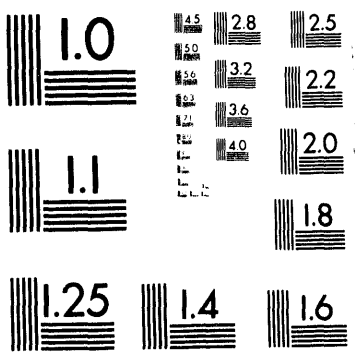
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Toxicity Evaluation and Hazard Review

Cold Smoke

Melecita M. Archuleta, William E. Stocum

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TOXICITY EVALUATION AND HAZARD REVIEW

COLD SMOKE

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Abstract

Cold Smoke is a dense white smoke produced by the reaction of titanium tetrachloride and aqueous ammonia aerosols. Early studies on the toxicity of this nonpyrotechnically generated smoke indicated that the smoke itself is essentially non-toxic (i.e. exhibits no systemic toxicity or organ damage due to exposure) under normal deployment conditions. The purpose of this evaluation was to review and summarize the recent literature data available on the toxicity of Cold Smoke, its chemical constituents, and its starting materials. An extensive literature search on this material and its chemical constituents was evaluated for the information contained in this report. The results of this evaluation indicate that the final reaction products of Cold Smoke (i.e. titanium dioxide and ammonium chloride) under full deployment conditions, have a low order of systemic toxicity. The Cold Smoke generators however, have been designed to release excess ammonia (NH_3), to reduce the possibility of hydrogen chloride (HCl) production from the hydrolysis of $TiCl_4$. Therefore, along with smoke particles, a normally functioning generator will also produce significant NH_3 vapor resulting in severe irritation to the tissues of the mucous membranes and upper respiratory tract. In addition, the reactant materials, titanium tetrachloride and ammonium hydroxide, are extremely corrosive materials especially under conditions of high relative humidity or upon exposure to water in any form including perspiration and tears. A potential hazard exists in using this material in an abnormal or partial deployment in which one of the chemical reactants is deployed alone or in extreme concentrations.

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Nomenclature

ACGIH	American Conference of Governmental Industrial Hygienists
CEIL	Ceiling, the concentration that should not be exceeded during any part of the working exposure.
HSDB	Hazardous Substances Data Bank
IARC	International Agency for the Research of Cancer
LCLo	Lethal concentration - Low = Lowest concentration reported to cause death in the population studied.
LC50	Lethal concentration - Fifty = Concentration that leads to death in 50% of the population studied.
LD50	Lethal dose - Fifty = Dose that leads to death in 50% of the population studied.
NA	information not available
NCI	National Cancer Institute
NCITR	National Cancer Institute Toxicology Report
NIOSH	National Institute of Occupational Safety and Health
NTP	National Toxicology Program
OSHA	Occupational Safety and Health Administration
PEL	permissible exposure limit
RTECS	Registry of Toxic Effects of Chemical Substances
STEL	short term exposure limit
TLV	threshold limit value
TWA	time weighted average (8 hour)

g	grams
hr.	hour
kg	kilograms
m^3	cubic meters
mg	milligrams
min.	minutes
ml	millimeters
ppm	parts per million

TOXICOLOGY EVALUATION AND HAZARD REVIEW
for
COLD SMOKE

Introduction

Cold Smoke composition following a normal deployment is considered to be a mixture of hydrated titanium dioxide ($TiO_2 \bullet 2H_2O$), intermediate hydroxychlorides, and ammonium chloride (NH_4Cl). The purpose of this study was to review and evaluate the recent literature on the toxicity of this non-pyrotechnic smoke. This smoke, which will restrict vision to several inches, is dispensed from individual vessels containing titanium tetrachloride ($TiCl_4$) , ammonium hydroxide (NH_4OH) and nitrogen gas (N_2). Upon initiation of the system, nitrogen pressure is applied to the vessels resulting in the simultaneous release of the chemicals. The reaction to form Cold Smoke therefore, occurs in a zone outside of the individual vessels. The dense white smoke (i.e. Cold Smoke) that is produced in this zone consists of titanium dioxide and ammonium chloride (1). Early studies on the white powder smoke have indicated that it is not hazardous for short exposure times (2,3), although the constituent ingredients, titanium tetrachloride and ammonium hydroxide and their hydrolysis products, are known toxic and corrosive materials (4,5).

This study therefore, has concentrated on evaluating the toxicity of the individual components of Cold Smoke. This report summarizes the current chemical and toxicological literature on Cold Smoke (i.e. titanium dioxide and ammonium chloride) and its starting materials (i.e. titanium tetrachloride and ammonium hydroxide) as well as any significant hydrolysis products; and reports on the health hazards associated with various exposure scenarios. Chemical and physical properties and exposure limits are also given when available.

Background

Cold Smoke is a non-pyrotechnic chemical smoke or powder that offers deterrent capability by seriously inhibiting an individuals vision. The production of Cold Smoke occurs by the reaction of liquid titanium tetrachloride with aqueous ammonia and/or atmospheric moisture (1).

$$TiCl_4 + 4NH_4OH \rightarrow TiO_2 \bullet 2H_2O + 4NH_4Cl$$

Several intermediate and final reaction products have been postulated to occur during the generation of Cold Smoke and are listed in Tables I and II taken from Smith et al., (1980). Furthermore, Cold Smoke generators have been designed to release excess ammonia, (NH_3) to reduce the possibility of hydrogen chloride (HCl) production from the hydrolysis of $TiCl_4$. Thus, along with smoke particles, a normally functioning generator will also produce significant NH_3 vapor resulting in toxic irritant effects due to the ammonia. In addition, inhalation exposures to high concentrations of ammonium chloride may cause HCl to form within the respiratory tract. The specific hazards therefore, associated with Cold Smoke following a normal deployment involves NH_3 vapors and NH_4Cl particulates. Exposure to $TiCl_4$ and NH_4OH must be also be considered in a situation of partial deployment of one of the reaction components without the full reaction to form Cold Smoke or exposure of an individual in the reaction zone (3).

TABLE I

Chemical Constituents of Cold Smoke
Reaction of $TiCl_4$ and NH_3

Reactants	Intermediate Compounds	Final Products
$TiCl_4$	Titanium tetramminotetrachloride ($TiCl_4 \cdot 4NH_3$)	Hydrated titanium dioxide ($TiO_2 \cdot 2H_2O$)
and	Titanium hexamminotetrachloride ($TiCl_4 \cdot 6NH_3$)	Ammonium Chloride (NH_4Cl)
NH_3		Ammonia (NH_3)

TABLE II

Chemical constituents of Cold Smoke
Hydrolysis of $TiCl_4$

Reactants	Intermediate Compounds	Final Products
$TiCl_4$	Pentahydrated titanium tetrachloride ($TiCl_4 \cdot 5H_20$)	Hydrated titanium dioxide ($TiO_2 \cdot 2H_2O$)
and	Titanium hydroxytrichloride ($Ti(OH)Cl_3$)	and
H_2O	Titanium dihydroxydichloride ($Ti(OH)_2Cl_2$)	Hydrogen Chloride (HCl)
	Titanium trihydroxychloride ($Ti(OH)_3Cl$)	
	Titanic oxydichloride ($TiOCl_2$)	

1. TITANIUM TETRACHLORIDE

Molecular Formula

$TiCl_4$

CAS Number:

7550-45-0

Chemical and Physical Properties (6):

Melting Point: -25°C (-13°F)
Boiling Point: 132°C (277.5°F)
Flash Point: None
Appearance: Clear fuming liquid
Odor: Acidic odor
Solubility in Water: Hydrolyzes
Log Octanol/water partition coefficient: NA

Exposure limits (7):

Occupational Exposure Limits for titanium tetrachloride have not been determined.

Toxicology: Titanium tetrachloride is a colorless, fuming, liquid that is decomposed by moisture to hydrochloric acid, titanium dioxide and heat (4). Inhalation of $TiCl_4$ fumes appears to be more toxic than the inhalation of HCl because $TiCl_4$ fumes can penetrate into the deep lung where further hydrolysis to HCl can occur. Pure HCl cannot penetrate to this level since it is highly soluble and largely dissolves in the moisture of the upper airways (8). Titanium tetrachloride reacts extremely vigorously with water in any form including perspiration on the skin, moisture in the air, or tears, liberating large quantities of heat. It is postulated that the initial thermal and chemical burns expose the deeper tissues to the effects of the hydrochloric acid released (4).
Rat-inhalation LC50 = 460 mg/m^3 , 4 hour (9,10)
Mouse-inhalation LC50 = 100 mg/m^3, 2 hour (9,10)

Acute Exposure: Titanium tetrachloride is considered harmful if ingested, inhaled, or absorbed through the skin. This material is extremely destructive to tissues of the mucous membranes and upper respiratory tract, eyes, and skin due to the extreme exothermic reaction and hydrolysis to HCl in water (4,11). The acute toxicity of titanium tetrachloride appears to be a function of large changes in humidity with the most severe toxicity occurring in the 60-100% relative humidity region (12).

Chronic Exposure: A two year, low level, (0.1 to 10 mg/m^3 for 6 hr/day, 5 days/week) chronic, inhalation study on rats exposed to titanium tetrachloride and its hydrolysis products revealed no abnormal clinical signs, body weight changes, or excess mortality in any exposed group (13). The pulmonary response at the 1.0

mg/m^3 was typical of that seen for a nuisance dust. In comparison, irritating effects of TiO_2 as a nuisance particulate occurs at 10 mg/m^3 (14). At exposures of 10 mg/m^3 $TiCl_4$ the pulmonary response in the rat suggests that long term chronic exposure may result in upper respiratory tract irritation and possibly acute or chronic bronchitis (8,13).

Eye Exposure: Severe corneal damage has been seen with exposure to the fumes of titanium tetrachloride and even brief direct contact with the liquid material can cause severe eye injury(9,16).

Inhalation Exposure: The vapors are very irritating to the respiratory tract. Cough, bronchoconstriction with wheezing, chemical pneumonitis, or pulmonary edema may occur following inhalation of the fumes(4). $TiCl_4$ is considered to be more toxic than HCl following inhalation exposure due to its ability to penetrate into the deep lung where insitu hydrolysis to HCl can result in further damage (8, 9). However, intermittent exposure to low concentrations (0.1 to 1 mg/m^3) of titanium tetrachloride did not result in progressive or cumulative changes in the lungs of workers (17).

Oral Exposure: Ingestion of this material can cause mouth, throat, esophageal, and gastrointestinal tract irritation or burns. Nausea, vomiting, diarrhea, and abdominal pain with bleeding and / or perforation may be seen due to its corrosive effects with water (9, 10).

Dermal Exposure: Direct skin contact can cause irritation or corrosive chemical and exothermic dermal burns, especially if moisture is present (4, 6).

Carcinogenicity: No mutagenic activity was seen in studies with titanium tetrachloride (18,19). Epidemiological studies of workers exposed to titanium tetrachloride found no statistically significant increased risk associated with titanium tetrachloride exposure and the development of lung cancer or other fatal respiratory diseases. Furthermore, no cases of pulmonary fibrosis were observed among these employees (20). Titanium tetrachloride is not listed as a carcinogen by NTP, IARC or ACGIH, nor is it regulated as a carcinogen by OSHA (9,10).

Reproductive Effects: There is no evidence of reproductive or teratogenic effects due to exposure to $TiCl_4$ (9,10).

Incompatibility: Titanium tetrachloride reacts strongly with water to release hydrogen chloride and heat (5).

Hazardous Decomposition Products: - Hydrolysis of titanium tetrachloride results in the production of toxic hydrogen chloride (6).

2. AMMONIA

Molecular Formula
NH_3

CAS Number:
7664-41-7

Chemical and Physical Properties (21):
Melting Point: -77.7°C
Boiling Point: -33.4°C
Flash Point: N/A
Appearance: Colorless gas
Odor: Characteristic odor detectable at 1 - 5 ppm
Solubility in Water: 51 g/100g water at 20°C.
Log Octanol/water partition coefficient: NA

Exposure limits (7):

ACGIH TLV:	TWA - 17 mg/m^3 (25 ppm)
	STEL - 24 mg/m^3 (35 ppm)
NIOSH REL:	TWA - 18 mg/m^3 (25 ppm)
	STEL - 27 mg/m^3 (35 ppm)
OSHA PEL:	TWA - 35 mg/m^3 (50 ppm) Transitional limit.
	STEL - 27 mg/m^3 (35 ppm) Final Rule limit.

Toxicology: The configuration of the Cold Smoke generator intentionally results in excess production of ammonia vapors. Ammonia, is a primary irritant that in an aqueous environment exists in equilibrium between ionized ammonium cation (NH_4^+) and the non-ionized ammonia (NH_3). Therefore, toxicity of this compound is examined in terms of both forms. Ammonia affects the eyes and upper respiratory tract. Chronic effects of a short term NH_3 exposure are minimal. Pathophysiological responses to NH_3 can range from odor repugnance to severe skin burns. Because of its low threshold of odor irritation (i.e., 0.7 - 3.5 mg/m^3), ammonia is treated as a material with good warning properties although individuals working with ammonia can become accustomed to levels as high as 14 mg/m^3 without serious complaints (22). Acute inhalation of 3500 mg/m^3 (5000 ppm) have occurred however, and have been fatal in humans (9, 10).
Rat-inhalation LC50 = 1400 mg/m^3 (2000 ppm)/4 hr. (9, 10)
Mouse-inhalation LC50 = 2950 mg/m^3 (4230 ppm)/1 hr. (9, 10)

Acute Exposure: Ammonia is a severe irritant of the eyes, respiratory tract, and skin. It may cause burning and tearing of the eyes, runny nose, coughing, chest pain, cessation of respiration, and death (23).

Chronic Exposure: Repeated or prolonged exposure to ammonia gas may cause chronic irritation of the eyes and upper respiratory tract. Repeated worker exposure to ammonia gas resulted in complaints of chronic cough and evidence of pulmonary damage (24).

Eye Exposure: Ammonia vapor is a severe irritant of the eyes, especially the cornea and has a greater tendency than other alkalis to penetrate and damage the iris, and to cause cataracts (15). The toxicity associated with eye exposure to ammonia can be affected by buffers, pH, temperature, and salinity. However, exposure of the eyes to high gas concentrations as in an accident has been shown to produce temporary blindness and severe eye damage with total corneal epithelial loss (10, 25).

Inhalation Exposure: Ammonia is a severe irritant to the upper respiratory tract and may cause breathing difficulties which may be delayed in onset (26). Inhalation of concentrations of 1750 to 4530 mg/m^3 (2500 to 6500 ppm) results in difficulty breathing, bronchospasm, chest pain and pulmonary edema which may be fatal (27,28). Consequences of chronic exposure to ammonia can include bronchitis or pneumonia and some residual reduction in pulmonary function has been reported. Therefore, individuals with impaired pulmonary function may be at increased risk from exposure.

Oral Exposure: Ingestion of ammonia can result in nausea, vomiting, and swelling of the lips, mouth, and larynx. Oral and esophageal burns may also occur when concentrated ammonia is ingested (9, 10).

Dermal Exposure: Ammonia produces tissue injury similar to corrosive alkali. The severity of the injury depends upon the concentration and duration of exposure. Concentrated ammonia produces liquefaction necrosis and deep penetrating burns. Household ammonia (5 to 10% ammonia) rarely causes burns but is irritating to the eyes, nose, throat, and tracheobronchial tree (9,10).

Carcinogenicity: Ammonia is not listed as a carcinogen by NTP, IARC or ACGIH, nor it is regulated as a carcinogen by OSHA (9,10).

Reproductive Effects: There is no evidence of teratogenic or reproductive effects due to exposure to ammonia (9,10).

Incompatibilities: Reaction of Ammonia with strong oxidizers may result in fires and explosions. Contact with calcium, hypochlorite bleaches, gold, mercury, and silver may form highly explosive products. And contact with halogens can result in violent spattering (21).

Hazardous Decomposition Products: None (21)

3. AMMONIUM HYDROXIDE

Molecular Formula:
NH_4OH

CAS Number:
1336-21-6

Chemical and Physical Properties (29):
Melting Point: -77.7°C (-107°F)
Boiling Point: NA
Flash Point: Nonflammable
Appearance: Colorless liquid
Odor: Characteristic odor detectable at 50 ppm
Solubility in Water: Complete
Log Octanol/water partition coefficient: NA

Exposure limits[a] (7):

ACGIH TLV:	TWA - 25 ppm (17 mg/m^3)
	STEL - 35 ppm (24 mg/m^3)
NIOSH REL:	TWA - 25 ppm (18 mg/m^3)
	STEL - 35 ppm (27 mg/m^3)
OSHA PEL:	TWA - 50 ppm (35 mg/m^3)
	STEL - 35 ppm (27 mg/m^3)

[a] Exposure limits for ammonia (gas).

Toxicology: Ammonium hydroxide produces tissue injury similar to corrosive alkali. The severity of injury depends upon the concentration and duration of exposure. The extent of injury ranges from mild erythemia (inflammatory redness) and edema (swelling) to severe full thickness burns and life-threatening pulmonary edema. Household ammonia (5 to 10% ammonia) rarely causes burns but is irritating to the eyes, nose, throat, and tracheobronchial tree (22).
Oral - human LDLo = 43 mg/kg (29)
Oral - rat LD50 = 350 mg/kg (29)

Acute Exposure: Ammonium hydroxide is harmful if swallowed, inhaled or absorbed through the skin. This material is extremely destructive to the tissues of the mucous membranes and upper respiratory tract, eyes and skin (23).

Chronic Exposure: Prolonged or repeated exposure to low concentrations of ammonium hydroxide (< 14 mg/m^3, 20 ppm) can result in tolerance to this material. (22). Symptoms of over exposure may include burning sensation, coughing, wheezing, laryngitis, shortness of breath, headache, nausea and vomiting (24).

Eye Exposure: Exposure to concentrated ammonia vapors results in mucosal burns of the eyes, nose, pharynx, and larynx. Acute eye exposure results in conjunctivitis, lacrimation, irritation and temporary or permanent blindness (10, 15).

Inhalation Exposure: Exposure to concentrated ammonium hydroxide results in bronchospasm, inflammation of the mucous membranes of the larynx and trachea, wheezing, shortness of breath, and chest pain (26).

Oral Exposure: Ingestion of concentrated ammonia solutions results in oral and esophageal burns, swelling of the lips, mouth and larynx, along with nausea and vomiting (9, 10).

Dermal Exposure: Concentrated ammonium hydroxide produces liquefaction necrosis and deep penetrating burns (9, 10).

Carcinogenicity: Ammonium hydroxide is not listed as a carcinogen by NTP, IARC or ACGIH, nor it is regulated as a carcinogen by OSHA (9,10).

Reproductive Effects: There is no evidence of teratogenic or reproductive effects due to exposure to ammonium hydroxide (9,10).

Incompatibilities: Ammonium hydroxide is incompatible with strong oxidizers, copper, copper alloys, galvanized iron and aluminum (29).

Hazardous Decomposition Products: May release corrosive vapors of ammonia and toxic oxides of nitrogen (29).

4. TITANIUM DIOXIDE

Molecular Formula:
TiO_2

CAS Number:
13463-67-7

Chemical and Physical Properties (30):
Melting Point: NA
Boiling point: NA
Flash Point: Will not burn
Appearance: White odorless powder
Odor: No Odor
Solubility in Water: insoluble
Log Octanol/water partition coefficient: NA

Exposure limits (7):

OSHA-PEL:	TWA - 5 mg/m^3 (respirable dust[a])
	TWA - 10 mg/m^3 (total dust)
ACGIH TLV:	TWA - 10 mg/m^3 (total dust)

[a] TiO_2 is regulated as a nuisance dust.

Toxicology: There has been little evidence to suggest that exposure to titanium, or titanium oxides result in any significant toxicity to laboratory animals or man (9,10). Titanium dioxide (TiO_2) is essentially nontoxic following an acute exposure (31). TiO_2 is regulated under OSHA as a nuisance dust at exposures of 10 mg/m^3 (14).

Acute Exposure: Titanium dioxide is considered to be non-toxic following an acute exposure with an approximate oral lethal dose in rats of greater than 25,000 mg/kg (31). Acute and subacute toxicity studies have not shown any detrimental effects of titanium dioxide in the lungs (22,26,32).

Chronic Exposure: A two year inhalation study on rats exposed to titanium dioxide indicated no significant abnormal effects due to chronic exposure (14). Furthermore, autopsy studies on workers exposed to titanium dioxide and epidemiological surveys made on working populations exposed to titanium dioxide dusts have not shown any evidence of fibrogenic activity or toxic effects (33).

Eye Exposure: This material in the eye is not known to cause a toxic reaction. However, as a nuisance particulate it may cause irritation as do other non-toxic dusts and particulates (15).

Inhalation Exposure: Most of the available studies suggest that inhaled titanium dioxide is biologically inert (26,34,35). However, titanium dioxide can behave as a mild pulmonary irritant and inhalation of extremely high concentrations of this or any dust can overwhelm the lung clearance mechanisms (36,37). Furthermore, the breathing of titanium dioxide in persons with impaired pulmonary function, as in obstructive airway disease, may cause exacerbation of symptoms due to its irritant properties(22).

Oral Exposure: Titanium compounds are generally considered to be poorly absorbed through the gastrointestinal tract upon ingestion. Therefore, toxicity by the oral route is not expected (26).

Dermal Exposure: There is no evidence that dermal exposure results in any adverse effects (22,38). However as with all particulate material proper chemical protective clothing and proper hygiene should be maintained.

Carcinogenicity: Animal studies with titanium dioxide have produced no evidence to suggest an association between titanium dioxide exposure and risk of lung cancer (39). Epidemiological studies of workers exposed to titanium dioxide between 1935 and 1983 found no statistically significant association between titanium dioxide exposure and risk of lung cancer, chronic respiratory disease, and chest abnormalities. Furthermore, no cases of pulmonary fibrosis were observed (40).

Reproductive Effects: There is no evidence of teratogenic or reproductive effects due to exposure to titanium dioxide (9, 10, 22).

Incompatibility: Titanium dioxide is incompatible with strong acids (9, 10).

Hazardous Decomposition Products: None (9,10)

5. AMMONIUM CHLORIDE

Molecular Formula
NH_4Cl

CAS Number:
12125-02-9

Chemical and Physical Properties (41):
Melting Point: 338°C
Boiling Point: 520°C
Flash Point: NA
Appearance: White Powder
Odor: Odorless
Solubility in Water: 29.7 g/100g water @ 0°C.
Log octanol/water partitions coefficient: NA

Exposure limits (7):

ACGIH TLV:	TWA - 10 mg/m^3 (fume)
	STEL - 20 mg/m^3 (fume)
OSHA PEL:	TWA - 10 mg/m^3 (fume)
	STEL - 20 mg/m^3 (fume)

Toxicology: Oral or inhalation exposure to large doses of ammonium chloride may cause nausea, vomiting, thirst, headache, hyperventilation, and progressive drowsiness (42). In addition, inhalation exposures to high concentrations of NH_4Cl may also result in HCl being produced in the respiratory tract, causing toxic effects due to this material.

Rat-oral LD50 = 1650 mg/kg (9, 10)
Mouse-oral LD50 = 1300 mg/kg (9, 10)

Acute Exposure: Ammonium chloride may be harmful if swallowed, inhaled or absorbed through the skin High blood chlorine and low blood potassium resulting in metabolic acidosis (decreased pH and bicarbonate concentration) is the most common manifestation of toxicity with this chemical (10).

Chronic Exposure: Prolonged or repeated exposure to ammonium chloride fumes can result in drowsiness, headache, and mental confusion progressing to coma (22,23).

Eye Exposure: Eye contact with ammonium chloride may cause eye redness and pain. Prolonged exposure to this chemical may cause damage to the retina of the eye (15).

Inhalation Exposure: Ammonium chloride dispersed as a dust may cause irritation of the nose and throat. Hyperventilation seen with over exposure to ammonium chloride reflects respiratory compensation for metabolic acidosis and may not be present for several hours. Thermal decomposition also forms toxic gases such as ammonia or hydrogen chloride which may irritate the respiratory tract (9,10).

Oral Exposure: Following oral exposure, ammonium chloride is rapidly absorbed from the gastrointestinal tract (42). Large doses may cause nausea, vomiting and acidosis (9).

Dermal Exposure: Contact with ammonium chloride may cause skin irritation (9, 10).

Carcinogenicity: Ammonium chloride is not listed as a carcinogen by NTP, IARC or ACGIH, nor is it regulated as a carcinogen by OSHA (9,10).

Reproductive Effects: There is no evidence of teratogenic or reproductive effects following exposure to ammonium chloride (9,10).

Incompatibilities: Ammonium chloride is incompatible with strong acids and/or bases, silver salts, potassium chlorate, ammonium nitrate, bromine trifluoride and iodine heptafluoride (41).

Hazardous Decomposition Products: Ammonium chloride decomposes or hydrolyzes to form toxic gases of hydrogen chloride and ammonia (41).

6. HYDROGEN CHLORIDE

Molecular Formula
HCl

CAS Number:
7647-01-0

Chemical and Physical Properties (43):
Melting Point: NA
Boiling Point: -85°C (-121°F)
Flash Point: NA
Appearance: colorless gas
Odor: irritating pungent odor
Solubility in Water: Complete
Log Octanol/water partition coefficient: NA

Exposure limits (7):
ACGIH TLV: CEIL - 7.5 mg/m^3 (5 ppm)
OSHA PEL: CEIL - 7 mg/m^3 (5 ppm)
NIOSH REL: CEIL - 7 mg/m^3 (5 ppm)

Toxicology: Hydrogen chloride gas can be corrosive to the skin, eyes, nose, mucous membranes, respiratory tract, and gastrointestinal tract at exposures at or above 7 mg/m^3 (5 ppm). In most animal lethality studies, death has been attributed to respiratory injury such as pulmonary edema, emphysema, and collapsed lung, with secondary injury resulting in passive congestion of the liver, intestine, and kidneys (44). Minimum lethal exposures in humans ranged from 4480 mg/m^3 (3000 ppm) for 5 minutes to 1940 mg/m^3 (1300 ppm) for 30 minutes (9).
Rat-inhalation LC50 = 4660 mg/m^3, 3124 ppm (45).
Mice-inhalation LC50 = 1650 mg/m^3, 1108 ppm (46).
Rabbit-inhalation LCLo = 6590 mg/m^3, 4416 ppm (46).
Guinea pig-inhalation LCLo = 6590 mg/m^3, 4416 ppm (46).

Acute Exposure: Hydrogen chloride gas is generally accepted as a local irritant at low concentrations of exposure for short duration (47). Acute inhalation of gas for fumes at levels of 7 - 50 mg/m^3 (5-35 ppm) may cause irritation and burning of the throat, coughing, and choking. Exposures of 75 mg/m^3 (50 ppm) may be barely tolerable for 1 hour and a concentration of 150 mg/m^3 (100 ppm) is considered immediately dangerous to life or health (48).

Chronic Exposure: Repeated or prolonged exposure may be associated with changes in pulmonary function, chronic bronchitis, dermatitis, erosion of dental enamel, conjunctivitis, and overt upper respiratory tract abnormalities (49). No significant systemic effects have been seen from chronic exposure to low levels of gaseous hydrogen chloride (9).

Eye Exposure: Exposure of the eyes to hydrogen chloride gas may cause severe irritation, conjunctivitis, corneal necrosis and burns with impairment or permanent loss of vision (15).

Inhalation Exposure: Inhalation of hydrogen chloride at concentrations of 7 - 50 mg/m^3 (5-35 ppm) may cause cough and choking with inflammation and ulceration of the respiratory tract. Hydrogen chloride in the lung can cause delayed pulmonary edema (50).

Oral Exposure: Ingestion of hydrogen chloride results in corrosion of the mucous membranes, esophagus, and stomach with nausea, vomiting, intense thirst and diarrhea (23).

Dermal Exposure: Prolonged exposure to hydrogen chloride vapors may cause severe irritation, inflammation, ulceration, necrosis and chemical burns. Shock symptoms may develop and photo sensitization reactions may occur in persons previously exposed (26,48).

Carcinogenicity: There is no data implicating hydrogen chloride as a carcinogen. Hydrogen chloride is not defined as a carcinogen by NTP, IARC or ACGIH, nor is it regulated as a carcinogen by OSHA (9,10).

Reproductive Effects: A report cited by RTECS reported results of fetotoxicity and specific developmental abnormalities in rats exposed to 450 mg/m^3 hydrogen chloride for 1 hour on day 1 of gestation. No other data was found implicating hydrogen chloride as a reproductive toxin (9,10).

Incompatibility: Contact with common metals produces hydrogen which may form explosive mixtures with air. Hydrogen chloride reacts exothermically with water or steam to produce toxic and corrosive fumes (43).

Hazardous Decomposition Products: None (43).

7. NITROGEN GAS

Molecular Formula

N_2

CAS Number:

7727-37-9

Chemical and Physical Properties (51):

Melting Point: NA
Boiling Point: -196°C (-321°F)
Flash Point: NA
Appearance: colorless gas
Odor: odor
Solubility in Water: 1.6 x @ 20°C
Log Octanol/water partition coefficient: NA

Exposure limits (7):

No occupational exposure limits have been established by OSHA, ACGIH or NIOSH.

Toxicology: Nitrogen gas is considered a simple asphyxiant capable of displacing oxygen. In sudden acute exposures, asphyxia with unconsciousness may be immediate. With slow development there may be rapid respiration and pulse, dizziness, reduced awareness, tingling sensations, incoordination, faulty judgment, emotional instability, and rapid fatigue. As the asphyxia progresses, nausea, vomiting, collapse, unconsciousness, convulsions, deep coma and death are possible.

Acute Exposure: Nitrogen gas is considered a simple asphyxiant in acute exposures to high concentrations capable of displacing oxygen. In addition, nitrogen under increased atmospheric pressure, (> 1.5 atmospheres) may dissolve in the fat-containing brain cells and act as an anesthetic.

Chronic Exposure: No adverse effects have been reported following low dose repeated and prolonged exposures to nitrogen gas.

Eye Exposure: Nitrogen gas sprayed directly into the eyes may cause irritation. Liquid nitrogen can cause frostbite with redness, pain, and blurred vision.

Inhalation Exposure: Inhalation exposure to nitrogen gas results in asphyxiation due to oxygen deficiency. The symptoms of asphyxia depend on the rapidity with which the oxygen deficiency develops and how long it continues.

Oral Exposure: Ingestion of nitrogen gas is unlikely. Ingestion of the liquid however, can result in frostbite damage to the lips, mouth, and mucous membranes.

Dermal Exposure: No adverse effects have been reported following dermal exposure to nitrogen gas. Dermal exposure to liquid nitrogen however, can result in frostbite with redness, tingling, and pain or numbness.

Carcinogenicity: Nitrogen is not listed as a carcinogen by NTP, IRAC, or ACGIH, nor is it regulated as a carcinogen by OSHA (9,10).

Reproductive Effects: There is no evidence of reproductive effects associated with exposure to nitrogen gas. However, asphyxiation during pregnancy may adversely affect the fetus (9,10).

Incompatibility: Lithium and titanium may ignite in a nitrogen atmosphere. Furthermore, mixtures of nitrogen gas with ozone may be explosive.

Hazardous Decomposition Products: Thermal decomposition may produce toxic oxides of nitrogen (9,10).

Conclusions

Cold Smoke is a nonpyrotechnic smoke that is essentially biologically inert under proper deployment conditions. It is formed by the reaction of two very toxic and corrosive materials, titanium tetrachloride ($TiCl_4$) and aqueous ammonia (NH_4OH), to form a non-toxic particulate smoke made up of titanium dioxide (TiO_2) and ammonium chloride (NH_4Cl). The hazard associated with this smoke is due to the irritating properties of the excess ammonia that is present in the system or to a partial or non-normal deployment of the smoke in which one or more of the reaction products is either deployed alone or in concentrations that are not optimal for normal smoke development. The reaction products of Cold Smoke, TiO_2 and NH_4Cl, are essentially nontoxic while the reactants, $TiCl_4$ and NH_4OH, are extremely hazardous especially under conditions of high humidity and / or in the presence of moisture such as perspiration or tears. $TiCl_4$ is considered corrosive to tissues and mucous membranes and the toxicity of this chemical appears to be due to the exothermic hydrolysis reaction with moisture and the subsequent release of hydrochloric acid resulting in chemical burns. Ammonium hydroxide is a severe irritant of the eyes, respiratory tract, and skin, producing tissue injury similar to corrosive alkali. In addition, excess ammonia present to ensure the complete reaction of $TiCl_4$ is a strong irritant of the mucous membranes and upper respiratory tract and is responsible for the irritating properties of the smoke. Nitrogen gas used to pressurize the system is a simple asphyxiant and could create an O_2 deficient atmosphere in the event of a sudden rupture of the gas pressurizing system.

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