

# CARBON DISULFIDE

**CAS number:** 75-15-0

**Synonyms:** Carbon disulphide, carbon bisulfide

**Chemical formula:** CS<sub>2</sub>

**Structural formula:**

## Workplace exposure standard (amended)

**TWA:** 1 ppm (3.13 mg/m<sup>3</sup>)

**STEL:** —

**Peak limitation:** —

**Notations:** Sk.

**IDLH:** 500 ppm

**Sampling and analysis:** The recommended value is readily quantifiable through currently available sampling and analysis techniques.

## Recommendation and basis for workplace exposure standard

A TWA of 1 ppm (3.13 mg/m<sup>3</sup>) is recommended to protect for the onset of adverse nervous system effects in exposed workers and is considered protective of other adverse health endpoints including cardiotoxicity.

## Discussion and conclusions

The critical effects in humans that are associated with exposure to carbon disulfide are neurotoxicity and cardiotoxicity. This has been demonstrated in a large range of observational studies, supported by evidence from experimental animal studies for neurotoxicity outcomes (ACGIH, 2018; DFG, 2004; HCOTN, 2011).

A LOAEL of 15 mg/m<sup>3</sup> (5 ppm) for incidence of ischaemic findings was reported in a prospective cohort study in Japanese workers (HCOTN, 2011). Motor nerve induction deficits and other adverse nervous system effects were reported to appear following exposures slightly above 1 ppm (ACGIH, 2018); which forms the basis of the TWA recommendation. A TWA of 1 ppm is considered protective of neurotoxic effects that may begin to develop at 3 ppm (9 mg/m<sup>3</sup>) (HCOTN, 2011).

Evidence was provided reporting human observations of absorption via dermal route leading to peripheral nerve damage and systemic toxicity (ACGIH, 2018; HCOTN, 2011).

## 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 is recommended based on sufficient evidence in humans demonstrating systemic effects following dermal exposure.

DRAFT

## APPENDIX

### Primary sources with reports

Source	Year set	Standard
<b>SWA</b>	<b>Year</b>	<b>TWA: 10 ppm (31 mg/m<sup>3</sup>)</b>
<b>ACGIH</b>	<b>2006</b>	<b>TLV-TWA: 1 ppm (3.13 mg/m<sup>3</sup>)</b>
<p>TLV-TWA recommended to protect for neurological endpoints and other adverse effects in all organ systems in exposed workers exposed.</p> <p>Summary of data:</p> <p>Human data:</p> <ul style="list-style-type: none"> <li>• Four worker exposure studies provide evidence of reduced motor conduction velocities (range of exposures 0.2–65.7 ppm)</li> <li>• Odour threshold reported at 0.1–0.2 ppm</li> <li>• Acute exposures at 500–1,000 ppm resulted in psychiatric disturbances (no further information provided)</li> <li>• Acute exposure at 5,000 ppm resulted in CNS depression, coma, respiratory paralysis and death (no further information provided)</li> <li>• Not all literature reviewed provided defined exposure-dose information for CNS symptoms</li> <li>• Physiological responses (adverse nervous system effects) resulting from exposure appear to occur at slightly above 1 ppm.</li> </ul> <p>Animal data:</p> <ul style="list-style-type: none"> <li>• No dermal LD50 reported</li> <li>• LC50: 220 ppm (mice; 1 h) and 3,200 and 8,000 ppm (rats and mice; 2 h)</li> <li>• No animal data available for eye and skin irritation and sensitisation.</li> </ul> <p>Results from <i>in vitro</i> studies provide little evidence of genotoxicity.</p> <p>Absorption via dermal route reported peripheral nerve damage and systemic toxicity in workers.</p>		
<b>DFG</b>	<b>2004</b>	<b>MAK: 5 ppm (16 mg/m<sup>3</sup>)</b>
<p>Provisional MAK assigned to be protective of neurotoxicity and cardiotoxicity that can occur at concentrations &lt;10 ppm. This MAK is accompanied by a recommendation for further research.</p> <p>Summary of additional data:</p> <ul style="list-style-type: none"> <li>• NOEL: 4 ppm (humans); based on 40 yr exposure; uncertainty in exposure concentrations noted</li> <li>• Endocrine effects reported in chronic human exposure studies; LH and FSH reduction with increasing exposure time; at 4.6±1.5 ppm (14.4±4.62 mg/m<sup>3</sup>)</li> <li>• Neurological effects on the eye and ototoxic effects reported (enhanced hearing loss at low frequencies) in humans</li> <li>• No carcinogenicity or genotoxicity data reported or reviewed</li> <li>• No reports available regarding skin or respiratory tract sensitisation; no notations assigned.</li> </ul>		



Source	Year set	Standard
<b>SCOEL</b>	<b>2008</b>	<b>TWA: 5 ppm (16 mg/m<sup>3</sup>)</b>
<p>TWA is assigned to protect for neurotoxicity and cardiotoxicity and is considered to be protective for other reported health effects.</p> <ul style="list-style-type: none"> <li>• Considered a threshold of 10 ppm (30 mg/m<sup>3</sup>) for earliest non-clinical changes in human studies and applied an uncertainty factor of 2 to account for seriousness of effects</li> <li>• Cardiotoxicity reported in humans at ≈63 mg/m<sup>3</sup> (20 ppm)</li> <li>• Local effects on skin and mucous membranes on dermal contact; blistering, ulceration, degradation of sweat glands and local nerve endings reported in animal studies</li> <li>• No investigations into dermal contact in humans.</li> </ul>		
<b>OARS/AIHA</b>	<b>NA</b>	<b>NA</b>
No report		
<b>HCOTN</b>	<b>2011</b>	<b>TWA: 2 ppm (5 mg/m<sup>3</sup>)</b>
<p>The TWA is assigned to protect for neurotoxicity and cardiotoxicity in exposed workers.</p> <p>Summary of additional data:</p> <ul style="list-style-type: none"> <li>• TWA of 5 mg/m<sup>3</sup> (2 ppm) derived by application of a factor of 3 to a reported LOAEL of 15 mg/m<sup>3</sup> (5 ppm) (prospective cohort study, incidence of ischaemic findings)</li> <li>• TWA is below the reported concentration at which neurotoxic effect may develop (9 mg/m<sup>3</sup>; 3 ppm)</li> <li>• A skin notation assigned based on evidence from an experimental study reporting absorption of liquid carbon disulfide.</li> </ul>		

## Secondary source reports relied upon

Source	Year	Additional information
NICNAS	✓ 2014	<ul style="list-style-type: none"> <li>• Median animal inhalational data, LC<sub>50</sub> ≈10.35 mg/L</li> <li>• NOAEC: 155.8 mg/m<sup>3</sup> (S-D rats; 6h/d. 5d/wk for at least 89 d)</li> <li>• Reproductive effects reported in animal and human studies.</li> </ul>

## Carcinogenicity — non-threshold based genotoxic carcinogens

Is the chemical mutagenic?

No

## Notations

Source	Notations
SWA	Skin
HCIS	-
NICNAS	-
EU Annex	-
ECHA	-



Source	Notations
ACGIH	Carcinogenicity Category A4; Skin
DFG	H (skin)
SCOEL	Skin
HCOTN	Skin

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: **yes**

Dermal LD<sub>50</sub> ≤ 1000 mg/kg:

Dermal repeat-dose NOAEL ≤ 200 mg/kg:

Dermal LD<sub>50</sub>/Inhalation LD<sub>50</sub> < 10:

*In vivo* dermal absorption rate > 10%:

Estimated dermal exposure at WES > 10%:

**a skin notation is warranted**

## IDLH

Is there a suitable IDLH value available? **Yes**

## Additional information

Molecular weight:	76.13
Conversion factors at 25°C and 101.3 kPa:	1 ppm = Number mg/m <sup>3</sup> ; 1 mg/m <sup>3</sup> = Number ppm
This chemical is used as a pesticide:	<input checked="" type="checkbox"/>
This chemical is a biological product:	<input type="checkbox"/>
This chemical is a by-product of a process:	<input type="checkbox"/>
A biological exposure index has been recommended by these agencies:	<input checked="" type="checkbox"/> ACGIH <input checked="" type="checkbox"/> DFG <input checked="" type="checkbox"/> SCOEL

## Workplace exposure standard history

Year	Standard
<a href="#">Click here to enter year</a>	



## References

American Conference of Industrial Hygienists (ACGIH®) (2018) TLVs® and BEIs® with 7<sup>th</sup> 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) (2005) Carbon disulfide – MAK value documentation.

EU Scientific Committee on Occupational Exposure Limits (SCOEL) (2008) Recommendation from the Scientific Committee on Occupational Exposure Limits for carbon disulfide. SCOEL/SUM/82.

Health Council of the Netherlands (HCOTN) (2011). Carbon disulfide. Health-based calculated occupational cancer risk values. The Hague: Health Council of the Netherlands; publication no. 2011/26.

National Industrial Chemicals Notification and Assessment Scheme (NICNAS) (2014) Carbon disulfide: Human health tier II assessment.

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