

HYDROGEN CYANIDE

CAS number: 74-90-8

Synonyms: Hydrocyanic acid, prussic acid, formonitrile

Chemical formula: HCN

Structural formula:

Workplace exposure standard (amended)

TWA: 0.9 ppm (1 mg/m³)

STEL: —

Peak limitation: 4.7 ppm (5 mg/m³)

Notations: Sk.

IDLH: 50 ppm

Sampling and analysis:

Recommendation and basis for workplace exposure standard

A TWA of 0.9 ppm (1 mg/m³) is recommended to protect for chronic neurological symptoms and thyroid enlargement in exposed workers.

A peak limitation of 4.7 ppm (5 mg/m³) is recommended to protect for immediate and severe health effects (death, coma, respiratory failure) in workers exposed at the workplace.

Discussion and conclusions

Hydrogen cyanide and cyanide salts have very similar toxicological endpoints and effects acting primarily through the release of the cyanide ion (ACGIH, 2018; DFG, 2001). Numerous reports identify acute toxicity relating to accidental poisoning with symptoms including headaches, light-headedness and loss of consciousness, which are reversed after removal from exposure.

A study in workers exposed at 4.2 to 12.4 ppm in the breathing zone reported incidence of headache, weakness, changes in taste and smell, irritation of the throat, vomiting and effort dyspnoea (ACGIH, 2018; DFG, 2001; HCOTN, 2001). Nasal irritation and septum ulceration were reported in workers exposed to approximately 5 ppm (5.1 mg/m³) (ACGIH, 2018).

The US EPA (2010) reported that exposure to low concentrations of cyanide in workers can cause thyroid effects and central nervous system symptoms. A LOAEL of 6.4 ppm (7.07 mg/m³) was reported for observed thyroid effects from a long-term occupational study involving 36 male workers and a control group of 20.

Based on the evidence above, a TWA of 0.9 ppm (1 mg/m³) is recommended to protect for chronic effects with a peak limitation of 4.7 ppm (5 mg/m³) recommended to protect acutely exposed workers.

Recommendation for notations

Not classified as a carcinogen according to the Globally Harmonized System of Classification and Labelling of Chemicals (GHS).

Not classified as a skin sensitiser or respiratory sensitiser according to the GHS.

A skin notation has been assigned as evidence indicates rapid absorption through skin.

DRAFT

APPENDIX

Primary sources with reports

Source	Year set	Standard
SWA	1986	Peak: 10 ppm (11 mg/m³)
ACGIH	2001	TLV-Ceiling: 4.7 ppm (5 mg/m³)
<p>TLV-Ceiling, measured as the cyanide ion (CN⁻), recommended to minimise the potential for headache, nausea, nasal, throat, and pulmonary irritation and enlargement of the thyroid gland.</p> <p>Generally accepted that hydrogen cyanide and cyanide salts act by the same mechanism, namely the release of the cyanide ion.</p> <p>Summary of data:</p> <p>Human data:</p> <ul style="list-style-type: none"> One study reports acute toxicity related to accidental poisoning resulting in headaches, light-headedness and loss of consciousness, which reversed after removal from exposure Severe acute exposure may result in death; however, if death did not occur, recovery was complete and prompt No ill effects reported by workers exposed to concentrations in the order of 10 ppm (no further information) Nasal irritation and septum ulceration in workers exposed to ≈5 ppm (5.1 mg/m³; no further information) A study in workers exposed to a range of concentrations from 4.2–12.4 ppm in the breathing zone (7 yr) reported increased incidence in exposed workers (compared to control) of headache, weakness, changes in taste and smell, irritation of the throat, vomiting and effort dyspnoea A study in 23 male workers reported complaints typical of hydrogen cyanide poisoning associated with airborne concentrations of cyanide at 0.8 mg/m³ (breathing zone) and 0.2 mg/m³ (general work area). <p>Animal data:</p> <ul style="list-style-type: none"> LC₅₀: 503 ppm (rats, 5 min) and 323 ppm (mice, 5 min) Only slight differences observed in acute toxicities of KCN and HCN (rabbits). <p>Rapidly absorbed through the skin warranting skin notation.</p> <p>Insufficient data available to recommend sensitiser or carcinogen notations.</p>		
DFG	2001	MAK: 1.9 ppm (2.1 mg/m³)
<p>MAK recommended to reduce symptoms such as headaches, weakness, dizziness, throat irritation, dyspnoea, thyroid enlargement and an increase in thiocyanate excretion in the urine in exposed workers.</p> <p>Summary of additional data:</p> <ul style="list-style-type: none"> Exposures (10 ppm, 8 h) may produce the metabolite thiocyanate which is in region of the LOEL for goitre development Detoxification in humans is reported as 0.1–1.0 mg/kg/h The MAK derived on the basis that a worker exposed to 2 mg/m³ takes up a dose of 20 mg (70 kg worker, inhaling 10 m³ per 8 h with 100% absorption) Dermal LD₅₀: 100 mg/kg (humans, no further details) 		



Source	Year set	Standard
<ul style="list-style-type: none"> Dermal LD₅₀: 6.90 mg/kg (rabbits). 		
SCOEL	2010	TWA: 0.9 ppm (1 mg/m³); STEL 4.5 ppm (5 mg/m³)
<p>TWA and STEL expected to prevent acute and chronic adverse health effects such as thyroid enlargement (goitre) and neurotoxicity symptoms (visual disturbances, convulsions, paresis).</p> <p>Summary of additional data:</p> <ul style="list-style-type: none"> TWA derivation: LOAEL 4.2 ppm (see ACGIH) coupled with an assessment factor of 5 to account for absence of a defined dose-response relationship No evidence for carcinogenicity or effects on reproduction. 		
OARS/AIHA	NA	NA
No report.		
HCOTN	2002	TWA: 0.9 ppm (1 mg/m³); Ceiling: 9 ppm (10 mg/m³)
<p>TWA and Ceiling recommended to protect for both chronic (headaches, weakness, change in taste and smell, giddiness, irritation of throat, vomiting, effort dyspnoea, lachrymation, precordial pain, salivation, disturbances of accommodation and psychosis) and acute effects (death, coma, respiratory failure).</p> <p>Summary of additional data:</p> <ul style="list-style-type: none"> Ceiling derivation: LOAEL 20 mg/m³ (no further information) coupled with an assessment factor of 2 to account for steep dose-response relationship High skin permeability warrants skin notation HCN and salts (KCN and NaCN) have very similar toxicological endpoints and effects, and therefore should be regulated together. 		

Secondary source reports relied upon

Source	Year	Additional information
US EPA	✓ 2010	<ul style="list-style-type: none"> Reported that exposing workers to low concentrations can cause thyroid effects and CNS symptoms Chronic or sub chronic inhalation studies in experimental animals were not found. LOAEL: 6.4 ppm (7.07 mg/m³); long-term occupational study; observed thyroid effects; 36 male workers exposed; 20 males in control group. LOAEL was adjusted for continuous exposure to 2.5 mg/m³ and used as the PoD for derivation of RfC (8 x 10⁻⁴ mg/m³). A total UF of 3,000 was applied to the PoD: <ul style="list-style-type: none"> 10 for extrapolation of a LOAEL to a NOAEL 3 for extrapolation from a sub chronic to chronic exposure duration 10 for human intraspecies variability 10 to account for database deficiencies.



Carcinogenicity — non-threshold based genotoxic carcinogens

Is the chemical mutagenic?

No

Notations

Source	Notations
SWA	Skin
HCIS	—
NICNAS	NA
EU Annex	NA
ECHA	—
ACGIH	Skin
DFG	H (skin)
SCOEL	Skin
HCOTN	Skin
IARC	NA
US NIOSH	NA

NA = not applicable (a recommendation has not been made by this Agency); — = the Agency has assessed available data for this chemical but has not recommended any notations

Skin notation assessment

Calculation	
Adverse effects in human case study:	no
Dermal LD ₅₀ ≤ 1000 mg/kg:	yes
Dermal repeat-dose NOAEL ≤ 200 mg/kg:	
Dermal LD ₅₀ /Inhalation LD ₅₀ < 10:	yes
<i>In vivo</i> dermal absorption rate > 10%:	
Estimated dermal exposure at WES > 10%:	
consider assigning a skin notation	

IDLH

Is there a suitable IDLH value available?

Yes

Additional information

Molecular weight:	27.03
Conversion factors at 25°C and 101.3 kPa:	1 ppm = Number mg/m ³ ; 1 mg/m ³ = Number ppm
This chemical is used as a pesticide:	✓



Molecular weight:	27.03
This chemical is a biological product:	<input type="checkbox"/>
This chemical is a by-product of a process:	<input checked="" type="checkbox"/>
A biological exposure index has been recommended by these agencies:	<input type="checkbox"/> ACGIH <input type="checkbox"/> DFG <input type="checkbox"/> SCOEL

Workplace exposure standard history

Year	Standard
Click here to enter year	

References

American Conference of Industrial Hygienists (ACGIH®) (2018) TLVs® and BEIs® with 7th Edition Documentation, CD-ROM, Single User Version. Copyright 2018. Reprinted with permission. See the [TLVs® and BEIs® Guidelines section](#) on the ACGIH website.

Deutsche Forschungsgemeinschaft (DFG) (2001) Hydrogen cyanide, potassium cyanide and sodium cyanide– MAK value documentation.

European Chemicals Agency (ECHA) (2012) Directive 98/8/EC concerning the placing biocidal products on the market Inclusion of active substances in Annex I or I A to Directive 98/8/EC Assessment Report

EU Scientific Committee on Occupational Exposure Limits (SCOEL) (2011) Recommendation from the Scientific Committee on Occupational Exposure Limits for cyanide (HCN, KCN, NaCN). SCOEL/SUM/115.

Health Council of the Netherlands (HCOTN) (2002) Hydrogen cyanide, sodium cyanide, and potassium cyanide. Health-based recommended occupational cancer risk values. The Hague: Health Council of the Netherlands; publication no. 2012/32.

US Environmental Protection Agency (US EPA) (2010) Toxicological Review of Hydrogen Cyanide and Cyanide Salts. EPA/635/R-08/016F.

US National institute for Occupational Safety and Health (NIOSH) (1994) Immediately dangerous to life and health concentrations – hydrogen cyanide.