



CARBON MONOXIDE

CAS number: 630-08-0

Synonyms:

Chemical formula: CO

Structural formula:

Workplace exposure standard (amended)

TWA: 20 ppm (23 mg/m³)

STEL: —

Peak limitation: —

Notations: —

IDLH: 1,200 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 20 ppm (23 mg/m³) is recommended to prevent blood carboxyhaemoglobin (COHb) concentrations in excess of 3.5% in exposed workers which in turn will reduce the risk of adverse effects associated with elevated blood COHb levels. The TWA is expected to provide a margin of safety for individuals particularly susceptible to the adverse effects carbon monoxide (CO) exposure including pregnant women and persons with cardiovascular disease.

Discussion and conclusions

Carbon monoxide is rapidly absorbed from the lungs into the blood binding to haemoglobin to produce a reversible complex known as carboxyhaemoglobin (COHb). COHb reduces the oxygen carrying capacity of blood resulting in lack of oxygen in tissues (hypoxia) resulting in CO-induced toxicity which largely affects the metabolic activity of cells primarily through hypoxic modes of actions. Carbon monoxide will also rapidly diffuse across placental membranes affecting the oxygen supply to foetuses. Pregnant woman and persons with underlying cardiovascular disease are particularly susceptible to the effects related to exposure to CO. COHb is reduced on removal from CO exposure (ACGIH, 2018).

Blood COHb levels in healthy, unexposed subjects are approximately 0.4 to 0.7%. Increase in blood COHb levels result in adverse health effects with levels greater than 4% being associated with adverse health effects in the brain, cardiovascular system and foetuses. Adverse effects on health has been observed in high risk groups at COHb levels greater than 2 to 3%. Blood COHb levels of 5% are expected from an adult, undertaking light work, exposed to 35 ppm for 6 to 8 hours (ACGIH, 2018; DFG, 1992). In rabbits, exposure to 50 ppm for 4 to 24 hours produced COHb levels of 4 to 5% which was reported as the threshold for myocardial damage (SCOEL, 1995).

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.

Insufficient data exists to recommend a skin notation.

DRAFT

APPENDIX

Primary sources with reports

Source	Year set	Standard
SWA	1995	TWA: 30 ppm (34 mg/m³)
CO exerts its main toxic effect via its ability to interfere with oxygen delivery to tissue. Organs most vulnerable to the effects are those with a high metabolic demand for oxygen. Of these the heart, the central nervous system (CNS) and the foetus can be regarded as the critical organs.		
ACGIH	2001	TLV-TWA: 25 ppm (29 mg/m³)
TWA intended to maintain blood COHb levels below 3.5%; to minimise the potential for adverse neurobehavioral changes, to maintain cardiovascular work and exercise capacities, and provide a margin of safety for susceptible individuals.		
Summary of data:		
Human data:		
<ul style="list-style-type: none"> 80–90% of the absorbed CO binds with haemoglobin, resulting in a reduction in the oxygen-carrying capacity of the blood Healthy unexposed persons at rest have blood COHb levels of 0.4–0.7% During pregnancy, increased maternal COHb levels of 0.4–2.6% have been reported COHb for the foetuses of non-smoking mothers reported at 0.7–2.5% Blood COHb levels are dependent on CO exposure concentrations; duration of exposure; size of worker; and activity levels Blood COHb of 5% expected from an adult, undertaking light work, exposed to 35 ppm for 6–8 h Incremental increases in COHb of <5% resulted in a range of cardiovascular effects in normal volunteers Tunnel workers exposed to an average of 70 ppm (2 h alternating over 8 h) had an average of 5% COHb with none above 10% COHb. The average exposure of 35 ppm resulted in no adverse health effects observed COHb levels of 10% associated with symptoms such as headaches while levels of 7% associated with effects on cognitive performance 30 ppm continuous exposure: ≈5% COHb in the pregnant mother; 6% in the foetus. 		
Animal data:		
<ul style="list-style-type: none"> 69% successful pregnancies in rats exposed to 30 ppm dropping to 38% at 90 ppm (no further details). 		
Insufficient data available to recommend Skin, DSEN, or carcinogenicity notations or a TLV–STEL.		



Source	Year set	Standard
DFG	1992	MAK: 30 ppm (33 mg/m³)
<p>MAK recommended to protect both healthy workers and at risk groups (persons with cardiac or vascular disease and pregnant women) from adverse effects associated with elevated blood COHb levels.</p> <p>Summary of additional data:</p> <ul style="list-style-type: none"> • COHb levels at 2.7% can intensify the symptoms of clinically manifest angina • Adverse effects on health in high risk groups at COHb levels >2–3% • Adverse health effects expected at COHb levels >4% in persons without vascular disease • Multiple animal studies demonstrate relationship between exposure, COHb levels and effects • Exposure to 30 ppm for 8-h shift expected to result in no more than 4% COHb level in healthy persons. 		
SCOEL	1995	TWA: 20 ppm (23 mg/m³); STEL: 100 ppm (117 mg/m³)
<p>TWA recommended to prevent blood COHb concentrations >4% which will prevent changes in CNS activity and susceptibility to heart disease.</p> <p>Summary of additional data:</p> <ul style="list-style-type: none"> • The critical effects of CO are on the brain, cardiovascular system and foetus • 50 ppm (59 mg/m³) (rabbits, 4–24 h) threshold for myocardial damage which produces 4–5% COHb saturation levels • Rats exposed to 50 ppm (59 mg/m³) (1–2 mo) showed effects on brain. 		
OARS/AIHA	NA	NA
No report		
HCOTN	NA	NA
No report		

Secondary source reports relied upon

Source	Year	Additional information
NICNAS	✓ 2014	<ul style="list-style-type: none"> • CO at levels producing blood COHb levels between 2.4%-5.9% exacerbates underlying cardiovascular conditions, including increased myocardial ischaemia and cardiac arrhythmias (controlled clinical studies in patients with coronary artery disease; acute-duration exposure) • LOAEC: 200 ppm (229 mg/m³) (rats, 72 wk); showed enlargement of the heart • Multiple studies in animals demonstrate adverse developmental effects of gestational postnatal CO exposure • May damage fertility or the unborn child • No dermal data identified.

Carcinogenicity — non-threshold based genotoxic carcinogens

Is the chemical mutagenic?

No

Notations

Source	Notations
SWA	—
HCIS	—
NICNAS	—
EU Annex	—
ECHA	—
ACGIH	—
DFG	—
SCOEL	—
HCOTN	—
IARC	—
US NIOSH	—

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 LD₅₀ ≤ 1000 mg/kg:

Dermal repeat-dose NOAEL ≤ 200 mg/kg:

Dermal LD₅₀/Inhalation LD₅₀ < 10:

In vivo dermal absorption rate > 10%:

Estimated dermal exposure at WES > 10%:

a skin notation is not warranted

IDLH

Is there a suitable IDLH value available?

Yes



Additional information

Molecular weight:	28.01
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:	<input type="checkbox"/>
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 checked="" type="checkbox"/> ACGIH <input checked="" 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) (1992) Carbon Monoxide – MAK value documentation.

EU Scientific Committee on Occupational Exposure Limits (SCOEL) (1995) Recommendation from the Scientific Committee on Occupational Exposure Limits for carbon monoxide. SCOEL/SUM/57.

National Industrial Chemicals Notification and Assessment Scheme (NICNAS) (2014) Carbon monoxide: Human health tier II assessment – IMAP report.

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