hematological or histopathological changes were observed.

D. Reproductive and Developmental Toxicity

No information is available for HCN. In 13-week studies, groups of F344/N rats and B6C3F1 mice (10 of each sex) were administered 0, 3, 10, 30, 100, or 300 mg sodium cyanide/l in drinking-water (NTP, 1993). The equivalent cyanide ion doses were 0, 0.2, 0.5, 1.4, 4.5, and 12.5 (in males) and 0, 0.2, 0.5, 1.7, 4.9, and 12.5 (in females) mg cyanide/kg body weight per day for rats and 0, 0.3, 1, 3, 9, and 26 mg cyanide/kg body weight per day for mice. There were no clinical signs related to cyanide exposure observed in the rats or mice, nor were there gross or histopathological lesions. Reproductive effects included a reduction in sperm motility and weight of the cauda epididymidis in all groups of male rats, and increased time in proestrus and diestrus relative to estrus and metestrus female rats at the concentrations of 100 and 300 mg/L.

E. Genotoxicity/Mutagenicity

Hydrogen cyanide induced mutations in S. typhimurium TA100 in the absence of S9 activation but was not mutagenic to strain TA98 with or without S9 activation (Kushi et al. 1983, as cited in ICPS-CICAD 2004).

F. Metabolism and Pharmacokinetics

Hydrogen cyanide reversibly inhibits cytochrome oxidase, a key enzyme in cellular respiration. This inhibition prevents efficient use of oxygen as an electron acceptor and ultimately causes death from cytotoxic anoxia. Detoxification is principally by formation of thiocyanate, which is excreted in urine. These molecular mechanisms are the same for all mammalian species.(Baskin and Brewer 1997)(IPCS 2004)(ATSDR 2006)(Egekeze and Oehme 1980)(Eyer 1999)

V. HUMAN EXPERIENCE

A. Odor Data

Cyanide is described as having the odor of bitter almond, or musty (AIHA 2019), although some individuals are unable to perceive the odor of cyanide (Eyer 1999). The odor threshold for HCN has been variously reported as 0.009 - 5.43 ppm (AIHA). It was estimated that at 10 ppm, less than 50% of distracted persons would perceive a warning odor. (Amoore 1983) The level of awareness has not been reported for HCN.

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B. Acute Toxicity

1. *Oral Toxicity*

Lethal adult dose of HCN is 50 - 100 mg as total or 0.7 – 3.5 mg/kg. (DuBois and Geiling 1959, cited in Ballantyne & Salem 2008) (Hallstrom and Moller 1945, cited in Ballantyne & Salem 2008)

2. *Eye Toxicity*

Rare instances of sub-lethal cyanide poisoning that caused transient blindness from injury to the optic nerve have occurred (Grant 1974).

3. *Dermal Toxicity*

HCN vapor can be absorbed in toxic amounts through intact skin (Fairley et al 1934). Four firefighters wearing self-contained breathing apparatus and structural fire-fighting protective clothing entered an office contaminated with HCN vapor (Lam and Lau 2000). Following exposures of 5 to 30 minutes, all developed symptoms of flushing, dizziness, headache, throat discomfort, chest tightness, skin itchiness, and eye irritation. Three were discharged from the hospital after 12 hours observation and the fourth was observed for 3 days due to EKG irregularities.

4. *Inhalation*

The exposure-response curve in humans is steep (IPCS 2004), and HCN does not follow Haber's law (C x t = k). (Baskin and Brewer 1997)(Eyer 1999). A summary of reported symptoms and estimated exposure concentrations is given in Table IV. A summary of calculated toxicity levels is given in Table V.

Acute hydrocyanic acid poisoning leading to prolonged unconsciousness with CNS anoxia might leave "more or less permanent" CNS injury. (von Oettingen 1952) Transient blindness and damage to the optic nerves and retina might be consequences of non-lethal acute HCN intoxication. (Grant 1974)

Signs and symptoms of cyanide inhalation include hyperventilation, a brief sensation of dryness and burning in the nose and throat, retrosternal pain, and conjunctival itching. Frontal headache, palpitations, air hunger, and nausea may follow but disappear rapidly if only a small amount of cyanide was inhaled. The first breath of high concentrations may produce immediate hyperpnea, a few gasps, collapse, and convulsions in less than a minute. (Eyer 1999)

5. *Other*

The estimated human LD50 is stated as 1.0 mg/kg intravenously and 100 mg/kg for liquid on skin. (Baskin and Brewer 1997)

C. Workplace Experience

While developing photographic plates, a worker experienced three separate episodes of mild cyanide poisoning (numbness, weakness, vertigo, nausea, rapid pulse, and flushing of the face) after an hour's work. (Parmenter 1926) Following improvements to the dark room ventilation system, air sampling found 25–50 ppm HCN in the general atmosphere. The day following these attacks, this worker reported headache and vague gastric distress with rapid return to normal. Two co-workers in the same dark room experienced no ill effects, and the affected worker was thought to have had increased susceptibility due to pre-existing heart disease.

Three flasks of dilute HCN were left uncovered overnight, and the following day a female worker was found unconscious after being alone in her office for 4 hours. (Lam and Lau 2000) The only information on exposure concentration was the statement that it exceeded the 30 ppm upper limit measurable by Drager tube, but it was not reported when that measurement was attempted. When discovered, the woman was removed from exposure by a co- worker. She was transported to the hospital, where she suffered convulsions and required intubation along with treatment for cyanide intoxication. She recovered consciousness and was extubated after 15 hours and was discharged from the hospital after 4 days. Her condition was normal at 2-week and 1-year follow-ups, except for complaints at 1 year of mild impairment of recent memory and concentration. After evacuating the unconscious woman, her rescuer returned to the office and opened windows. After 10 minutes of exposure he experienced severe dizziness and almost fainted. Transported to the hospital, he was treated for cyanide intoxication and was discharged after 3 days. There was no follow-up.

Four firefighters who responded to this HCN incident entered the office wearing self-contained breathing apparatus and structural fire-fighting protective clothing. (Lam and Lau 2000) Their stay in the office ranged from 5 to 30 minutes. All were sent to the emergency department after developing mild symptoms of flushing, dizziness, headache, throat discomfort, chest tightness, skin itchiness, and eye irritation. Three were discharged from the hospital after 12 hours of observation. The fourth exhibited EKG abnormalities but was discharged after 3 days of observation. A fifth firefighter, who wore no protective equipment but remained about 100 feet away from the contaminated office, also experienced

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chest discomfort, flushing, headache, and eye and skin itchiness. He was transported to the emergency department and discharged after 12 hours of observation.

A summary of reported symptoms is given in Table III. A summary of calculated toxicity levels is given in Table IV.

Table III. Reported Human Responses to Estimated Concentrations of HCN Vapor

Concentration (ppm)	Duration	Effect	References
20	Prolonged exposure	Maximum concentration allowable	Henderson and Haggard 1943
18 – 36		slight effects	ICPS 2004, Lehmann and Hess 1942
20 – 40	Several hours	Slight symptoms	Henderson and Haggard 1943
45 – 54	20 min to 1 h	tolerated without immediate or late effects	ICPS 2004, Lehmann & Hess 1942
50 – 60	1 hour	Maximum concentration without serious consequences	Henderson and Haggard 1943
100 – 240	30 min – 1 hr	Dangerous	Henderson and Haggard 1943
109 – 136	0.5–1 h	dangerous to life and may lead to death	ICPS 2004, Lehmann & Hess 1942
181	10 min	likely to be fatal	ICPS 2004
136	30 min	likely to be fatal	ICPS 2004
271	Immediate	fatal	ICPS 2004, Lehmann & Hess 1942
200 – 480	30 min	fatal	Henderson and Haggard 1943
3000		Rapidly fatal	Henderson and Haggard 1943

It should be emphasized that many of these estimates represent crude average exposure estimates, based on various studies.

Table IV. Calculated Effect Levels in Humans

Concentration (ppm)	Duration	Effect	References
44	60 min	EC50, severe effects (incapacitation, convulsions, unconsciousness)	Sommerville 2011
2261 - 4523	1 min	LC50	Baskin 1997
208	30 min	LC10	E.I DuPont Nemours 1986
277	30 min	LC50	E.I DuPont Nemours 1986
373	30 min	LC90	E.I DuPont Nemours 1986
41	60 min	LC01	INERIS (in Sommerville) 2011
63	60min	LC05	Sommerville 2011
82	60 min	LC50	Sommerville 2011
78	60 min	LC50	Sommerville 2011Sommerville 2011)
71	60 min	LC50	Sommerville 2011

D. Epidemiology

No information available.

V. CURRENT EXPOSURE GUIDELINES

A. The National Research Council's 1-hour Acute Exposure Guideline Levels (AEGLs) (NRC 2005) are:

AEGL-3 – 15 ppm (17 mg/m^3)
AEGL-2 – 7.1 ppm (7.8 mg/m^3)
AEGL-1 – 2 ppm (2.8 mg/m^3)

B. The Occupational Safety and Health Administration Permissible Exposure Limit (PEL) is 10 ppm (11 mg/m^3) as an 8-hour TWA with a "skin" notation. (OSHA Table Z-1)

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C. The NIOSH Recommended Exposure Limit (REL) is a 10-minute short-term limit of 4.7 ppm (5 mg/m^3 expressed as CN) with a “skin” notation.(4,5)

D. The American Conference of Governmental Industrial Hygienists Threshold Limit Value (TLV) is a ceiling value of 5 mg/m^3 (4.7 ppm) (expressed as CN), with a “skin” notation. (31, 33)

VI. RECOMMENDED ERPGS™ AND RATIONALES

A. ERPG–3: 25 ppm (27.75 mg/m^3)

25 ppm in the maximum concentration below which nearly all individuals could be exposed to for up to 1 hour without experiencing or developing life-threatening health effects. Reported 1-hour LC50 values are 139, 143, and 158 ppm in rats (Ballantyne 1983) (Ballantyne 1994)(DuPont 1981). The lowest non-lethal level is 86 ppm in rats and 100 ppm in mice. The 60-min human LC01 has been calculated as 41 ppm (INERIS 2002, 2005 in Sommerville 2011). Human experience is reported as indicating that 100 ppm and higher is dangerous to life after a 30-minute to 1-hour exposure, whereas 20 to 50 ppm is described as producing symptoms but having no serious effect after 1 to several hours of exposure. (IPCS 2004)(Henderson and Haggard 1943)(Lehmann and Hess 1942) Dogs exposed to 45 ppm HCN for 30 min every third day for 28 days exhibited extensive vascular and cellular central nervous system lesions, including vasodilation and hemorrhages (Valade 1952 as cited in ICPS 2004). Based on these data, an ERPG-3 level of 25 ppm was selected in order to protect most individuals in the general population.

B. ERPG-2: 10 ppm (11.1 mg/m^3)

10 ppm is the maximum airborne concentration below which nearly all individuals could be exposed for up to 1 hour without experiencing or developing irreversible or other serious adverse health effects or symptoms that could impair an individual’s ability to take protective action. The calculated 1-hour EC50 for incapacitation is 44 ppm (Sommerville 2011). In human exposures, 20 – 40 ppm was tolerated for several hours with slight sympoms (Henderson and Haggard 1943) and 45 – 54 ppm was tolerated for up to 1-hour without immediate or late effects (ICPS 2004). Rats tolerated 30-minute exposures to 55 ppm well. (Bhattacharya et al 1994) Monkeys exposed to 100 ppm or more were incapacitated in less than 30 minutes, whereas monkeys exhibited only minor effects after 30-minute exposure to 60 ppm. (Purser 1984a) (Purser et al 1984b) The RD50 in mice was 63 ppm and the RD50 in rats was 125 ppm.

C. ERPG-1: Not applicable

An ERPG-1 is not applicable because one would not expect to experience effects or definitely perceive the odor at levels below the ERPG-2. Some individuals are unable to perceive the odor of HCN, and it has been estimated that less than 50% of distracted individuals would perceive the odor at 10 ppm. (Amoore and Hautala 1983)(Eyer 1999).

VII. History of ERPGs

First Published in 1994: ERPG-1 = NA; ERPG-2 = 10 ppm; ERPG-3 = 25 ppm.
Updated and republished in 2006 and 2022- ERPG values unchanged.

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FAST FACTS



Emergency Response Planning Guidelines (ERPG) Save Lives

ERPG values are developed for chemicals with a high potential for uncontrolled releases or pose hazards due to their volatility or toxicity. The values assist occupational and environmental health and safety professionals in the development of emergency response strategies for protecting workers and the public and emergency response personnel in planning for catastrophic chemical releases to a community.



ERPG Stakeholders

- Community emergency planners
- Emergency responders
- Air dispersion modelers
- Industrial process safety engineers
- Community Action Emergency Response (CAER) participants
- Local Emergency Planning Committees (LEPCs)
- State Emergency Response Commissions (SERCs)
- Toxicologists
- Transportation safety engineers
- Fire protection specialists
- Government agencies
- Risk assessors and risk managers
- Resource Conservation and Recovery Act (RCRA) managers
- Product Stewards
- Industrial hygienists

Fields of Focus

- Toxicology
- Emergency Medicine
- Industrial Hygiene



ERPGs Details

- 10 to 15 updated values annually
- 1 to 3 new values annually
- 150 chemicals with assigned ERPGs
- ERPGs are referenced in 10's of millions of copies of the DOT ERG book

Reasons Companies Use ERPGs

- Process risk or safety management
- Community right-to-know and awareness campaigns

A recent survey of chemical manufacturers who produce products for which ERPG values exist found

- **62%** say their company produces at least 1 of the 150 chemicals listed in the Emergency Response Planning Guide
- **72%** say their company uses ERPGs in emergency response to spills or releases of chemicals the company produces
- **96%** say AIHA's ERPGs are valuable to their company
- **100%** say it is important to update or increase the development of new ERPGs*
- **59%** say AIHA should be leading the efforts to update or increase the development of new ERPGs.

**Findings supported in a DC Policy Influencer survey conducted in December 2021*

Did you know...

- No other organization produces these values for community exposure levels...not even the Department of Transportation nor the EPA?
- Industry does not have a platform to establish their own ERPs and that's why they refer to AIHA's?
- First responders refer to AIHA's ERPGs to determine if they should evacuate, shelter in place, etc.?
- Disaster response personnel refer to AIHA's ERPGs to determine standoff distances?

The Emergency Response Planning Committee develops guidelines for responding to potential releases of airborne substances for use in community emergency planning.

Learn mores at aiha.org/erpgs

Contributions supporting the ERPGs are tax-deductible.