IMMEDIATELY DANGEROUS to LIFE or HEALTH

IDLH

VALUE PROFILE

Hydrogen Chloride
CAS® No. 7647-01-0

U.S. Centers for Disease Control and Prevention
National Institute for Occupational Safety and Health

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IMMEDIATELY DANGEROUS TO LIFE OR HEALTH (IDLH) VALUE PROFILE

HYDROGEN CHLORIDE

[CAS® No. 7647-01-0]

DEPARTMENT OF HEALTH AND HUMAN SERVICES
Centers for Disease Control and Prevention
National Institute for Occupational Safety and Health

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On August 10, 2023, NIOSH published a request for public review in the Federal Register [88 FR 54319] on the draft version of the Immediately Dangerous to Life or Health for Hydrogen Chloride. We invited comments from manufacturers, distributors/vendors, healthcare providers, government agencies, academia, professional organizations, non-government organizations, and members of the public. NIOSH did not receive public comments on the draft document for hydrogen chloride.

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Suggested Citation

NIOSH [2025]. Immediately dangerous to life or health (IDLH) value profile: hydrogen chloride, CAS® No. 7647-01-0. By Frank E, Chittiboyina S, Leshner M, Edmondson M, Niemeier RT. Cincinnati, OH: U.S. Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 2025-110 (Revised 06/2025).

DHHS (NIOSH) Publication No. 2025-110 (revised 06/2025)

June 2025

Summary of changes: updated authorship and acknowledgements.

Worker Summary of the NIOSH Immediately Dangerous to Life or Health (IDLH) Value Profile for Hydrogen Chloride

CAS Number: 7647-01-0
IDLH Value: 45 parts per million (ppm) or 67 milligrams per cubic meter (mg/m^3)

General Substance Information

Other names:
- HCl
- hydrochloric acid
- Muriatic acid

HCl:
- is a colorless to slightly yellow gas with a strong, irritating smell
- is corrosive
- is **not** flammable
- is used in many industrial processes
- is formed during burning of plastics and chemicals
- liquid gives off fumes that sink in air

Health Effects of HCl

Short-term exposure to dangerous levels causes:
- eye irritation (stinging and burning)
- coughing
- difficulty breathing
- symptoms get worse as exposure continues

As HCl levels increase:
- throat pain
- asthma-like symptoms
- lung injury and pulmonary edema (fluid in the lungs)

For more information on HCl visit: LINK TO CHEMICAL DOCUMENT

What is an IDLH Value?

NIOSH develops IDLH values for workplace conditions carrying immediate, unacceptable risks. As a safety margin, IDLH values are based on the effects that might occur from 30-minute exposures. Workers should not stay in an IDLH environment longer than absolutely necessary. **EVERY EFFORT SHOULD BE MADE TO EXIT IMMEDIATELY!** Short exposures to highly concentrated chemicals in the air can quickly overwhelm workers and harm worker health. Harmful effects may include:
- Long-term health issues
- Inability to escape the area
- Death

Workers should **never** be exposed to air concentrations that exceed the IDLH value without proper respiratory protection. NIOSH sets IDLH values to make sure that a worker can escape **immediately** from an area before severe injuries occur.

Employers **must require workers** to wear a NIOSH Approved® full facepiece self-contained breathing apparatus (SCBA) or a combination supplied air respirator with SCBA when entering IDLH conditions. These respirators deliver clean air to the worker in dangerous conditions, and these provide the greatest protection.

NIOSH Approved is a certification mark registered in the United States and several international jurisdictions.

Basis for IDLH Value: In an experiment done in guinea pigs, exposure for 30 minutes caused severe shortness of breath and death after exposure to increasingly high concentrations of HCl gas. The concentration these effects occurred at in guinea pigs was used to estimate an IDLH value for humans. Because humans could be more sensitive than guinea pigs, the IDLH concentration was divided by an uncertainty factor to account for the expected difference. The IDLH value was calculated to be 45 ppm. Reports reviewed by NIOSH indicate that people exposed to this concentration may experience burning sensations in the nose and throat. Continuous exposure to this concentration is expected to cause corrosive injury to the airways and lungs.

For more information on HCl visit: NIOSH POCKET GUIDE

Foreword

Chemicals are a frequent component of the modern workplace. Occupational exposures to chemicals have long been recognized as having the potential to adversely affect the lives and health of workers. Acute or short-term exposures to high concentrations of some airborne chemicals can quickly overwhelm workers, affecting their ability to escape from the exposure environment. These exposures can result in a spectrum of negative outcomes—from eye and respiratory irritation to severe, irreversible health effects—and in extreme cases, death.

Airborne concentrations of chemicals capable of causing such adverse health effects or of impeding escape from high-risk conditions may come from a variety of nonroutine workplace situations affecting workers. These may include special work procedures (e.g., in confined spaces), industrial incidents (e.g., chemical spills or explosions), and chemical releases into the community (e.g., during transportation incidents or other uncontrolled-release scenarios).

This technical report presents the scientific basis, toxicologic data, and risk assessment methodology used to derive a health-based immediately dangerous to life or health (IDLH) value for hydrogen chloride (CAS No. 7647-01-0). The IDLH values are based on the scientific rationale and logic outlined in the *Current Intelligence Bulletin (CIB) 66: Derivation of Immediately Dangerous to Life or Health Values* [NIOSH 2013].

This approach is intended to (1) update the scientific basis and risk assessment methodology used to derive IDLH values from quality toxicity and human health effects data and (2) provide transparency behind the rationale and derivation process for IDLH values. The IDLH value for hydrogen chloride has been established through the approach outlined in CIB 66 and is intended to protect against health effects that impair escape, are irreversible effects, or result in death from exposures of 30 minutes or less.

John Howard, M.D.
Director
National Institute for Occupational Safety
and Health
Centers for Disease Control and Prevention

Contents

Abbreviations*

ACGIH®	American Conference of Governmental Industrial Hygienists
AEGLs	acute exposure guideline levels
AIHA®	American Industrial Hygiene Association
ARDS	acute respiratory distress syndrome
atm	atmosphere (unit of pressure)
BMC	benchmark concentration
BMCL	benchmark concentration lower confidence limit
BMD	benchmark dose
BMR	benchmark response
C	ceiling value
°C	degree Celsius
CAS®	chemical abstract service
CIB	Current Intelligence Bulletin
ERPGs™	Emergency Response Planning Guidelines
HCl	hydrogen chloride
hr	hour
IDLH	immediately dangerous to life or health
IFA	Institut für Arbeitsschutz der Deutschen Gesetzlichen Unfallversicherung (Institute for Occupational Safety and Health of the German Social Accident Insurance)
LC	lethal concentration
LC_{01}	1% lethal concentration
LC_{50}	median lethal concentration
LC_{LO}	lowest concentration of a chemical that caused death in humans or animals
LD_{50}	median lethal dose
LD_{LO}	lowest dose that caused death in humans or animals
LEL	lower exposure limit
LOAEL	lowest observed adverse effect level
mg/m^3	milligram(s) per cubic meter
min	minute
mm Hg	millimeter(s) of mercury
NAC	National Advisory Committee
NAS	National Academy of Sciences
NIOSH	National Institute for Occupational Safety and Health
NLM	National Library of Medicine
NOAEL	no observed adverse effect level
NRC	National Research Council
OEL	occupational exposure limit
OSHA	Occupational Safety and Health Administration
PEL	permissible exposure limit
ppm	parts per million

RADS	reactive airways disorder syndrome
RD_{50}	concentration of a chemical in the air that is estimated to cause a 50% decrease in the respiratory rate
REACH	Registration, Evaluation, Authorization, and Restriction of Chemicals (European Union regulatory program)
REL	recommend exposure limit
RfC	reference concentration
STEL	short term exposure limit
TEEL	temporary emergency exposure limit
TERA	Toxicology Excellence for Risk Assessment
TLV®	threshold limit value
TWA	time weighted average
UEL	upper explosive limit
UF	uncertainty factor
WEELs®	workplace environmental exposure levels

**Abbreviations listed are based on recurring use in IDLH documents and do not necessarily indicate usage in this assessment*

Glossary

Acute exposure: Exposure by the oral, dermal, or inhalation route for 24 hours or less.

Acute exposure guideline levels (AEGLs): Threshold acute exposure limits for the general public, applicable to exposure periods ranging from 10 minutes to 8 hours. AEGL-1, AEGL-2, and AEGL-3 values for individual chemicals are developed for reversible and nondisabling, irreversible or disabling, and lethal effects, respectively. Five values at each severity level are developed for 10 minutes, 30 minutes, 1 hour, 4 hours, and 8 hours [NRC 2004]. AEGLs are intended to be guideline levels used during rare events or single once-in-a-lifetime exposure to airborne concentrations of acutely toxic, high-priority chemicals [NRC 2004]. AEGLs are designed to protect the general population, including the elderly, children, and other potentially sensitive groups that are generally not considered in the development of workplace exposure recommendations. (Additional information is available at https://www.epa.gov/aegl.)

Acute reference concentration (Acute RfC): An estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure for an acute duration (24 hours or less) of the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from a NOAEL, LOAEL, or benchmark concentration, with uncertainty factors (UFs) generally applied to reflect limitations of the data used. Generally used in EPA noncancer health assessments [EPA 2022].

Acute toxicity: Any poisonous effect produced within a short period of time following an exposure, usually 24 to 96 hours.

Adverse effect: A substance-related biochemical change, functional impairment, or pathologic lesion that affects the performance of an organ or system or alters the ability to respond to additional environmental challenges.

Benchmark dose/concentration (BMD/BMC): A dose or concentration that produces a predetermined change in response rate of an effect (called the benchmark response, or BMR) compared with background [EPA 2022]. (Additional information is available at https://www.epa.gov/bmds.)

Benchmark response (BMR): A predetermined change in response rate of an effect. Common defaults for the BMR are 10% or 5%, reflecting study design, data variability, and sensitivity limits used.

Benchmark concentration lower confidence limit (BMCL): A statistical lower confidence limit on the concentration at the BMC [EPA 2022].

Bolus exposure: A single, relatively large dose.

Ceiling value ("C"): Term in occupational exposure indicating the airborne concentration of a potentially toxic substance that should never be exceeded in a worker's breathing zone.

Chronic exposure: Repeated exposure for an extended period of time. Typically, exposures are more than approximately 10% of life span for humans and >90 days to 2 years for laboratory species.

Critical study: The study that contributes most significantly to the qualitative and quantitative assessment of risk [EPA 2022].

Dose: The amount of a substance available for interactions with metabolic processes or biologically significant receptors after crossing the outer boundary of an organism [EPA 2022].

Emergency Response Planning Guidelines (ERPGs™): Maximum airborne concentrations below which nearly all individuals can be exposed without experiencing health effects for 1-hour exposure. ERPGs are presented in a tiered fashion with health effects ranging from mild or transient to serious, irreversible, or life threatening (depending on the tier). ERPGs are developed by the American Industrial Hygiene Association [AIHA 2016].

Endpoint: An observable or measurable biological event or sign of toxicity ranging from biomarkers of initial response to gross manifestations of clinical toxicity.

Exposure: Contact made between a chemical, physical, or biological agent and the outer boundary of an organism. Exposure is quantified as the amount of an agent available at the exchange boundaries of the organism (e.g., skin, lungs, gut).

Extrapolation: An estimate of the response at a point outside the range of the experimental data, generally through the use of a mathematical model, although qualitative extrapolation may also be conducted. The model may then be used to extrapolate to response levels that cannot be directly observed.

Hazard: A potential source of harm. Hazard is distinguished from risk, which is the probability of harm under specific exposure conditions.

Immediately dangerous to life or health (IDLH) condition: A situation that poses a threat of exposure to airborne contaminants when that exposure is likely to cause death or immediate or delayed permanent adverse health effects or prevent escape from such an environment [NIOSH 2004, 2013].

IDLH value: A maximum (airborne concentration) level above which only a highly reliable breathing apparatus providing maximum worker protection is permitted [NIOSH 2004, 2013]. IDLH values are based on a 30-minute exposure duration.

LC_{01}: The statistically determined concentration of a substance in the air that is estimated to cause death in 1% of the test animals.

LC_{50}: The statistically determined concentration of a substance in the air that is estimated to cause death in 50% (one half) of the test animals; median lethal concentration.

LC_{LO}: The lowest lethal concentration of a substance in the air reported to cause death, usually for a small percentage of the test animals.

LD_{50}: The statistically determined lethal dose of a substance that is estimated to cause death in 50% (one half) of the test animals; median lethal concentration.

LD_{LO}: The lowest dose of a substance that causes death, usually for a small percentage of the test animals.

Lethality: Pertaining to or causing death; fatal; referring to the deaths resulting from acute toxicity studies. May also be used in lethality threshold to describe the point of sufficient substance concentration to begin to cause death.

Lower explosive limit (LEL): The minimum concentration of a gas or vapor in air, below which propagation of a flame does not occur in the presence of an ignition source.

Lowest observed adverse effect level (LOAEL): the lowest tested dose or concentration of a substance that has been reported to cause harmful (adverse) health effects in people or animals.

Mode of action: The sequence of significant events and processes that describe how a substance causes a toxic outcome. Mode of action is distinguished from the more detailed mechanism of action, which implies a more detailed understanding on a molecular level.

No observed adverse effect level (NOAEL): The highest tested dose or concentration of a substance that has been reported to cause no harmful (adverse) health effects in people or animals.

Occupational exposure limit (OEL): Workplace exposure recommendations developed by governmental agencies and nongovernmental organizations. OELs are intended to represent the maximum airborne concentrations of a chemical substance below which workplace exposures should not cause adverse health effects. OELs may apply to ceiling, short-term (STELs), or time-weighted average (TWA) limits.

Peak concentration: Highest concentration of a substance measured during a certain period of observation.

Permissible exposure limit (PEL): Occupational exposure limits developed by OSHA (29 CFR § 1910.1000) or MSHA (30 CFR § 57.5001) for allowable occupational airborne exposure concentrations. PELs are legally enforceable and may be designated as ceiling, STEL, or TWA limits [OSHA 2019].

Point of departure (POD): The point on the dose–response curve from which dose extrapolation is initiated. This point can be the lower bound on dose for an estimated incidence or a change in response level from a concentration-response model (BMC). It can also be a NOAEL or LOAEL for an observed effect selected from a dose evaluated in a health effects or toxicology study.

RD_{50}: The statistically determined concentration of a substance in the air that is estimated to cause a 50% (one half) decrease in the respiratory rate.

Recommended exposure limit (REL): Recommended maximum exposure limit to prevent adverse health effects based on human and animal studies and established for occupational (up to 10-hour shift, 40-hour week) inhalation exposure by NIOSH. RELs may be designated as ceiling, STEL, or TWA limits.

Short-term exposure limit (STEL): An exposure concentration limit that shall not be exceeded at any time during a working day, usually based on a 15-minute time-weighted average unless otherwise noted.

Target organ: Organ in which the toxic injury manifests in terms of dysfunction or overt disease.

Threshold limit values (TLVs®): Recommended guidelines for occupational exposure to airborne contaminants, published by the American Conference of Governmental Industrial Hygienists

(ACGIH). TLVs refer to airborne concentrations of chemical substances and represent conditions under which it is believed that nearly all workers may be repeatedly exposed, day after day, over a working lifetime, without adverse effects. TLVs may be designated as ceiling, short-term (STELs), or 8-hour TWA limits [ACGIH 2021].

Time-weighted average (TWA): A worker's 8-hour (or up to 10-hour) time-weighted average exposure concentration that shall not be exceeded during an 8-hour (or up to 10-hour) work shift of a 40-hour week. The average concentration is weighted to take into account the duration of different exposure concentrations.

Toxicity: The degree to which a substance is able to cause an adverse effect on an exposed organism.

Uncertainty factors (UFs): Mathematical adjustments applied to the POD when developing exposure limits or IDLH values. The UFs for IDLH value derivation are determined by considering the study and effect used for the POD, with further modification based on the overall database.

Workplace Environmental Exposure Levels (WEELs®): Exposure levels that provide guidance for protecting most workers from adverse health effects related to occupational chemical exposures expressed as a TWA or ceiling limit.

Acknowledgments

This document was developed by the Division of Science Integration, Christine Whittaker, PhD, Director. The authors of this report are Evan Frank, PhD, DABT; Shirisha Chittiboyina, PhD; Molly Leshner, MPH; Melissa Edmondson, PhD, CIH; and R. Todd Niemeier, PhD, CIH.

The following NIOSH staff are acknowledged for providing technical and editorial review of the report:

Theresa Boots, MS
Kathleen MacMahon, DVM
Sudha Pandalai, MD, PhD
Christine Whittaker, PhD

NIOSH would like to acknowledge the contribution of the following subject matter experts for their critical technical review of this report:

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The basis for this document was a report contracted by NIOSH and prepared by Andrew Maier, PhD, DABT, CIH; Ann Parker; and Lynne Haber, PhD, DABT (Toxicology Excellence for Risk Assessment [TERA]).

Krystin Carlson, PhD (formerly of the Division of Science Integration) also contributed to earlier versions of this document.

IDLH Value for Hydrogen Chloride

IDLH Value: 45 ppm (67 mg/m^3)

Basis for IDLH Value: The immediately dangerous to life and health (IDLH) value for hydrogen chloride (HCl) is based on lethality. Hartzell et al. [1988] obtained a 30-minute LC_{50} value for HCl gas exposure in guinea pigs, the most sensitive 30-minute LC_{50} value identified for HCl. An uncertainty factor (UF) of 30 was applied to estimate the risk of severe injury or death in a workplace emergency. The IDLH was calculated to be 45 ppm. This updates the previous IDLH value of 50 ppm that was based on case reports of acute inhalation toxicity in humans.

1 Introduction

1.1 Purpose

This *Immediately Dangerous to Life and Health (IDLH) Value Profile* presents (1) a brief summary of technical data associated with acute inhalation exposures to hydrogen chloride (HCl) and (2) the scientific rationale behind the IDLH value for HCl. IDLH values are developed based on the scientific rationale and logic outlined in the *Current Intelligence Bulletin (CIB) 66: Derivation of immediately dangerous to life or health (IDLH) Values* [NIOSH 2013]. NIOSH performs in-depth literature searches (outlined generally in CIB 66 and further described in Section 1.2 of this document) to ensure that all relevant data from human and animal studies with acute exposures to the substance are identified. The data identified in this literature search were evaluated for relevance by considering the methods used in the studies (i.e., species, study protocol, exposure concentration, and duration), the health endpoint(s) evaluated, and the critical effect levels (e.g., NOAELs, LOAELs, LC_{50} values).

1.2 How IDLH Values Are Set

An IDLH situation is one that poses a threat of exposure to airborne contaminants when that exposure is likely to cause death or immediate or delayed permanent adverse health effects or prevent escape from such an environment [NIOSH 2004]. An IDLH value is a maximum (airborne concentration) level above which only a highly reliable breathing apparatus providing maximum worker protection is permitted [NIOSH 2004]. IDLH values are based on a 30-minute (min) exposure duration and signal that every effort should be made to evacuate the area. These values are designed to protect workers from acute or short-term exposures to high concentrations of airborne chemicals that could quickly overwhelm them, affecting their ability to escape. These exposures could result in a range of undesirable outcomes, from eye and respiratory tract irritation to severe, irreversible health effects, and in extreme cases, death. IDLH values also protect workers against non-toxicological safety hazards, including deprivation of oxygen, impairment of visibility, and ignition in the air.

1.2.1 Health Effects Considered

For the purposes of setting an IDLH value, NIOSH typically considers health effects data for the following acute health endpoints [NIOSH 2013]:

- Lethality/death
- Acute deficits in neurological and/or psychomotor functions that impair escape by inter-

fering with workers' ability to recognize the escape routes and any actions needed to get away through those routes, such as the operation of lifts, elevators, and door mechanisms

- Eye irritation severe enough to affect workers' ability to see adequately and escape the area
- Respiratory irritation severe enough to impair breathing assuming a non-rest scenario, or that results in long-term respiratory complications
- Cardiac and hematological effects, including cardiac sensitization
- Any other specific target organ effects that are incapacitating/escape impairing or have the potential for long-term injury, disability, or deficits in function

1.2.2 Time Scaling

Effect levels for acute exposures are adjusted to 30-min effect levels when needed using the ten Berge et al. [1986] method, where a "k" constant value is calculated from concentration (C) and time (t) using the equation $C^n \times t = k$. When the value of the exponent n can be derived from data, the data-based n is used. Otherwise, default values of 1 for adjusting from a shorter exposure to 30 min and 3 for adjusting from longer exposures are used as described in CIB 66. For effects that are understood to occur based on threshold concentration regardless of exposure duration, time scaling may not be required.

1.2.3 Uncertainty Factor Considerations

The time-scaled effect levels for immediately dangerous health effects are modified by a UF to estimate the concentration correlating to an unacceptable risk of immediately dangerous effects in workers and account for the possibility of underestimating the degree of risk. When estimating an overall UF, NIOSH considers the following types of uncertainty and variability [NIOSH 2020a]:

- Interspecies variability: When the effect level is obtained from animal data, the potential difference between animal and human responses must be accounted for. When data are not available to calculate factors based on chemical-specific variability, a half-log of 10 (equivalent to 3) may be used to account for toxicokinetic differences and another half-log of 10 (equivalent to 3) would account for toxicodynamic differences.
- Human variability in sensitivity: When data are not available to calculate factors based on chemical-specific variability, a half-log of 10 (equivalent to 3) may be used to account for toxicokinetic differences and another half-log may account for toxicodynamic differences between individuals. NIOSH generally assumes workers to be adults and in reasonable health and therefore tends to use smaller factors than when considering variability among the general population.
- Severity of effect: A UF may be applied when the IDLH is based on health effects severe enough that overestimation of the threshold of immediately dangerous or lethal effects in workers becomes a concern. This may be done to ensure that the IDLH is sufficiently protective of workers' health when the boundary between adverse and immediately dangerous risk is difficult to interpret.
- Other factors or database deficiencies: If gaps in the database create the possibility of significantly overestimating the IDLH value, UFs may be used to account for this. In addition, in special cases, other factors may arise that warrant inclusion of a UF.

1.3 Literature Search

Primary Literature Search

NIOSH performed an initial broad literature search and screened literature as outlined in NIOSH Current Intelligence Bulletin 66: Derivation of Immediately Dangerous to Life or Health (IDLH) Values [NIOSH 2013]. This included several public databases consisting of non-peer reviewed literature that were reviewed for toxicity information on HCl.

For searching the peer-reviewed primary literature, the following literature databases were used based on relevance and current availability. These were most recently queried in December 2024 and searches were limited to publication dates between 2004 and 2024:

- PubMed/Medline
- Scopus
- Embase

Search terms used to search the primary literature for effect level data for animal and human endpoints relevant to the IDLH assessment are given in Table 1.1. These terms were used in conjunction with chemical identifiers of "hydrogen chloride" and "hydrochloric acid," as well as "muriatic acid" and the CAS number. The search terms were selected to best reflect the body of literature specific to HCl and most effectively retrieve relevant toxicity data.

Table 1.1: Search Terms Used to Find Human and Animal Acute Toxicity Data

Acute	Symptoms	Accident
Irritation	Lethality	Confusion
Behavioral	LC_{50}	Toxicity
Neuro*	RD_{50}	Occupational
Psycho*	Poisoning	Volunteers
Subjects	Clinical	Animal
Inhalation	ppm	Fatality

*Denotes terms searched as prefixes

Tree Search for Government Reports and Non-peer Reviewed Literature

In addition to primary literature searches, NIOSH reviewed references cited in authoritative reviews and other literature to identify relevant toxicity data. NIOSH primarily used acute exposure guideline level (AEGL) documentation for HCl [NRC 2004]. The REACH (Registration, Evaluation, Authorisation and Restriction of Chemicals) chemical information dossier for HCl [ECHA 2022] was also reviewed for toxicity data. All datasets identified through these means were reviewed by NIOSH to identify effect levels from endpoints relevant to the IDLH assessment.

Screening Methods and Study Inclusion Criteria

NIOSH used the following inclusion criteria to screen for relevant datasets:

- Populations included in the review were human adults, workers, and mammalian test species.
- Exposures included in the review were acute exposures, meaning less than ~1 day for reports and <8 hour (hr) for experiments by any route where dose/concentration is known or estimated. Reports were excluded when the exposure concentration and/or duration were not estimated or reported.
- Comparators/controls included any comparisons between known doses/concentrations including comparisons between nonexposed, lower-exposed, and baseline prior to acute exposure.
- Outcomes included escape-impairing signs, symptoms, and endpoints in humans or animals; persistent adverse signs or symptoms in humans; persistent adverse effects in any organ/species; lethality; or RD_{50} values. For the purposes of the IDLH assessment, "escape-impairing" endpoints include acute neurological symptoms (e.g., recognition of letters and numbers, reaction time, psychomotor performance), irritation of the eyes and/or airways, or self-reported symptoms of the same.

2 General Substance Information

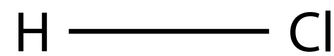
Chemical: Hydrogen Chloride

CAS No: 7647-01-0

Synonyms: HCl, anhydrous hydrochloric acid; muriatic acid*

Chemical category: Inorganic chlorine compounds; inorganic gases†

Structural formula*:



References: *[NLM 2022] †[IFA 2019]

Hydrogen chloride (HCl) is widely used in a broad range of industrial processes. HCl may be supplied as a liquid or compressed gas and work processes where HCl is commonly used include fumigation of ships, buildings, and outdoor agricultural areas, manufacture of acrylics, synthetic rubber, plastics, and dyes, steel and iron hardening, and mining [NIOSH 2019]. HCl is also generated as a decomposition product of burning of various materials, especially combustion of plastics or chlorine-containing compounds such as vinyl chloride. HCl is hygroscopic and forms hydrochloric acid on contact with water. HCl fumes strongly in its liquid form and has a pungent, irritating odor [NLM 2022]. As a gas/vapor, HCl is colorless to slightly yellow, and is heavier than air. HCl is corrosive and nonflammable. In a review of occupational poisonings, Downs et al. (2021) identified 967 catalogued clinical cases arising from occupational exposures to HCl. It is not classified as a human carcinogen [ATSDR 2002; IARC 1992]. Table 2.1 summarizes the physicochemical properties of HCl.

Several agencies and other safety and health organizations have developed occupational exposure limits (OELs) based on the human health effects of HCl exposure. Existing exposure limits for HCl are given in Table 2.2. These range from OELs for daily 8-hr exposures (NIOSH REL, OSHA PEL, ACGIH TLV®) to short-term acute exposures (AIHA ERPG). Limit values estimated for shorter exposure periods are typically at higher concentrations than those estimated for longer periods. The NIOSH IDLH value is estimated for a 30-min exposure period to give workers time to leave the area as quickly and safely as possible.

AEGL values are emergency safety limits developed by the National Research Council (NRC) and designed to protect members of the general public from adverse health effects from airborne chemicals for periods ranging from 10 min to 8 hr. AEGL values are estimated for three ranges of effects: nondisabling (AEGL-1), disabling (AEGL-2), and lethal (AEGL-3). The AEGL value most analogous to the IDLH is the 30-min AEGL-2 value, which is estimated to protect people from irreversible, serious, or escape-impairing effects, including in susceptible individuals. The AEGL values for HCl are listed in Table 2.3.

Table 2.1: Physiochemical Properties of Hydrogen Chloride

Property	Value
Molecular weight	36.5
Description	Colorless to slightly yellow gas
Odor	Pungent, irritating
UEL	Not flammable
LEL	Not flammable
Vapor pressure	40.5 atm
Flash point	Not flammable
Ignition temperature	Not flammable
Solubility in water	67% (86°F)
Relative gas density	1.27
Reactivities and Incompatibilities	Hydroxides, amines, alkalis, copper, brass, zinc (Note: hydrochloric acid is highly corrosive to most metals)

UEL: upper explosive limit; LEL: lower explosive limit
Reference: NIOSH [2020b]

Table 2.2: Exposure Values and Limits for Hydrogen Chloride

Organization	Value (ppm)
NIOSH REL*	5 ppm (7 mg/m^3), ceiling
OSHA PEL[†]	5 ppm (7 mg/m^3), ceiling
ACGIH TLV	2.98 ppm (3 mg/m^3), ceiling; A4
AIHA ERPG[§]	ERPG-1: 3 ppm; ERPG-2: 20 ppm; ERPG-3: 150 ppm

References: [†]Recommended Exposure Limit, NIOSH [2020b]; [‡]Permissible Exposure Limit, OSHA [2019]; Threshold Limit Value, ACGIH [2021]; [§]Emergency Response Planning Guideline, AIHA [2016]

Table 2.3: Acute Exposure Guideline Level Values for Hydrogen Chloride

Classification	10-min	30-min	1-hr	4-hr	8-hr	Endpoint (Reference)
AEGL-1 (Nondisabling)	1.8 ppm 2.8 mg/m^3	1.8 ppm 2.8 mg/m^3	1.8 ppm 2.8 mg/m^3	1.8 ppm 2.8 mg/m^3	1.8 ppm 2.8 mg/m^3	NOAEL in exercising asthmatic subjects [Stevens et al. 1992]
AEGL-2 (Disabling)	100 ppm 153.4 mg/m^3	43 ppm 65 mg/m^3	22 ppm 33.7 mg/m^3	11 ppm 16.9 mg/m^3	11 ppm 16.9 mg/m^3	Mouse RD50 [Barrow et al. 1977]; histopathology in rats [Stavert et al. 1991]
AEGL-3 (Lethal)	620 ppm 950.9 mg/m^3	210 ppm 322.1 mg/m^3	100 ppm 153.4 mg/m^3	26 ppm 39.9 mg/m^3	26 ppm 39.9 mg/m^3	Estimated NOEL for death from 1-hr rat LC50 [Vernot et al. 1977; Wohlslagel et al. 1976]

Reference: NRC [2014]

3 Health Effects of Hydrogen Chloride

Overview of Health Effects: HCl is a respiratory and eye irritant gas that has corrosive properties at higher concentrations. It is rapidly absorbed by the upper and lower respiratory tract in humans due to its high solubility and reactivity [Flury and Zernick 1931]. It has an odor threshold between <1 and ~10 ppm [AIHA 1989].

3.1 Physical Safety

HCl is noncombustible, and ignition is not a safety hazard at any concentration. HCl does not displace oxygen in the concentration ranges causing health effects, but care should be taken in confined spaces since HCl gas is heavier than air.

3.2 Lethality

3.2.1 Overview

Reports of lethality from acute exposure to HCl were limited to animal studies. Although deaths have been reported in animals exposed to as low as 500 ppm HCl for 15 min, the same studies also reported exposing rats and primates to up to 5,000–10,000 ppm for up to 15 min without recording any deaths [Kaplan et al. 1988,1993a,b]. Several LC_{50} values have been reported for HCl and are summarized below. Animals that succumb to HCl exposure experience severe respiratory distress, and show severe injury, membrane denudation, and necrosis to the entire respiratory tract. Airways showed marked congestion. Data also indicate that increases in ventilation during periods of exertion may exacerbate the respiratory effects of HCl [Malek and Alarie 1989].

3.2.2 Human Data

No reports of deaths in humans due to exposure to HCl meeting the inclusion criteria were identified. In their review of clinically significant occupational poisonings, Downs et al. (2021) identified 3 deaths attributed to occupational exposure to HCl in the US between 2008-2018.

3.2.3 Animal Data

Multiple LC_{50} values for HCl were identified for three animal species (rats, mice, and guinea pigs). These are summarized in Table 3.1. The lowest LC_{50} value identified was 1,108 ppm in mice exposed for 60 min [Wohlslagel et al. 1976].

Other reports of lethality in animals included experiments in mice, rats, guinea pigs, and baboons and are summarized here. Kaplan et al. [1985] reported pneumonia, pulmonary edema, tracheitis, and severe dyspnea in two juvenile baboons that died in the days following exposure to 16,000 ppm and 17,000 ppm HCl for 5 min in a study involving escape tasks. Baboons exposed to up to 11,400 ppm survived these experiments. In a series of experiments measuring respiratory function and toxicity, Kaplan et al. [1993a] reported several deaths among male ICR mice and male English guinea pigs exposed to 4,200 ppm HCl for 15 min, with pulmonary congestion, severe tracheitis, and necrosis/desquamation of airway surfaces observed at necropsy. Three out of six male ICR mice died after exposure to 500 ppm for 15 min in the same study, despite showing no histological abnormalities in the lung. In contrast, female Sprague-Dawley rats survived 4,200 ppm for 15 min [Kaplan et al. 1993a]. Burleigh-Flayer et al. [1985] reported deaths in several guinea pigs exposed to 1,040 ppm or 1,380 ppm for 15 min. The deaths occurred within 15 days following the exposure. No deaths occurred in guinea pigs exposed to 640 ppm in this experiment.

Barrow et al. [1979] reported deaths in male Swiss-Webster mice exposed to 12,000 ppm HCl for 10 min. No deaths were reported at the next highest concentration tested, which was approximately 1,000 ppm. The deaths occurred within 24 hr following exposure, after which surviving mice were euthanized.

Malek and Alarie [1989] reported that the lethal concentration of HCl became precipitously lower during periods of exertion. Male English shorthair guinea pigs were exposed to 0 ppm, 107 ppm, 140 ppm, 162 ppm, or 586 ppm HCl in groups of 2–4 animals/concentration. The animals were exposed while running on a treadmill system and ran for 10 min before being exposed to HCl. Exposure continued for 30 min or until incapacitation. Animals exposed to room air or 107 ppm HCl were able to run for the entire 30-min exposure, with similar performance. Animals in the 140 ppm group did not complete the exposure, with a mean incapacitation time of 16 min. Animals exposed to 162 ppm were incapacitated in 1.3 min. HCl was lethal to guinea pigs in the 586 ppm group. Animals in this group ran for an average time of only 0.65 min before incapacitation, and all four of the animals in this test group died with a mean time of 2.8 min elapsing between the cessation of the exposure and death. Airways and lungs of deceased animals did not show obvious obstruction from bronchoconstriction or severe hemorrhage, but coughing, frothing at the mouth, and cyanosis were reported in animals prior to death. The authors estimated that the running guinea pigs were performing at 30% of their maximum VO2 (oxygen uptake), equivalent to a 2–2.5 fold increase over resting baseline.

Sakurai [1989] exposed ICR mice to several concentrations of HCl between 1.0% and 3.5% (10,000 ppm and 35,000 ppm) and recorded the time until animals were “incapacitated” (as described by the author) and the time until they stopped breathing. The longest time to incapacitation observed was 55 min at 1% HCl, and the mice exposed to greater than 2.5% were incapacitated within minutes. The study observed that the time to incapacitation and the time to apnea were roughly equivalent in mice exposed to increasing HCl concentrations. In other words, acutely incapacitating effects in these animals were observed at similar concentrations as acutely lethal effects.

Stavert et al. [1991] exposed male F344 rats to 1,300 ppm HCl for 30 min in either nose-breathing or mouth-breathing (orotracheally tubed) experimental conditions. In the nose-breathing group, 6% of rats died within 24 hr of exposure. In the mouth-breathing group, 46% of rats died within this period.

Table 3.1. Acute Lethality (LC_{50}) Data for Hydrogen Chloride

Species	Reference	LC_{50} (ppm)	Time (min)
Guinea pig	Hartzell et al. [1988]	2,884	15
Guinea pig	Kirsch and Drabke [1982]	2,519	30
Guinea pig	Hartzell et al. [1988]	1,341	30
Rat	Higgins et al. [1972]	40,895	5
Rat (gas)	Darmer et al. [1974]	40,855	5
Rat (aerosol)	Darmer et al. [1974]	20,186	5
Rat	Hartzell et al. [1990]	15,890	5
Rat	Hartzell et al. [1990]	8,380	10
Rat	Hartzell et al. [1990]	6,910	15
Rat	Hartzell et al. [1990]	5,900	22
Rat (aerosol)	Darmer et al. [1974]	5,564	30
Rat (gas)	Darmer et al. [1974]	4,693	30
Rat	Babrauskas et al. [1987]	4,592	30
Rat	Hartzell et al. [1990]	3,821	30
Rat	Wohlslagel et al. [1976]	3,124	60
Rat	Vernot et al. [1977]	3,120	60
Rat	Hartzell et al. [1987]	3,017	30
Rat	Hartzell et al. [1990]	2,816	60
Mouse	Wohlslagel et al. [1976]	66,480	60
Mouse	Wohlslagel et al. [1976]	64,260	30
Mouse (gas)	Darmer et al. [1974]	14,042	5
Mouse	Higgins et al. [1972]	13,743	5
Mouse (aerosol)	Darmer et al. [1974]	11,065	5
Mouse	Alarie and Anderson [1979]	10,123	10
Mouse	Esposito and Alarie [1988]	2,997	30
Mouse (gas)	Darmer et al. [1974]	2,642	30
Mouse (aerosol)	Darmer et al. [1974]	2,146	30
Mouse	Vernot et al. [1977]	1,110	60
Mouse	Wohlslagel et al. [1976]	1,108	60

3.3 Neurotoxicity

No evidence of neurological effects arising from acute exposure to HCl were identified in human or animal data.

3.4 Respiratory and Eye Irritation

3.4.1 Overview

Available human and animal reports indicate that HCl is rapidly absorbed by airway surfaces and causes damage and corrosive injury to tissue in addition to causing typical sensory irritation symptoms (coughing, dyspnea, stinging/burning of eyes). Because of this, HCl can cause persistent adverse effects through the accumulation of injury to local tissue during prolonged or high-level exposures.

In terms of eye irritation, effects appear to develop over several minutes rather than immediately. In a study of baboons, despite visible signs of discomfort, animals were able to see well enough to complete a simple escape task following 5-min exposures to concentrations high enough that some animals died of respiratory injury in the days following [Kaplan et al. 1985]. However, Burleigh-Flyer et al. [1985] reported corneal opacity in guinea pigs exposed to 680 ppm for 30 min. Although several reports do not include details about eye irritation effects, the data are consistent with an understanding that caustic damage plays a large role in the effects of HCl on the eye. Therefore, effects on eye irritation are expected to accumulate over a 30-min exposure period and may be more dependent on cumulative exposure rather than peak concentration.

In terms of respiratory irritation, HCl causes some classic sensory airway effects (cough, dyspnea) as well as corrosive damage to airway tissue. Data from sensory irritation experiments in animals suggest that the effect of inhaled HCl on respiratory function (i.e., changes in breathing frequency and minute volume) varies based on how much HCl is absorbed by upper airways and how much reaches the lower airways and lung [Burleigh-Flyer et al. 1985; Stavert et al. 1991]. At lower exposure concentrations, breathing frequency and minute volume decrease immediately similarly to other sensory irritants. At higher exposure concentrations where greater amounts of HCl appear to be reaching the lung, breathing and minute volume can compensate back to normal levels and even increase over baseline.

In a comparison study of rats exposed to HCl via mouth-only or nose-only, Stavert et al. [1991] estimated that mouth-exposed rats absorbed more HCl than nose-exposed due to the relative increase in minute volume. This suggests that mouth-breathing species may be more susceptible than obligate nose-breathers to lung effects of HCl exposure due to differences in absorbed dose and lack of absorption in the nasopharyngeal region. In addition, HCl exposures in the Stavert et al. study were markedly more lethal in rats exposed via mouth as compared with the nose. This indicates that caution should be exercised when extrapolating from HCl animal data. Available case summaries (described below) of human patients hospitalized following HCl inhalation reported coughing, breathlessness, and chest tightness/pain as the primary clinical symptoms. These symptoms were experienced during exposure and continued to worsen afterwards, though all patients eventually made full recoveries.

3.4.2 Human Data

Limited human subject data are available and are summarized below. Effects reported in available studies are qualitative descriptions and describe the “maximum tolerated” concentration of HCl to be potentially between 10 ppm and 100 ppm in exposures ranging from 10–15 min to 1 hr. It is generally difficult to interpret whether the effect descriptions represent immediately dangerous effects for the purposes of this assessment, and details on how exposure concentrations were measured are lacking for some studies. These reports suggest that concentrations above approximately 100 ppm should be considered acutely and immediately dangerous, whereas exposures in the range of 10–70 ppm are noticeably uncomfortable but may not be incapacitating over a 30-min exposure. Case reports [Boulet et al. 1988, Bansal et al. 2011, Li & Cheng 2014, Xia et al.2018] of workers briefly exposed to high concentrations of HCl gas or fumes indicated that in addition to immediate pungency and discomfort, severe respiratory symptoms continued to develop in the hours following acute inhalation. Although workers in these reports experienced severe respiratory distress requiring hospitalization, all made full recoveries with no lung abnormalities observed at discharge. These reports involved spills and/or workers exposed to pockets of highly concentrated hydrochloric acid fumes, and estimation of concentrations or absorbed doses was not possible. The available human data are summarized below.

Two textbooks contain citations for previously reported data not otherwise identified in literature. Jacobs [1967] cited several previous sources reporting concentrations of 0.13%–0.2% (1300–2000 ppm) as being "lethal for humans in exposures lasting a few minutes," and that the "maximum concentration tolerated" for 60-min exposures was in the range of 0.001%–0.005% (10–50 ppm). Henderson and Haggard [1943] cited 50–100 ppm as being the "maximum concentration tolerable" for humans over a 1-hr exposure.

Matt in 1889 [as cited in NRC 1998] exposed two adult subjects to 10 ppm, 70 ppm, and 100 ppm HCl for 15 min each, concluding that work was "difficult, but possible" at 70 ppm, but that work was "impossible" at 100 ppm. Further, both subjects had to frequently leave the room because of the discomfort at the 100 ppm concentration. No other details about the exposure design were available and given the age of this report the reported exposure concentrations may be approximate.

Stevens et al. [1992] exposed 10 young adult asthmatic men and women to up to 1.8 ppm HCl for a 45-min exposure period that included a 15-min light exercise (walking treadmill) task in between two 15-min rest periods. Changes in lung function were measured and symptoms were assessed. Exposure to up to 1.8 ppm HCl did not cause any symptoms or lung function changes in asthmatic adults. This study was used as the basis for the ACGIH TLV ceiling value of 2 ppm [ACGIH 2021].

Reports of incidental exposure to high levels of HCl are limited. Boulet et al. [1988] reported on a 41-year-old nonsmoking man who rapidly developed bronchospasm following a roughly 1-hr exposure to pool cleaner containing HCl. He was later diagnosed with reactive airway dysfunction syndrome (RADS). As reviewed by NRC [2004], RADS is an asthma-like outcome of single, high-level exposures to chemical irritants, including HCl. This condition involves a nonspecific hyper-responsiveness toward chemical irritants. The Boulet et al. report was unable to estimate the concentration of HCl exposure that resulted in this outcome.

Bansal et al. [2011] reported on a case of a 56-year-old male patient who inhaled hydrochloric acid fumes for several minutes while doing metallurgical work and experienced cough, breathlessness, rapid breathing, and chest pain that caused him to seek treatment 4 hr following exposure. Despite treatment with corticosteroids and bronchodilators, the patient's condition worsened over 24 hr and required intubation up to seven days following exposure. The patient was discharged after ten days with normal breathing and chest radiography.

Liu and Cheng [2014] reported on two workers exposed to an unknown concentration of hydrochloric acid vapors for approximately 5 min during an incidental spill of a large amount of concentrated acid solution. Both were males aged between 43–47 years. Patients experienced cough, chest tightness, gasping, chest pain, rales, and sputum production and were treated with supplemental oxygen and corticosteroids. Symptoms improved in both patients after 3 days and patients were discharged after 7 days and 20 days with no abnormalities in chest radiographs.

Xia et al. [2019] reported on five workers who were exposed to hydrochloric acid vapor during a chemical tank cleaning incident. The workers were males aged between 51–65 years. Three workers were exposed for approximately 30 min while the other two were exposed twice for roughly 1 min. The exposure concentrations were not known. All five workers were hospitalized with varying degrees of adverse effects that included cough, chest tightness, sputum production, and shortness of breath. One patient developed acute respiratory distress syndrome (ARDS) and required intubation. Chest radiographs showed bilateral patch-like high-density shadows. All patients recovered with hospital stays ranging from 12–21 days. Chest radiographs showed no abnormalities at discharge and patients were able to return to work.

3.4.3 Animal Data

Acute exposure studies reporting irritation effects in animals have been reported in mice, rats, guinea pigs, and baboons. Reactions to exposures among different species and test concentrations were mixed, which appeared to reflect the water-soluble and corrosive properties of HCl. In mice and rats breathing normally, breathing frequency decreased with increasing HCl concentration consistent with the basis of the RD_{50} test in rodents [Barrow et al. 1977; Hartzell et al. 1985]. In baboons, breathing frequency was slightly increased during exposure [Kaplan et al. 1988].

Stavert et al. [1991] examined differences between rats exposed to HCl via the nose or mouth, finding that exposure by mouth resulted in increased minute volume relative to baseline. NIOSH interprets these observations to indicate that HCl is highly reactive with airway surfaces and rapidly absorbed, and that irritation effects vary based on how deeply HCl penetrates into the respiratory system. The available animal data are summarized below.

Barrow et al. [1977] exposed male Swiss-Webster mice to 0 ppm, 40 ppm, 99 ppm, 245 ppm, 440 ppm, or 943 ppm HCl for 10 min in groups of four. Breathing frequency was measured for 20 min starting at the introduction of the exposure. Respiratory rates decreased in a dose-dependent manner. The effect was apparent at all exposure concentrations above 40 ppm. The decrease in breathing frequency persisted during the 10-min post-exposure period. The authors calculated the RD_{50} to be 309 ppm with a 95% confidence range of 219–435 ppm based on a simple regression of percent decrease against log concentration. They predicted this concentration would be "intolerable and rapidly incapacitating" in humans, while 31 ppm (derived from one tenth of the RD_{50} concentration) was predicted to be "slightly irritating" to the eyes, nose, and throat.

Barrow et al. [1979] exposed male Swiss-Webster mice to HCl concentrations ranging from 20 to 20,000 ppm for 10 min via head-only inhalation. Respiratory rates averaged over the 10-min exposure were recorded and lung histology at 24 hr was investigated. HCl exposure caused a decrease in breathing frequency at 200 ppm with no effect at 50 ppm. The authors did not calculate an RD_{50} value but the concentration corresponding to a 50% decrease appeared to fall between 500 ppm and 1,000 ppm based on a graph of the data. Death occurred at 12,000 ppm and higher. At approximately 200 ppm, animals exhibited nasal epithelial ulcerations. Higher concentrations caused more extensive damage and necrosis of nasal mucosa. Ocular damage was observed at approximately 730 ppm in the form of moderate to marked clouding and inflammation. The study included a comparison experiment using thermal decomposition products of polyvinyl chloride, of which HCl is a major constituent. The results of exposure to these pyrolysis products were similar to HCl on a mass-to-volume basis.

Burleigh-Flayer et al. [1985] exposed male English smooth-haired guinea pigs to 0 ppm, 320 ppm, 680 ppm, 1,040 ppm, or 1,380 ppm HCl in groups of 4–8 for 30 min. Five animals in the two highest dose groups died within 15 days following exposure. Corneal opacity was seen beginning at 680 ppm. HCl exposure caused initial decreases in breathing frequency at all dose levels, which was followed by a transient increase at concentrations above 320 ppm that the authors attributed to pulmonary irritation of the lower airways and lung. A limited subset of animals exposed to 1,080 ppm were examined histologically, showing multifocal acute alveolitis and mild hemorrhage when examined 2 days after exposure.

Hartzell et al. [1985] exposed male Sprague-Dawley rats to 0 ppm, 200 ppm, 295 ppm, 784 ppm, 1,006 ppm, or 1,538 ppm in groups of three for 30 min and measured breathing frequency and minute volume. The 50-percent (median) effect levels for these endpoints were 560 ppm and 605 ppm, respectively. The effect profile of the 784 ppm group showed that maximal depression of respiration was achieved quickly, within 2 min, and plateaued for the rest of the 30-min exposure period.

Kaplan et al. [1985] exposed juvenile male baboons to 190 ppm, 810 ppm, 890 ppm, 940 ppm, 2,780 ppm, 11,400 ppm, 16,570 ppm, or 17,290 ppm HCl for 5 min before the animals completed an escape task, with one animal being tested at each concentration. The task was to press one of two levers designated by colored light cues to open a door and escape an electric shock given in a test chamber over a 30-second trial. Although all animals were able to escape the chamber, all concentrations above 190 ppm caused visible signs of irritation such as coughing, hypersalivation, blinking/rubbing eyes. Animals exposed to the two highest concentrations died in the following days with signs of pneumonia, pulmonary edema, tracheitis, and severe dyspnea. The same study also exposed male Sprague-Dawley rats in a similar experimental design (an escape task). All concentrations tested produced signs of severe irritation, with 11,800 ppm being the lowest dose. Exposure to concentrations of 15,250 ppm and greater caused immediate, persistent respiratory damage.

Kaplan et al. [1988] exposed anesthetized adult male baboons in groups of three to 0 ppm, 500 ppm, 5,000 ppm, or 10,000 ppm HCl for 15 min. Pulmonary functions were measured. Increasing concentrations of HCl increased breathing frequency by up to 2-fold, but tidal volume was unchanged. Arterial blood oxygen was decreased by 40% in the two highest dose groups, and this effect persisted for up to 10 min after the exposure ended. No differences in pulmonary function were found when animals were tested again 3 days and 3 months after exposure.

Stavert et al. [1991] exposed male F344 rats to 1,284 ppm HCl for 30 min via nose- or mouth-only inhalation. Rats exposed to HCl via the nose showed severe necrosis of nasal epithelium and minimal change in the trachea, whereas rats exposed via the mouth showed severe necrosis of tracheal epithelium. Breathing parameters were recorded during the exposures, where HCl-exposed rats showed an abrupt decrease in minute volume, which recovered to baseline within minutes before again declining for the remainder of the exposure. The approximate mean changes were a 6% decrease in minute volume, a 4% increase in breathing frequency, and a 7% decrease in tidal volume. In mouth-exposed rats, minute volume was increased 8% and tidal volume was increased 10%. The author estimated that because of the different pulmonary effects of exposure by mouth, mouth-exposed rats inhaled 23% more HCl than nose-exposed rats; 6% of the animals exposed to HCl via the nose and 46% exposed via mouth died within 24 hr following exposure.

Kaplan et al. [1993a] exposed male ICR mice to 500 ppm or 2,500 ppm, female Sprague-Dawley rats to 4,200 ppm, and male English smooth-haired guinea pigs to 500 ppm or 4,200 ppm HCl for 15 min in groups of six animals. Respiratory function was measured during the exposure, and respiratory tracts were assessed at 3-days and 3-months post-exposure. Mice exposed to 500 ppm or 2,500 ppm HCl showed 10% and 40% decreases in respiration, respectively. Four mice exposed to 500 ppm died during the 3-month follow-up, and all mice died following exposure to 2,500 ppm. On follow-up examination, animals in the high exposure group showed airway necrosis and lung edema, whereas mice exposed to 500 ppm appeared normal despite treatment-related mortality in that group. Rats exposed to 4,200 ppm HCl for 15 min experienced a 40% decrease in respiratory rate. Lungs of exposed rats were histologically normal at 3-months post-exposure. Guinea pigs exposed to 500 ppm or 4,200 ppm HCl experienced a 20% decrease in breathing frequency. Three animals in the high dose group died post-exposure showing pulmonary congestion, tracheitis, and desquamation of bronchiolar epithelia, and the survivors showed focal pneumonia and other respiratory lesions at 3-months post-exposure.

3.5 Cardiac and Hematological Effects

3.5.1 Overview

Exposure to HCl is not expected to be a specific cardiac or hematological hazard based on its chemistry. Any effects on hematology appear to be secondary to respiratory effects.

3.5.2 Human

No reports of hematological or cardiac effects in humans exposed to HCl were identified.

3.5.3 Animal

Kaplan et al. [1993a] observed fluctuations in arterial blood oxygen in female English guinea pigs and male Sprague-Dawley rats exposed to 4,200 ppm HCl for 15 min. These fluctuations were characterized by sharp, transient decreases followed by a rapid return to baseline or above baseline. These transient changes were particularly dramatic in guinea pigs. Arterial pH and CO2 were not affected in either species.

Kaplan et al. [1988, 1993b] observed loss of arterial blood oxygen in male baboons exposed to 5,000 ppm or 10,000 ppm HCl for 15 min. The partial pressure of arterial blood oxygen declined from 83 mm Hg to 44–47 mm Hg at both concentrations at the end of the exposure period and remained at these levels when measured after a 10-min recovery. Arterial blood oxygen in exposed baboons had returned to normal when measured after a 3-day recovery. The authors found no significant impact on blood O2 in baboons exposed to 500 ppm, and no changes in blood CO2 or pH were observed in any exposure group. The authors attributed the blood oxygen effects at high concentrations to uneven ventilation caused by pulmonary edema and small airway constriction.

3.6 Other Relevant Health Effects

No other target organ effects arising from acute inhalation exposures were identified.

4 Determination of IDLH Value

4.1 Selection of Critical Data

The immediately dangerous effects that can be caused by acute exposure to HCl in humans are irritation and corrosion of the eyes and especially of the respiratory tract, and death. HCl exposure is not an explosive or asphyxiant hazard, does not have acute neurological effects (impaired awareness or coordination), and does not induce any specific cardiac or other target organ effects.

Although HCl is an eye and respiratory irritant, evidence that HCl causes immediate irritation effects severe enough to prevent escape from contamination is mixed. Multiple studies in rodents, guinea pigs, and primates demonstrate that animals exposed to high levels of HCl experience irritation symptoms without being immediately incapacitated, even at concentrations that result in lethality after the exposure [Kaplan et al. 1985]. The body of data as a whole suggest that the immediately dangerous effects of acute HCl exposure are due to airway reactivity as well as an accumulation of corrosive damage to the eye and respiratory surfaces. It is unclear whether HCl is an eye irritant in the sense of causing immediate stinging and tearing up in a way that would impair sight. Based on the available data, NIOSH considered the following endpoints as a potential basis for the IDLH value:

Eye Irritation: Kaplan et al. [1985] reported signs of severe irritation in baboons exposed to extremely high HCl levels for 5 min, including scratching and rubbing of the eyes at concentrations above 800 ppm, but the animals could see well enough to complete a simple escape task. The lowest LOAEL identified specific to eye irritation is 680 ppm that caused corneal opacity observed in guinea pigs exposed to HCl for 30 min [Burleigh-Flayer et al. 1985]. The NOAEL for this effect was 320 ppm, and the exposure concentrations in the study were confirmed via analysis. Available human data (summarized in Section 3.4.1) refer to levels exceeding 100 ppm as being unpleasant or intolerable to human subjects, but do not specifically refer to eye irritation symptoms.

Respiratory Irritation: Rodent respiratory depression assays are available for making a quantitative estimate of immediately dangerous respiratory irritation thresholds in humans. The Barrow et al. [1977] study found an RD_{50} of 309 ppm after a 10-min exposure in mice; this concentration was confirmed via analysis and was the most sensitive RD_{50} value identified.

Lethality (LC_{50}): The most sensitive 30-min LC_{50} reported was 1,341 ppm by Hartzell et al. [1988]. This value was obtained from groups of six male guinea pigs exposed to six concentrations ranging from 900 to 2,347 ppm and observed for 14 days post exposure. The authors noted that guinea pigs appear roughly three times more susceptible to the lethal effects of HCl inhalation compared with rats. Wohlslagel et al. [1976] and Vernot et al. [1977] also reported 60-min LC_{50} values of 1,108 and 1,110 ppm, respectively, in female and male mice, but the guinea pig 30-min LC_{50} of 1,341 ppm stands as the most sensitive LC_{50} after factoring in time adjustment. This exposure concentration was verified by the authors via analytical techniques that were reported in detail.

Furthermore, the Stavert et al. [1991] study in rats observed an almost 50% mortality rate 24 hr following a 30-min exposure to 1,300 ppm when rats were exposed via the mouth instead of the nose. Considering that the most sensitive animal models (both guinea pigs breathing normally and rats breathing through an orotracheal tube) both show approximately 50% lethality at similar concentrations of HCl, the LC_{50} of 1,341 ppm reported by Hartzell et al. [1988] was selected to derive a potential IDLH value based on lethality.

4.2 Application of Time Scaling

The NOAEL for eye irritation was obtained from a 30-min acute exposure and did not need adjustment.

The RD_{50} value of 309 ppm reported by Barrow et al. [1977] was obtained from 10-min exposures in mice, during which the maximum decrease in respiratory rates was observed very quickly, within minutes of exposure. This is consistent with other reports discussed in Section 3.4.2 that observed rapid attainment of maximal respiratory depression within minutes of exposure, after which respiration plateaus or recovers. Given that these studies did not observe a consistent relationship between respiratory depression and increased exposure time, no time adjustment was made for the RD_{50} of 309 ppm.

The LC_{50} value of 1,341 ppm was obtained from a 30-min exposure and did not need adjustment.

4.3 Application of Uncertainty Factors

Eye Irritation

The UF for eye irritation is based on extrapolation of corneal opacity in guinea pigs exposed to HCl gas to human outcomes. Because the effect is occurring at the surface of the eye, no adjustment for toxicokinetic variation is needed for animal or interindividual extrapolation is needed. It is not clear whether there are toxicodynamic differences that would complicate the extrapolation of this outcome to humans. To account for any differences between guinea pigs and humans, a UF of 3 is applied. Because the effect level being adjusted is a no-effect level and significant variation in susceptibility to chemical damage to the cornea is not expected among humans, the total UF applied is 3.

Respiratory Irritation

The UF for respiratory irritation is based on extrapolation of the rodent RD_{50} value to humans. The RD_{50} represents an effect level presumed to be strongly irritating to humans [Alarie 1981]. It is not known whether effects at this level fall above or below the definition of immediately dangerous. To account for this uncertainty, a factor of 3 is applied to extrapolate to a level presumed to be low enough that human workers would not be incapacitated. A further factor to account for human variability was not used because significant variability in these estimated effects are not expected among healthy working-age humans.

Lethality

The uncertainty factor for lethality is based on the severity of the effect and extrapolation from guinea pig to human. Death due to HCl exposure is attributable to frank damage to the respiratory tract. Because HCl is rapidly absorbed and reacts locally in tissue, interspecies and interindividual differences in HCl hydrolysis, metabolism and clearance among guinea pigs and humans, respectively, are expected to have minimal impact on local tissue effects. There is still considerable uncertainty in extrapolation from guinea pig to human because of differences in respiratory anatomy, so a full factor of 10 is applied to account for interspecies extrapolation [NIOSH 2013]. A further factor of 3 is applied to account for the severity of the effect as well as the potential that increased exertion (i.e., during an evacuation scenario) may make individuals more susceptible to HCl toxicity as demonstrated in guinea pigs [Malek and Alarie 1989]. The total factor is 30.

Table 4.1: Potential IDLH Values Based on Immediately Dangerous Health Outcomes of Hydrogen Chloride Exposure

Health outcome	Immediately dangerous effect level (ppm)		30-Min adjusted value (ppm)	Uncertainty factor	Candidate IDLH value (ppm)
Eye irritation	320	NOAEL	320	3	107
Respiratory irritation	309	RD_{50}	309	3	103
Lethality	1,341	LC_{50}	1,341	30	45

4.4 Final IDLH Calculation

Table 4.1 summarizes the immediately dangerous health outcomes of HCl exposure and potential IDLH values. NIOSH set the IDLH value based on the 45 ppm limit value for lethality. This value was chosen because it is both the most sensitive value and is based on the most severe effect. This value is supported by the limited collection of older references that describe concentrations in the range of 10–70 ppm as being poorly tolerated by human subjects without being immediately hazardous [Henderson and Haggard 1943; Jacobs 1967; and in Matt 1889, as cited in NRC 1998], and is also approximately seven times lower than the RD_{50} value of 309 ppm identified in mice (Barrow et al. 1977).

In summary, NIOSH set the IDLH value for HCl at 45 ppm based on the risk of immediately dangerous and/or lethal respiratory effects in humans assumed to be in a state of exertion.

References

ACGIH [2021]. Annual TLVs® (threshold limit values) and BEIs® (biological exposure indices) booklet. Cincinnati, OH: ACGIH Signature Publications.

AIHA [1989]. Odor thresholds for chemicals with established occupational health standards, p. 20. Fairfax, VA: American Industrial Hygiene Association.

AIHA [2016]. Emergency response planning guidelines (ERPG) and workplace environmental exposure levels (WEEL) handbook. Fairfax, VA: American Industrial Hygiene Association Press.

Alarie Y [1981]. Dose-response analysis in animal studies: prediction of human responses. Environ Health Perspect *42*:9–13, https://doi.org/10.1289/ehp.81429.

Alarie Y, Anderson RC [1979]. Toxicologic and acute lethal hazard evaluation of thermal decomposition products of synthetic and natural polymers. Toxicol Appl Pharmacol *51*(2):341–362, https://doi.org/10.1016/0041-008X(79)90476-9.

ATSDR [2002]. ATSDR fact sheet: hydrogen chloride. U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry, https://www.atsdr.cdc.gov/toxfaqs/tfacts173.pdf.

Babrauskas V, Levin BC, Gann RG [1987]. A new approach to fire toxicity data for hazard evaluation. Fire J *81*(2):22–28.

Bansal DP, Ambegaonkar R, Radhika P, Sharma M [2011]. ARDS following inhalation of hydrochloric acid. J Assoc Physicians India *59*:115–117.

Barrow CS, Alarie Y, Warrick JC, Stock MF [1977]. Comparison of the sensory irritation response in mice to chlorine and hydrogen chloride. Arch Environ Health *31*:68–76. https://doi.org/10.1080/00039896.1977.10667258

Barrow CS, Lucia H, Alarie Y [1979]. A comparison of the acute inhalation toxicity of hydrogen chloride versus the thermal decomposition products of polyvinylchloride. J Combust Toxicol *6*:3–12.

Boulet L-P [1988]. Increases in airway responsiveness following acute exposure to respiratory irritants: reactive airway dysfunction syndrome or occupational asthma? Chest *94*(3):476–481, https://doi.org/10.1378/chest.94.3.476.

Burleigh-Flayer H, Wong KL, Alarie Y [1985]. Evaluation of the pulmonary effects of HCl using CO2 challenges in guinea pigs. Fundam Appl Toxicol *5*(5):978–985, https://doi.org/10.1016/0272-0590(85)90179-4.

Darmer KI, Kinkead ER, DiPasquale LC [1974]. Acute toxicity in rats and mice exposed to hydrogen chloride gas and aerosols. Am Ind Hyg Assoc J *35*(10):623–631, https://doi.org/10.1080/0002889748507082.

Downs JW, Wills BK, Cumpston KL, & Rose SR [2021]. Descriptive epidemiology of clinically significant occupational poisonings, United States, 2008–2018. Clinical Toxicol *59*(12): 1259–1263, https://doi.org/10.1080/15563650.2021.1892717.

ECHA [2022]. REACH chemical information dossier: hydrogen chloride, https://echa.europa.eu/registration-dossier/-/registered-dossier/15859.

EPA [2022]. Integrated Risk Information System (IRIS): glossary. Washington, DC: Environmental Protection Agency, https://www.epa.gov/iris/iris-glossary.

Esposito FM, Alarie Y [1988]. Inhalation toxicity of carbon monoxide and hydrogen cyanide released during the thermal decomposition of polymers. J Fire Sci *6*(3):195–239, https://doi.org/10.1177/073490418800600303.

Flury F, Zernick F [1931]. Noxious gases, vapors, mist, smoke, and dust. [Article in in German.] Berlin, Germany: Verlag von Julius Springer.

Hartzell GE, Stacy HH, Switzer WG, Priest DN, Packham SC [1985]. Modeling of toxicological effects of fire gases: IV. Intoxication of rats by carbon monoxide in the presence of an irritant. J Fire Sci *3*(4):263–279. https://doi.org/10.1177/073490418500300403

Hartzell GE, Grand AF, Switzer WG [1987]. Modeling of toxicological effects of fire gases: VI. Further studies on the toxicity of smoke containing hydrogen chloride. J Fire Sci 5:368–391. https://doi.org/10.1177/073490418700500602

Hartzell GE, Grand AF, Switzer WG [1988]. Modeling of toxicological effects of fire gases: VII. Studies on evaluation of animal models in combustion toxicology. J Fire Sci 6:411–431.

Hartzell GE, Grand AF, Switzer WG [1990]. Toxicity of smoke containing hydrogen chloride. In: Nelson GL ed. Fire and polymers. Chapt. 2, pp. 12–20. ACS Symposium Series, Vol. 425. New York: Oxford University Press, https://doi.org/10.1021/bk-1990-0425.ch002

Henderson Y, Haggard HW [1943]. Hydrochloric acid (hydrogen chloride). In: Noxious gases and principles of respiration influencing their action, 2nd Rev. ed., pp. 126–127. New York: Reinhold Publishing.

Higgins EA, Fiorca V, Thomas AA, Davis HV [1972]. Acute toxicity of brief exposures to HF, HCl, NO2 and HCN with and without CO. Fire Technol *8*(2):120–130, https://doi.org/10.1007/BF02590576.

IARC [1992]. IARC monographs on the evaluation of the carcinogenic risk to humans. Group 3, hydrochloric acid. Vol. 54. Lyon, France: World Health Organization, International Agency for Research on Cancer, https://inchem.org/documents/iarc/vol54/03-hydrochloric-acid.html#:~:text=Human%20Carcinogenicity%20Data.

IFA [2019]. GESTIS: database on hazardous substances. Bonn, Germany: Institut für Arbeitsschutz der Deutschen Gesetzlichen Unfallversicherung, https://www.dguv.de/ifa/gestis/gestis-stoffdatenbank/index-2.jsp.

Jacobs MB [1967]. Hydrochloric acid and chlorides. In: The analytical toxicology of industrial inorganic poisons, pp. 635–641. New York: Interscience Publishers.

Kaplan HL, Anzeuto A, Switzer WG, Hinderer RK [1988]. Effects of hydrogen chloride on respiratory response and pulmonary function of the baboon. J Toxicol Environ Health *23*(4):473–493, https://doi.org/10.1080/15287398809531129.

Kaplan HL, Grand AF, Switzer WG, Mitchell DS, Rogers WR, Hartzell GE [1985]. Effects of combustion gases on escape performance of the baboon. J Fire Sci *3*(4):228–244, https://doi.org/10.1177/073490418500300401.

Kaplan HL, Switzer WG, Hinderer RK, Anzueto A [1993a]. Studies of the effects of hydrogen chloride and polyvinylchloride (PVC) smoke in rodents. J Fire Sci *11*(6):512–552, https://doi.org/10.1177/073490419301100603.

Kaplan HL, Switzer WG, Hinderer RK, Anzueto A [1993b]. A study on the acute and long-term effects of hydrogen chloride on respiratory response and pulmonary function and morphology in the baboon. J Fire Sci *11*(6):459–484, https://doi.org/10.1177/073490419301100601.

Kirsch H, Drabke P [1982]. Evaluation of biological effects of hydrogen chloride. [Article in German.] Z Gesamte Hyg *28*(2):107–109.

Liu S, Cheng W [2014]. Two cases of acute hydrochloric acid mist inhalation poisoning. [Article in Chinese.] Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi 32(3):201.

Malek DE, Alarie Y [1989]. Ergometer within a whole-body plethysmograph to evaluate performance of guinea pigs under toxic atmospheres. Toxicol Appl Pharmacol *101*(2):340–355. https://doi.org/10.1016/0041-008X(89)90282-2.

NIOSH [1994]. Documentation for immediately dangerous to life or health concentrations (IDLHs): hydrogen chloride. Cincinnati, OH: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, https://www.cdc.gov/niosh/idlh/7647010.html.

NIOSH [2004]. NIOSH respirator selection logic. By Bollinger N. Cincinnati, OH: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 2005-100, https://www.cdc.gov/niosh/docs/2005-100/default.html.

NIOSH [2013]. Current intelligence bulletin 66: derivation of immediately dangerous to life or health (IDLH) Values. Cincinnati, OH: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 2014-100, https://www.cdc.gov/niosh/docs/2014-100/.

NIOSH [2019]. Hydrogen Chloride: Overview. https://www.cdc.gov/niosh/topics/hydrogen-chloride/default.html. Accessed October 17, 2023.

NIOSH [2020a]. Current intelligence bulletin 69: NIOSH practices in occupational risk assessment. By Daniels RD, Gilbert SJ, Kuppusamy SP, Kuempel ED, Park RM, Pandalai SP, Smith RJ, Wheeler MW, Whittaker C, Schulte PA. Cincinnati, OH: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health. DHHS (NIOSH) Publication No. 2020-106, (Revised 03/2020), https://doi.org/10.26616/NIOSHPUB2020106revised032020.

NIOSH [2020b]. NIOSH pocket guide to chemical hazards (online). Barsan ME, ed. Cincinnati, OH: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 2005-149, https://www.cdc.gov/niosh/npg/default.html. Accessed November 28, 2022.

NLM [2022]. PubChem compound summary for hydrogen chloride, https://pubchem.ncbi.nlm.nih.gov/compound/313. Date accessed: November 28, 2022.

NRC [1998]. Assessment of exposure-response functions for rocket-emission toxicants. National Academies Press, https://doi.org/10.17226/6205.

NRC [2004]. Acute exposure guideline levels (AEGLs) for selected airborne chemicals. Vol. 4, pp. 77–122. Washington, DC: The National Academies Press. https://www.epa.gov/sites/default/files/2014-11/documents/tsd52.pdf.

OSHA [2019]. Occupational safety and health standards. 29 CFR § 1910. Subpart Z: toxic and hazardous substances. Washington, DC: Occupational Safety and Health Administration, http://www.osha.gov/pls/oshaweb/owadisp.show_document?p_table=standards&p_id=9992.

Sakurai T [1989]. Toxic gas tests with several pure and mixed gases using mice. J Fire Sci *7(*1):22–77, https://doi.org/10.1177/073490418900700102.

Stavert DM, Archuleta DC, Behr MJ, Lehnert BE [1991]. Relative acute toxicities of hydrogen fluoride, hydrogen chloride, and hydrogen bromide in nose- and pseudo-mouth-breathing rats. Fundam Appl Toxicol 16(4):636–655, https://doi.org/10.1016/0272-0590(91)90152-T.

Stevens B, Koenig JQ, Rebolledo VN, Hanley QS, Covert DS [1992]. Respiratory effects from the inhalation of hydrogen chloride in young adult asthmatics. J Occup Med *34*(9):923–929.

ten Berge WF, Zwart A, Appelman LM [1986]. Concentration-time mortality response relationship of irritant and systematically acting vapours and gases. J Hazard Mater *13*(3):301–309, https://doi.org/10.1016/0304-3894(86)85003-8.

Vernot EH, MacEwen JD, Haun CC, Kinkead ER [1977]. Acute toxicity and skin corrosion data for some organic and inorganic compounds and aqueous solutions. Toxicol Appl Pharmacol *42*(2):417–423, https://doi.org/10.1016/0041-008x(77)90019-9.

Wohlslagel J, DiPasquale LC, Vernot EH [1976]. Toxicity of solid rocket motor exhaust: effects of HCl, HF, and alumina on rodents. J Combust Toxicol *3*:61–70.

Xia ML, Lou YF, Ma WJ [2019]. Clinical analysis of 5 cases of acute poisoning by inhalation of hydrochlogen chloride. [Article in Chinese.] Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi *37*(11):855–857, https://doi.org/10.3760/cma.j.issn.1001-9391.2019.11.015.

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DHHS (NIOSH) Publication No. 2025–110 (revised 06/2025)