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CHEMICAL SUMMARY FOR CHLORINE

prepared by
OFFICE OF POLLUTION PREVENTION AND TOXICS
U.S. ENVIRONMENTAL PROTECTION AGENCY
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This summary is based on information retrieved from a systematic search limited to secondary sources (see Appendix A). These sources include online databases, unpublished EPA information, government publications, review documents, and standard reference materials. No attempt has been made to verify information in these databases and secondary sources.

I. CHEMICAL IDENTITY AND PHYSICAL/CHEMICAL PROPERTIES

The chemical identity and physical/chemical properties of chlorine are summarized in Table 1.

TABLE 1. CHEMICAL IDENTITY AND CHEMICAL/PHYSICAL PROPERTIES OF CHLORINE

Characteristic/Property	Data	Reference
CAS No.	7782-50-5	
Common Synonyms	none	
Molecular Formula	Cl2	
Chemical Structure	Cl-Cl	
Physical State	greenish yellow diatomic gas	Budavari et al. 1989
Molecular Weight	35.453	Budavari et al. 1989
Melting Point	-101.00øC	Budavari et al. 1989
Boiling Point	-34.05øC at 760 mm Hg	Budavari et al. 1989
Water Solubility (see note)	7.3 g/L @ 20øC	
	14.6 g/L @ g/L	U.S. EPA 1989
Density	1.468 mg/L @ 0øC/ 3.65 atm	U.S. EPA 1989
Vapor Density	3.209-3.214 mg/L (dry gas) @ 0øC/1 atm	U.S. EPA 1989
KOC	no data were found	
Log KOW	no data were found	
Vapor Pressure	5.85 x 103 mm Hg @ 25øC	CHEMFATE 1994
Reactivity	highly reactive; forms explosive mixtures with flammable gases and vapors	HSDB 1994
Flash Point	nonflammable	HSDB 1994
Henry's Law Constant	no data were found	
Fish Bioconcentration Factor	no data were found	
Odor Threshold	0.0020 mg/L (water), 0.31 ppm (air)	HSDB 1994
Conversion Factors	1 ppm = 2.9 mg/m3	
	1 mg/m3= 0.34 ppm	U.S. EPA 1989

Note: When chlorine is added to fresh water, the solution will usually contain two forms of free chlorine: hypochlorous acid (HOCl) and the hypochlorite ion (OCl) (U.S. EPA 1985). Other chlorinated compounds may be present, depending on other materials present in the water. The

term "total residual chlorine" is used to refer to the sum of free chlorine and combined chlorine in fresh water.

II. PRODUCTION, USE, AND TRENDS

A. Production

There are 18 producers producing chlorine at 40 facilities in the United States. Table 2 lists producers, plant locations, and plant capacities. Annual US capacity is over 13 million short tons. In 1992, approximately 11.7 million short tons of chlorine were produced in the US. In that same year, 274,000 short tons of chlorine were imported into the US and 34,000 short tons were exported (Mannsville 1993).

B. Use

Chlorine is used in a number of industrial applications. Its largest use is as a raw material in the production of ethylene dichloride, an intermediate for vinyl chloride monomer and polyvinyl chloride (PVC) resins. Chlorine is also used in the paper industry to bleach pulp, in the production of chlorinated solvents and chlorofluorocarbons (CFCs), as a disinfectant or fungicide for a variety of purposes, including water purification, cooling systems, meat, fish, vegetable, and fruit processing, foot baths, dairy equipment, laundries, and dishwater, as well as for shrink-proofing wool, in special batteries (with lithium or zinc), and in the manufacture of propylene oxide and pesticides. Table 3 shows the estimated 1993 end-use pattern for chlorine.

C. Trends

Chlorine use in pulp bleaching and in the production of some chlorinated solvents and CFCs is expected to decline due to environmental concerns. Overall, demand for chlorine is expected to increase approximately 1 percent per year for a few years, followed by a period of moderate decline (Mannsville 1993).

TABLE 2. United States Producers of Chlorine

Company	Plant Location(s)	Plant Capacity (in 1,000s of short tons)
BF Goodrich	Calvert City, KY	115
Dow Chemical	Freeport, TX	2,400
	Plaquemine, LA	1,150
DuPont	Niagara Falls, NY	85
Elf Atochem	Portland, OR	150
	Tacoma, WA	90
Formosa Plastics	Baton Rouge, LA	200
	Point Comfort, TX	625
General Electric	Burkeville, AL	25
	Mt. Vernon, IN	55
Georgia Gulf	Plaquemine, LA	450
Georgia Pacific	Bellingham, WA	90
	Brunswick, GA	30
Hanlin	Acme, NC	53
	Brunswick, GA	101
	Orrington, ME	80

La Roche Chemicals	Gramercy, LA	205
Miles	Baytown, TX	130
Niachlor	Niagara Falls, NY	240
Occidental Chemical	Convent, LA	308
	Corpus Christi, TX	455
	Deer Park, TX	396
	Delaware City, DE	139
	LaPorte, TX	525
	Mobile, AL	42
	Muscle Shoals, AL	145
	Niagara Falls, NY	330
	Tacoma, WA	220
	Taft, LA	645
Olin	Augusta, GA	110
	Charleston, TN	255
	McIntosh, AL	400
Pioneer Chlor-Alkali Co.	Henderson, NV	115
	St. Gabriel, LA	180
PPG Industries	Lake Charles, LA	1,265
	Natrium, WV	380
Vulcan Chemicals	Geismar, LA	249
	Wichita, KS	255
	Port Edwards, WI	73
Weyerhauser	Longview, WA	140

Source: Mannsville 1993.

TABLE 3. Estimated 1993 United States End-Use Pattern of Chlorine

Use of Chlorine (typical Standard Industrial Classification (SIC) Code) (see end note 1)	Percentage of all Chlorine Use
Ethylene dichloride (intermediate for PVC production) (production, SIC 2869; use, 2821)	35%
Pulp and paper (production, SIC 2621)	11%
Propylene oxide (production, SIC 2869; use, various industries)	8%
Chlorinated ethanes (production, SIC 2869; use, various industries)	5%
Chlorinated methanes (production, SIC 2869; use, various industries)	4%
Other organic chemicals (production, SIC 286; use, various industries)	16%
Inorganic chemicals (production, SIC 281; use, various industries)	11%
Water treatment (used in various industries)	5%
Miscellaneous (no applicable SIC Code(s))	5%

Source: Mannsville 1993.

III. ENVIRONMENTAL FATE

A. Environmental Release

In addition to the chlorinated water that is released into the environment from sewage treatment operations and other sources, chlorine is potentially released during its transport; from its

manufacturing/processing facilities, such as the pulp and paper industries and plastic and resin industries; and through the disposal of consumer products containing chlorine, such as automotive fluids, household bleaches, and refrigerants (HSDB 1994; NTP 1992). Limited monitoring studies have detected chlorine in the ambient atmosphere at concentrations ranging from 1 to 3.7 mg/m3 (0.344 to 1.27 ppm) (HSDB 1994).

In 1992, releases of chlorine to environmental media, as reported to the Toxic Chemical Release Inventory by certain types of U.S. industries, totaled about 69.9 million pounds to the atmosphere, 1.2 million pounds to surface water, 48 thousand pounds to underground injection sites, and 46 thousand pounds to land (TRI92 1994).

B. Transport

Vaporization of molecular chlorine (Cl2) from water to the atmosphere may be significant at low pH values and high concentrations (e.g., pH 2 and 3500 mg/L chlorine), but is insignificant at neutral pH and low concentrations (U.S. EPA 1989).

C. Transformation/Persistence

1. Air - Information regarding the environmental fate of chlorine in air was not found in the secondary sources searched.

2. Soil - Chlorine may react with soil components to form chlorides; depending on their water solubility, these chlorides are easily washed out from the soil (Seiler et al. 1988).

3. Water - Chlorine hydrolyzes very rapidly in water (rate constants range from 1.5 x 10-4 at 0øC to 4.0 x 10-4 at 25øC; half-life in natural waters, 0.005 s) (U.S. EPA 1989). In fresh and wastewaters at pH >6, complete hydrolysis occurs with the formation of hypochlorous acid (HOCl) and chloride ion (Cl-). The hypochlorous acid ionizes to hydrogen ion (H+) and hypochlorite ion (OCl-). At pH values >5, OCl- predominates; at pH values <5, HOCl predominates (U.S. EPA 1989). Free chlorine (Cl2, HOCl, and OCl-) reacts rapidly with inorganics such as bromide and more slowly with organic material present in natural waters. These reactions yield chloride, oxidized organics, chloroganics (including trihalomethanes), oxygen, nitrogen, chlorate, bromate and bromoorganics.

4. Biota - There is no potential for the bioaccumulation or bioconcentration of chlorine (HSDB 1994).

D. Chlorine in Drinking Water

Chlorine, added to drinking water as chlorine gas (Cl2) or hypochlorite salts (e.g., NaOCl), effectively inactivates bacteria in 20 minutes at concentrations of 0.03 to 0.06 mg/L at pH range of 7.0 to 8.5 and temperature range of 4øC to 22øC (NTP 1992). Data for over 80 water supplies throughout the United States indicate that the facilities applied chlorine at ranges of 1 to 29.7 mg/L and residual levels ranged from 0.2 to 6 mg/L (HSDB 1994).

V. HEALTH EFFECTS

A. Pharmacokinetics

1. Absorption - Following oral administration of (HO)36Cl to rats, 36Cl was rapidly absorbed into the blood where concentrations

peaked in 2 hours for fasted animals and in 4 hours for nonfasted animals (U.S. EPA 1989).

2. Distribution - Seventy-two hours after oral administration of 3.26 mg/kg (HO)36Cl to male Sprague-Dawley rats, the concentration of (HO)36Cl was highest in the plasma (0.77% of initial dose), followed by bone marrow, kidney, testes, lung, skin, duodenum, spleen, liver and carcass, and was lowest in the ileum (U.S. EPA 1989). A similar pattern of distribution was observed 96 hours after administration of 2.61 mg/kg (U.S. EPA 1989).

3. Metabolism - Free chlorine (Cl2, OCl-, or HOCl) is a strong oxidizing agent that reacts readily with biological materials (including proteins and nucleotide bases) to produce a variety of organic chlorinated compounds (U.S. EPA 1989). At least one investigator demonstrated trihalomethanes in the plasma of rats given oral doses of sodium hypochlorite (NTP 1992). Experiments in fasted rats have shown that 81% of the total 36Cl in the plasma, 96 hours after administration, was in the form of chloride ion (U.S. EPA 1989).

4. Excretion - Chlorine is excreted from the body primarily in the urine and feces. Rats given oral doses of 2.61 mg/kg of (HO)36Cl excreted 36.4% of the administered dose in the urine and 14.80% in the feces within 96 hours of dosing (U.S. EPA 1989). 36Cl compounds were not detected in expired air.

B. Acute Effects

1. Humans - Oral doses of 2.5 mg chlorine/day, administered to 10 men for 12 weeks, had no adverse effects (U.S. EPA 1990). No adverse effects were noted in persons ingesting water containing 50-90 ppm of chlorine (~1.4 to 2.6 mg Cl/kg/day) for a short periods of time (U.S. EPA 1989). Drinking water concentrations of >90 ppm chlorine caused irritation of membranes of throat and mouth (U.S. EPA 1989). Concentrations of chlorine in the drinking water of greater than 25 ppm make the drinking water unpalatable (U.S. EPA 1989).

   Chlorine is a primary irritant to the mucous membranes of the eyes, nose, and throat and to the linings of the entire respiratory tract (Stokinger 1982). The extent of acute injury to humans depends on the concentration and duration of exposure as well as the water content of the tissue involved and the presence of underlying cardiopulmonary disease (HSDB 1994). The estimated clinical effects of varying concentrations of chlorine are as follows: mild mucous membrane irritation at 1-3 ppm; moderate irritation of the upper respiratory tract at 5-15 ppm; immediate chest pain, vomiting, dyspnea, and cough at 30 ppm; toxic pneumonitis and pulmonary edema at 40-60 ppm; death at 430 ppm for 30 minutes or 1000 ppm for a few minutes (HSDB 1994). Seventy-six individuals, exposed during a football game when approximately 1100 pounds of chlorine gas were released from a plant, suffered no serious or prolonged incapacitation due to the release (HSDB 1994). If one survives acute exposure to chlorine, recovery is usually complete and rapid (U.S.EPA 1989).

2. Animals - LC50 values for rats and mice are 293 ppm for 1 h and 137 ppm for 1 h, respectively (HSDB 1994). LCLo values for other species range from 330 ppm for 7 h (guinea pigs) to 660 ppm for 4 h (cats and rabbits) (U.S. EPA 1989). Mice and rats exposed to chlorine at the RD50 concentration (9-11 ppm, 6 h/day for 1, 3, or 5 days) developed degeneration of

olfactory sensory cells in the olfactory mucosa, loss of cilia of the respiratory epithelium, and cellular exfoliation, primarily of the naso- and maxilloturbinates (HSDB 1994). Signs and symptoms of the acute oral toxicity of chlorine in rats include decreased blood glutathione (30 minutes after 0.2 mg chlorine/kg administered as HOCl); decreased hypothalamic norepinephrine levels and increases in normetanephrine levels (3 and 24 hours after intubation of 250 mg/kg free chlorine as HOCl); morphological and biochemical liver changes (within 2 days of dosing with 142.9 mg/kg free chlorine as NaOCl); kidney enlargement (200 mg/kg/day available chlorine for 14 days) (U.S. EPA 1989); and dose-related increase in liver and kidney weights (210 mg dietary chlorine/kg body weight/day for 28 days) (U.S. EPA 1987). C57Bl/6N mice given chlorine (25-30 ppm) in the drinking water for one to three weeks had significant decreases in the number of peritoneal exudate macrophages, and decreased in vitro cytotoxicity against mouse melanoma (B16) and fibrosarcoma (UV-112) target cells (U.S. EPA 1989).

C. Subchronic/Chronic Effects

The major target organs for the subchronic/chronic toxicity of chlorine in humans are the respiratory tract and the blood. The major target organs for the subchronic/chronic toxicity of chlorine in animals are the immune system, the blood, the cardiovascular system and the respiratory tract. EPA has derived an oral RfD (reference dose) (see end note 3) of 0.1 mg/kg/day for chlorine, based on a no-observed-adverse-effect level of 14.4 mg/kg/day in a chronic drinking water study in rats.

1. Humans - Fifty-five workers exposed to about 1.0 ppm of chlorine, 8 h/day for at least 90 days exhibited (based on reported symptoms and x-rays) asthma, chronic bronchitis, tuberculosis, and emphysema (U.S. EPA 1987). The concentration of 1 ppm is roughly equivalent to 0.415 mg/kg/day (see end note 4).

   In one case study, exposure to 0.015 mg/L of chlorine, 8 h/day for 6 years resulted in dyspnea, marked emphysema of both lower lung lobes, and reduced respiratory mobility (U.S. EPA 1987).

   Fifteen male workers exhibited decreased residual levels of maximal mid-expiratory flow rates and FEV/FVC after exposure to 0.18 ppm chlorine, 8 h/day, 5 days/week for 8.9 years (U.S. EPA 1990). Workers in 25 chlor-alkali plants (332 males), exposed to chlorine concentrations of 0.006 to 1.42 ppm (8-h TWAs) for about 10.9 years showed no dose-related correlation between chlorine exposure and the prevalence of colds, dyspnea, palpitation, chest pain, or permanent lung damage (544 exposed workers had chest x-rays) (Stokinger 1982). Electrocardiograms were abnormal in 9.4% of the exposed workers, compared with 8.5% of unexposed controls, and the incidence of fatigue was increased in workers exposed to >0.5 ppm. There were small correlations between exposure to chlorine and anxiety and dizziness ($p = 0.02$), leukocytosis ($p<0.05$) and decreased hematocrit ($p< 0.017$). A smaller study included 52 workers exposed to about 0.289 ppm chlorine for 10 years (Stokinger 1982). These workers exhibited a slightly significant, lower value for a single pulmonary function test.

2. Animals - Groups of 70 F344/N male and female rats were given drinking water containing up to 275 ppm of chlorine (14.4 mg/kg/day) for 2 years. No hematologic abnormalities were detected at interim sacrifices (14 and 66 weeks) and no non neoplastic abnormalities were observed in extensive gross

and microscopic examinations at the end of the study (NTP 1992). The EPA used the no-observed-adverse-effect level, 14.4 mg/kg/day, to calculate a chronic oral RfD of 0.1 mg/kg/day for chlorine (U.S. EPA 1994).

Sprague-Dawley rats, given 5, 15, or 30 ppm chlorine in drinking water from weaning to 12 weeks of age exhibited significantly reduced spleen weights, suppressed delayed-type hypersensitivity reactions, and decreased oxidative metabolism by macrophages at the highest dose; no effects were noted for the other doses or on other parameters of immunotoxicity (NTP 1992). F344 rats given doses of chlorine up to 60 mg/kg/day for 92 days exhibited no histopathology in major organs (U.S. EPA 1989). Other species, exposed subchronically to chlorine exhibited the following: mice had no adverse effects on weight gain, food or water consumption, histological parameters (12.5 mg/kg/day for 50 days or 25 mg/kg/day for 33 days) (U.S. EPA 1989); New Zealand rabbits had increased hydroxyproline levels in heart tissue (1.6 mg chlorine/kg/day for 3 months; no effect at 0.1 mg/kg/day) (U.S. EPA 1989); Carneau pigeons exhibited cardiovascular effects (10 mg chlorine/ kg/day in drinking water for 9 months) (U.S. EPA 1989).

In various studies, Sprague Dawley and F344 rats treated with chlorine orally for 12-24 months at doses ranging from 0.14 to 22 mg/kg/day exhibited one or more of the following symptoms: significant decreases in red blood cell count and hematocrit (reversed at 6 months), increased osmotic fragility, increased mean corpuscular hemoglobin, significant decreases in body and liver weights, decreased brain and heart weights ($p<0.05$), and decreased salivary gland and kidney weights (U.S. EPA 1989), decreased spleen weight, and decreased thyroid weight (U.S. EPA 1987).

Rhesus monkeys (4/sex/dose) exposed to ó2.3 ppm of chlorine 6 h/day, 5 days/week for 1 year experienced ocular irritation, conjunctival irritation, hyperplasia of the nasal mucosa, and, in some females, epithelial hyperplasia of the trachea with minimal evidence of early nonkeratinizing squamous metaplasia (U.S. EPA 1990).

D. Carcinogenicity

No conclusion on the carcinogenicity of chlorine can be made based on the limited information available from human and animal studies.

1. Humans - NTP (1992) notes that some epidemiologic studies have shown an association between the consumption of chlorinated drinking water and an increased risk for the development of cancer, primarily of the urinary tract. EPA has established drinking water standards for several chlorinated by-products found in chlorinated drinking water.

2. Animals - F344/N male and female rats and B6C3F1 male and female mice (50/sex/group) received 0, 70, 140, or 275 ppm chlorine (based on atomic chlorine) in their drinking water for 2 years. (NTP 1992). The female rats had a slignt increase in the incidence of mononuclear cell leukemia that was not clearly dose-related (control, 8/50; low-dose, 7/50; mid-dose, 19/51; high-dose, 16/50), but was considered by the NTP to be "equivocal evidence of carcinogenic activity". There was "no evidence of carcinogenic activity" in male rats or male and female mice at the doses tested.

In another study, chlorine administered as NaOCl to rats in drinking water at levels up to 1000 mg/L for males and 200 mg/L for females was not carcinogenic (NTP 1992). Information on the cocarcinogenicity of sodium hypochlorite are conflicting. When applied to the skin of mice, sodium hypochlorite enhanced tumor development initiated by 4-nitroquinoline-1-oxide and reduced tumor development initiated by benzo[a]pyrene (U.S. EPA 1989).

E. Genotoxicity

Chlorine was mutagenic in Salmonella typhimurium strains TA1530 and TA100, without metabolic activation; produced chromosome aberrations in human lymphocytes and other mammalian cells (ò20 ppm); interacted with DNA in E. coli polA test (as sodium hypochlorite); and was negative for the induction of erythrocyte micronuclei or chromosome aberrations of bone marrow cells of Swiss CD-1 mice (up to 8 mg/kg/day of NaOCl) for up to 5 days (U.S. EPA 1989).

F. Developmental/Reproductive Toxicity

No conclusion on the developmental/reproductive toxicity of chlorine can be made based on the limited information available from human and animal studies.

1. Humans - No information was found in the secondary sources searched to indicate that chlorine is a developmental/ reproductive toxicant in humans.

2. Animals - Female Sprague-Dawley rats were given chlorine concentrations of 0, 1.0, 10, or 100 mg/L in the drinking water (as HOCL) for 2.5 months prior to conception and throughout gestation, were killed on gestation day 20, and the fetuses were examined (NTP 1992). Resorptions were not increased at any concentration; however, fetuses in the 100 mg/L group had soft tissue defects, including improper orientation of the heart and adrenal agenesis, and a slightly increased incidence of skeletal variants, such as incompletely ossified or missing sternebrae or rudimentary ribs. Statistical analyses were not available. In other studies, chlorine administered to pregnant mice was negative for reproductive and teratogenic effects when given in drinking water as 10-13 ppm of sodium hypochlorite and hydrochloric acid (to maintain water pH of 2.5) or when given in heavily chlorinated municipal drinking water (U.S. EPA 1989).

   Sperm head abnormalities were observed in B6C3F1 mice given 1.6 and 4.0 mg/kg/day of chlorine as OCl-, but not at 8.0 mg/kg or when given as HOCl (U.S. EPA 1994). BDII rats (236 total males and females) given 10 mg/kg/day of free residual chlorine in the drinking water throughout 7 generations had no adverse effects on weight gain, food consumption, water consumption, fertility, lifespan, growth pattern, hematology, histology (liver, spleen, kidney or other organs) (U.S. EPA 1989).

G. Neurotoxicity

No information was found in the secondary sources searched regarding the neurotoxicity of chlorine.

V. ENVIRONMENTAL EFFECTS

A. Toxicity to Aquatic Organisms

Chlorine has high acute toxicity to aquatic organisms; many toxicity values are less than or equal to 1 mg/L. Twenty-four-hour LC50 values range from 0.076 to 0.16 mg/L for Daphnia magna (water flea) and from 0.005 to 0.1 mg/L for Daphnia pulex (cladocern) (AQUIRE 1994); 48-hour LC50 values range from 5.3 to 12.8 mg/L for Nitocra spinipes (snail); and 96-hour LC50 values range from 0.13 to 0.29 mg/L for Oncorhynchus mykiss (rainbow trout), from 0.1 to 0.18 mg/L for Salvelinus fontinalis (brook trout), and from 0.71-0.82 mg/L for Lepomis cyanellus (green sunfish) (AQUIRE 1994). Papillomas of the oral cavity in fish have been associated with exposure to chlorinated water supplies (NTP 1992).

Low level chlorination (0.05 to 0.15 mg/L) results in significant shifts in the species composition of marine phytoplankton communities (HSDB 1994).

B. Toxicity to Terrestrial Organisms

Chlorine is phytotoxic but is also essential to plant growth; crops need around 5 pounds or more of chlorine per acre. Acute toxicity to plants is characterized by defoliation with no leaf symptoms and, in higher plant forms, by spotting of the leaves (at 1.5 mg/m3) and marginal and interveinal injury (at 150-300 mg/m3) (Seiler et al. 1988). No data were found in the secondary sources searched regarding the toxicity of chlorine to terrestrial animals; however, the data from experimental studies indicate that injury to animals would occur only in the presence of high concentrations of chlorine, either in drinking water or the ambient atmosphere.

C. Abiotic Effects

Information regarding the abiotic effects of chlorine was not found in the secondary sources searched.

VI. EPA/OTHER FEDERAL/OTHER GROUP ACTIVITY

The Clean Air Act Amendments of 1990 list chlorine as a hazardous air pollutant. Occupational exposure to chlorine is regulated by the Occupational Safety and Health Administration. The permissible exposure limit (PEL) is 1 part per million parts of air (ppm) or 3 mg/m3 as a ceiling value (i.e., exposures may not exceed these levels) (29 CFR 1910.1000).

Federal agency and other group activities for chlorine are summarized in Tables 4 and 5.

TABLE 4. EPA OFFICES AND CONTACT NUMBERS FOR INFORMATION ON CHLORINE

EPA OFFICE	LAW	PHONE NUMBER
Pollution Prevention & Toxics	Toxic Substances Control Act (Sec. 8A/8D/8E)	(202) 554-1404
	Emergency Planning and Community Right-to-Know Act (EPCRA)	
	Regulations (Sec. 313)	(800) 424-9346
	Toxics Release Inventory data	(202) 260-1531
Air	Clean Air Act	(919) 541-0888
Solid Waste & Emergency Response	Comprehensive Environmental Response, Compensation, and Liability Act (Superfund)/ Resource Conservation and Recovery Act / EPCRA (Sec. 302/304/311/312)	(800) 424-9346
Water	Clean Water Act	(202) 260-7588

TABLE 5. OTHER FEDERAL OFFICE/OTHER GROUP
CONTACT NUMBERS FOR INFORMATION ON CHLORINE

Other Agency/Department/Group	Contact Number
Agency of Toxic Substances & Disease Registry	(404) 639-6000
American Conference of Governmental Industrial Hygienists (Recommended Exposure Limit (see end note 5): 0.5 ppm) (Recommended Short Term Exposure Limit (see end note 6): 1 ppm)	(513) 742-2020
Consumer Product Safety Commission	(301) 504-0994
Food & Drug Administration	(301) 443-3170
National Institute for Occupational Safety & Health Recommended Exposure Limit (see end note 6): 0.5 ppm)	(800) 356-4674
Occupational Safety & Health Administration (Permissible Exposure Limit (see end note 7): 1 ppm) (Check local phone book for phone number under Department of Labor)	

VII. END NOTES

1. Standard Industrial Classification code is the statistical classification standard for all Federal economic statistics. The code provides a convenient way to reference economic data on industries of interest to the researcher. SIC codes presented here are not intended to be an exhaustive listing; rather, the codes listed should provide an indication of where a chemical may be most likely to be found in commerce.

2. The RD50 concentration is that concentration that reduces respiratory rate by 50%.

3. The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during the time period of concern.

4. Calculated using the factor 2.9 mg/m3 (U.S. EPA 1989) to convert 1 ppm to 2.9 mg/m3 which is multiplied by 0.143 (the occupational standard 8-hour breathing rate, 10 m3, divided by the assumed adult body weight, 70 kg, and assuming 100% absorption) to obtain the dose in mg/kg/day (U.S. EPA 1988).

5.The ACGIH exposure limit is a time-weighted average (TWA) concentration for an 8-hour workday for a 40-hour workweek.

6. This is a 15-minute short term exposure limit value that should not be exceeded at any time.

7. This is a 15-minute ceiling exposure limit value that should not be exceeded at any time.

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APPENDIX A. SOURCES SEARCHED FOR FACT SHEET PREPARATION

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