

CARBON BLACK

CAS number: 1333-86-4

Synonyms: CB, acetylene black, channel black, furnace black, lampblack, thermal black

Chemical formula: C

Structural formula:

Workplace exposure standard (retained)

TWA: 3 mg/m³ (as inhalable particulate matter)

STEL: —

Peak limitation: —

Notations: —

IDLH: —

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 3 mg/m³ (as inhalable particulate matter) is recommended to protect for particle-induced bronchitis in exposed workers.

Carbon black is considered an animal carcinogen with unknown relevance to humans (ACGIH, 2018; DFG, 1999). Positive genotoxicity is reported in animals with the generated reactive oxygen species (ROS) or polycyclic aromatic hydrocarbons considered possible drivers for this end point (NICNAS 2015). It is noted that toxic effects vary greatly with particle size and surface area (ACGIH, 2018; DFG, 1999; IARC 2010; NICNAS 2015; OECD, 2006). Therefore, an assessment of additional data sources, particularly for carcinogenicity, is recommended during subsequent reviews.

Discussion and conclusions

Carbon black is used primarily as a source of elemental carbon in chemical manufacture of rubber and pigments.

There is limited evidence to suggest the material possesses intrinsic chemical toxicity. The toxicity is reported to stem from lung clearance inhibition like other nuisance dusts; with higher toxicity due to impeded lung clearance and is associated with decreasing particle size (and increasing surface area).

Reported carcinogenic effects in rats with high lung burdens are caused by inflammatory responses that induce oxidative stress. There is no evidence of carcinogenicity in humans from epidemiological studies. However, reversible radiographic changes in the lungs at low exposures and respiratory dysfunction at higher exposures are reported (ACGIH, 2018). In an extensive US case study, non-smokers had slight increase in the incidence of self-reported bronchitis at the lowest average exposure of 3.44 mg/m³ (ACGIH, 2018).

The current TWA of 3 mg/m³ (as inhalable particulate matter) is recommended to be retained based on weight of evidence and is considered to protect for the onset of pulmonary effects.

Recommendation for notations

Not classified as a carcinogen according to the Globally Harmonized System of Classification and Labelling of Chemicals (GHS). A review of the GHS classification is recommended for the carcinogenicity notation due to inconsistency of available source data.

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

A skin notation is not recommended as there is no indication of systemic effects resulting from skin absorption.

DRAFT

APPENDIX

Primary sources with reports

Source	Year set	Standard
SWA	1995	TWA: 3 mg/m³
ACGIH	2011	TLV-TWA: 3 mg/m³ (inhalable particulate matter)
<p>TLV-TWA to protect for bronchitis caused by exposure to the inhalable particulate fraction. Critical effects are respiratory symptoms, decreased lung function and reversible radiographic changes to the lungs.</p> <p>Carcinogenicity is observed in rats but case studies unable to confirm this effect in humans; classified as an A3 carcinogen.</p> <p>TLV-TWA is based on weight of evidence supported by a case study of exposed workers that reported higher incidence of bronchitis in non-smokers at the lowest level of 3.44 mg/m³ over 40 yr (137.92 mg × yr/m³).</p> <p>Insufficient information to recommend a STEL or notations for skin absorption or sensitisation.</p> <p>Summary of data:</p> <p>Median aerodynamic diameters of particles in the carbon black handling areas range from 10–400 nm. Dust measurements in such areas may be confounded by emissions from hydrocarbon-fuelled engines.</p> <p>Human data:</p> <ul style="list-style-type: none"> • High exposure (unspecified) can lead to emphysema, pneumoconiosis and fibrosis • Chest X-ray of exposed workers (n=94) reported: <ul style="list-style-type: none"> ○ reversible formation of lung opacities at 1.5 mg/m³ ○ after 6 yr, final study phase with 38 of the original subjects showed reversion to normal conditions with decrease in average exposure levels to 0.6 mg/m³ ○ exposure level at which these changes occur is unknown • Multiple epidemiological studies unable to associate excess risk of lung cancer exposure based on SMRs: <ul style="list-style-type: none"> ○ generally, there is cumulative exposure resulting in respiratory dysfunction that is also indicative of excess morbidity • One epidemiological study of 22 plants (n=1755) with mean exposure period 14.1 yr, reported: <ul style="list-style-type: none"> ○ 40 yr exposure at 3 mg/m³ inhalable dust is expected to have 80 mL FEV₁ excess of the normally expected age-related lung capacity loss ○ more recently acquired data in the same study suggests no adverse effects to lung function at 3.82 mg/m³ over 40 yr ○ non-smokers had a slightly higher incidence (from 5% to 9%) of self-reported bronchitis at exposure equivalent to 3.44 mg/m³ over 40 yr • Case study reporting increased risk of bladder cancer in dock workers is considered questionable due to implausible assumption of systemic uptake <i>via</i> inhalational or oral routes. <p>Animal studies:</p> <ul style="list-style-type: none"> • 3 studies reported oxidative DNA damage at macrophage overload concentrations (unspecified concentration; rats, 3 mo) 		

Source	Year set	Standard
		<ul style="list-style-type: none"> ○ particle surface area more decisive than particle mass ○ relevance of studies to human exposures considered questionable • NOAEL of 1 mg/m³ respirable high surface area carbon black (HSCb) for lung inflammation in repeat inhalation study (rats, mice, hamsters, 13 wk): <ul style="list-style-type: none"> ○ rats most sensitive due to less efficient clearance mechanism • Dose-related accumulation and lung neoplasms observed in chronically exposed rats at 2.5 or 6.5 mg/m³ (23 mo) <ul style="list-style-type: none"> ○ no significant increase in neoplasms in mice under similar conditions at 11.6 mg/m³ (18 h/d, 5 d/wk, 13.5 mo) ○ relevance of rat inhalation model questioned regarding human cancer risk assessment • Mutagenic mechanism of action <i>in vitro</i> potentially elicited <i>via</i> particle-induced inflammatory response and associated ROS.
DFG	1999	MAK: not established
<p>Summary of additional data:</p> <p>Derivation of MAK not possible with currently available occupational, medicinal, and toxicological data; evidence for lung carcinogenicity from epidemiological studies. Inconclusive carcinogenic effects observed in rats are of unknown relevance to humans.</p> <p>Human data:</p> <ul style="list-style-type: none"> • Available epidemiological studies frequently lack exposure concentrations or report findings for mixed exposures, which complicates assessment of the carcinogenic activity: <ul style="list-style-type: none"> ○ significant lung function impairment in workers with mixed exposures (n=125) versus controls (n=145); average exposures of 105 was 9.7–31 mg/m³ over 1–11 yr (unspecified dust fraction) ○ progressive lung function impairment in 35 exposed workers at 8.5 mg/m³ inhalable fraction and 7.9 mg/m³ respirable fraction over average exposure of 12.9 yr ○ no relationship between lung function impairment exposure in 913 workers exposed at 0–2 mg/m³; follow-up study after 3 yr with 697 workers showed statistically non-significant decrease in lung function. <p>Animal data:</p> <ul style="list-style-type: none"> • In general, high lung load (≈3–7.8 mg), from 3.5–52.8 mg/m³ exposures, results in delayed alveolar lung clearance, inflammation, and fibrosis in rats, mice, and hamsters • Higher specific surface areas (up to 227 m²/g) may be associated with severity of effects • Localised tumours from subcutaneous injection of carbon black extracts or carbon black impregnated with 300 ppm benzo(a)pyrene reported in mice • Suspended particulate matter is generally non-mutagenic <i>in vitro</i>, but was shown to be weakly clastogenic in Syrian hamster epithelial lung cells • Inconclusive results for DNA damage reported for <i>in vivo</i> study, which may be due to presence of desorbed polycyclic aromatic hydrocarbons (PAHs) • Both <i>in vivo</i> and <i>in vitro</i> mutagenicity tests with suspended particulate matter are inconsistent with those of organic extracts, possibly due to the greater solubilisation of adsorbed contaminants. 		
SCOEL	NA	NA
No report.		

Source	Year set	Standard
OARS/AIHA	NA	NA
No report.		
HCOTN	NA	NA
No report.		

Secondary source reports relied upon

Source	Year	Additional information
NICNAS	✓ 2015	<p>Human data:</p> <ul style="list-style-type: none"> Three-phase epidemiological study (n >2000) concluded minimal effects on pulmonary function at 1 mg/m³ over 8 h/d for 40 yr based on cumulative measurements. Modelled data indicate the forced expiratory volumes (FEV1) were reduced relative to exposure level in non-smoking males: (FEV1) of 48, 91 and 169 mL at 1, 2 and 3.5 mg/m³ exposure, respectively. Physical examinations showed exposure-related symptoms such as cough and phlegm, lethargy, chest pains, skin irritation, reduced senses of smell and hearing, and discoloured sputum and stools. Respiratory effects included bronchitis, pneumosclerosis and myocardial dystrophy, especially in workers with 2–4 yr exposure. <p>Animal data:</p> <ul style="list-style-type: none"> LD₅₀ >10,000 mg/kg (rats, oral) LC₅₀ >4.6 mg/m³ (rats, 4h) Acute inhalational toxicity unclear; particle sizes may contribute to potential toxicity due to lung burden from the particles Severe lung burden occurs between 0.5–1 mg in rats Inflammatory effects in rats exposed to 1 mg/m³ ultrafine particles (20 nm), but no effects when exposed to fine particles (200 nm) Negative results for skin sensitisation in guinea pigs Positive genotoxic results possibly due to action of generated reactive oxygen species or adsorbed PAHs.
IARC	✓ 2010	<ul style="list-style-type: none"> Sufficient evidence for carcinogenic activity in animals, but inadequate data for human assessment.
Nordic Council	✓ 2013	<ul style="list-style-type: none"> Carbon black used as comparison in toxicity studies of carbon nanotubes (CNTs) Intratracheal instillation of 0.5 mg of CNTs or carbon black in mice causes necrosis and lung inflammation only in CNT-treated mice.
OECD	✓ 2006	<ul style="list-style-type: none"> Several cases of skin cancer identified in production workers in the US; cohort in the UK showed no excesses of skin cancer Separate study of rubber and tyre manufacturing industry also showed no excess skin cancers in exposed workers.



Source	Year	Additional information
US NIOSH	✓ 1994	<ul style="list-style-type: none"> No evidence that an acute exposure to a high concentration of carbon black would impede escape or cause any irreversible health effects within 30 min. Revised IDLH of 1,750 mg/m³ based 500-fold factor of the NIOSH REL and OSHA PEL of 3.5 mg/m³.

Carcinogenicity — non-threshold based genotoxic carcinogens

Is the chemical mutagenic? No

Is the chemical carcinogenic with a mutagenic mechanism of action? No

The chemical is not a non-threshold based genotoxic carcinogen.

Notations

Source	Notations
SWA	—
HCIS	—
NICNAS	—
EU Annex	NA
ECHA	NA
ACGIH	Carcinogenicity – A3
DFG	Carcinogenicity – 3B
SCOEL	NA
HCOTN	NA
IARC	Carcinogenicity – Group 2B
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: no
Dermal repeat-dose NOAEL ≤ 200 mg/kg:
Dermal LD ₅₀ /Inhalation LD ₅₀ < 10:
<i>In vivo</i> dermal absorption rate > 10%:
Estimated dermal exposure at WES > 10%:
a skin notation is not warranted



IDLH

Is there a suitable IDLH value available? No

Additional information

Molecular weight:	12.01
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 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) (1999) Carbon black in the form of inhalable dust – MAK value documentation.

International Agency for Research on Cancer (IARC) (2010) Carbon Black, Titanium Dioxide, and Talc. IARC Monographs on the evaluation of the carcinogenic risk to humans, volume 93.

National Industrial Chemicals Notification and Assessment Scheme (NICNAS) (2015) Carbon black: Human health tier II assessment – IMAP report.

Nordic Expert Group for Criteria Documentation of Health Risks of Chemicals (2013) 148. Carbon nanotubes. NR 2013; 47(5).

Organisation for Economic Cooperation and Development (OECD) (2006) SIDS initial assessment profile – Carbon black.

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