# diesel engine emissions

| CAS number: | — |
| --- | --- |
| Synonyms: | — |
| Chemical formula: | — |
| Structural formula: | — |

Workplace exposure standard (interim)

| TWA: | — |
| --- | --- |
| STEL: | — |
| Peak limitation: | — |
| Notations: | — |
| IDLH: | — |
| **Sampling and analysis:** | |

## Recommendation and basis for workplace exposure standard

There are insufficient data available to recommend a suitable TWA.

Given the data available from the primary sources, it is recommended that a review of additional sources be conducted as priority at the next scheduled review.

## Discussion and conclusions

Emissions from diesel engines consist of a mixture of hundreds of chemical compounds, which are emitted in the gaseous and the particulate phase. The composition of emissions varies depending on several factors including engine type, fuel type and operating conditions. Diesel engine emissions contain carcinogenic substances such polycyclic aromatic hydrocarbons (PAHs) and benzene (DFG, 2014; HCOTN, 2019; SCOEL, 2016).

Epidemiological studies have shown an increased relative lung cancer risk for occupations with exposure to diesel engine emissions. The evidence strongly suggests that diesel engine emissions, and many of its components, can induce lung cancer in humans through genotoxic mechanisms that include DNA damage (DFG, 2014; HCOTN, 2019; IARC, 2014; SCOEL, 2016). Consequently, diesel engines emissions are characterised as a non-threshold genotoxic carcinogen. At present, no appropriate inhalation unit risk exists with which to derive a suitable TWA to protect for carcinogenic effects.

HCOTN (2019) has derived a risk-based exposure concentration of 0.011 µg/m3 as respirable elemental carbon, for 40 years occupational exposure with lung cancer as the critical effect. This is considered the “target risk level”, a concentration at which an additional four extra lung cancer deaths per 100,000 are likely to occur due to 40 years of occupational exposure

New engine technologies will replace existing engines, impacting on the exhaust components and possibly the toxicological relevance. Given that diesel engine emission composition can vary depending on several factors and there are limited data regarding diesel engine emissions from modern engines (post 2007) in the primary sources, a priority review of additional data sources is recommended at the next scheduled review.

## Recommendation for notations

Not classified as a carcinogen according to the Globally Harmonized System of Classification and Labelling of Chemicals (GHS). Evidence suggests that diesel engine emissions are carcinogenic, and a review of the literature is recommended to establish a classification.

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

There are insufficient data to recommend a skin notation.

# Appendix

### Primary sources with reports

| Source Year set Standard |
| --- |
| SWA NA NA | |
| No report. |
| ACGIH NA NA |
| No report. |
| DFG 2014 Not assigned |
| No MAK assigned due to evidence of carcinogenicity in humans and animals. Carcinogenic effects of diesel engine exhaust emissions (DEEE) attributable to soot particles and their PAH content.  Diesel engine technology has changed significantly; but available epidemiology data, which were evaluated in 2007, are based on exposures to emissions from older diesel engines.  Increased lung cancer risk reported in epidemiology studies but poor dose-response relationship (i.e., no data on level of exposure) and DEEE composition unknown and dependent on diesel engine technology.  Carcinogenicity category 2 based on insufficient data for category 1 assignment.  Summary of data:  Human data:   * Non-neoplastic disorder: * no changes in lung function during 4 mo exposure of highway toll collectors (exposure level unknown) * statistically significant increased relative risk of death with increasing period of exposures due to obstructive pulmonary disease in railroad workers (adjusted for age, race, healthy worker effect and smoking habits) * Lung cancer: * several cohort (1981–2004) and case-control (1976–2006) studies available to assess lung cancer incidence and DEEE exposure response relationship (that is, relative risk based on period of exposure and not exposure level) * exposure duration of ≥20 yr had the highest relative risk of lung cancer * occupational-based cohort studies reported RR of lung cancer from appropriate studies ranging from 1–2.7 * in railroad workers, when smoking was considered, relative lung cancer risk for those with the longest exposure duration decreased by 4–12% ; further analysis identified no evidence of an exposure-response relationship * case-control studies reported RR of lung cancer ranging from <1–2.5 * inhalation study in non-smoking volunteers concluded early stage of inflammatory response following exposure to 0.2 mg/m3 (<10 µm) for 2 h * inhalation study in non-smokers (with mild asthma and normal lung function) reported initial airway inflammation response to 0.1 mg/m3 (<10 µm) for 2-h exposure; with a significant increase in pro-inflammatory cytokines * a meta-analysis of 29 studies reported a significantly increased relative lung cancer risk of 1.33 (1.27–1.40, 95% CL) for highest exposure group * Bladder cancer: * cohort and case-control studies assessed for correlation between bladder cancer incidence and DEEE exposure; no association reported in cohort studies but case-control studies reported increased bladder cancer incidences in truck driver occupational group * meta-analysis of 35 studies (with ≥5 yr exposure to DEEE) reported increased bladder cancer risk of 1.17 (1.06–1.45) for bus drivers and 1.44 (1.18­–1.76) for the longest exposed groups * Asthma: relationship with exposure and asthma identified in 3 case reports where increased bronchial hyper-activity noted; no further information * Biomonitoring studies identified increases in exposure to genotoxic compounds in workers (origin is unclear, considered PAH from DEEE and lubricating oils responsible); increase in DNA adducts only at higher concentrations * Genotoxicity: no significant increase in urinary mutagenicity or urinary levels of thioether concentrations were reported in exposed individuals versus non-exposed.   Animal data:   * NOAEL: 0.35 mg/m3 (rats; 2 yr, inhalation); biochemical or cytoplasmic changes * Main effects found in rats were hyper- and metaplastic changes and fibroses; BW loss at high concentrations and increases in lung weights * Carcinogenic potential demonstrated in rats only (not in other test species) * significant lung tumour incidence at 2 mg/m3 (particles) for 24 mo * Significant increase in lung tumour incidence in mice at soot concentration of 4 mg/m3 * No increase in tumour incidences in newborn mice exposed to 2–4 mg/m3 * Various DNA adducts detected in human lymphocytes incubated in diesel particle extraction * An adjuvant effect has been demonstrated in sensitisation studies in various animal models and humans. |
| SCOEL 2016 Not assigned |
| Summary of additional data:   * Refers to ‘traditional DEEE’ (Euro 2 emission standards) vs ‘new technology DEEE’ (Euro 3 and Euro III) engines * Mixture of hundreds of chemical compounds, emitted partly in the gaseous phase, partly in the particulate phase * composition varies depending on engine, fuel formulations, lubricating oil, additives and other factors (mobile vs non-mobile sources) * Traditional DEEE contains products of incomplete combustion of toxicological relevance including formaldehyde, acetaldehyde, acrolein, benzene, 1,3-butadiene, toluene, PAH and nitro-PAH and particles of different sizes * Mode of action: i) direct genotoxicity and/or ii) inflammatory response causing oxidative stress and reactive oxygen species resulting in tumours * classified as Carcinogen Category B or C based on non-linear dose-response and threshold mechanism * New technology DEEE contains 90% lower mass of particulates and adsorbed mutagenic compounds when compared to Euro I and II engines * toxic effects in lung attributable to NO2 exposures and as such, no tumours or genotoxicity *in vivo* in 1 yr mouse and 2 yr rat study * Genotoxicity and Carcinogenicity: while toxicological data support a threshold (≤0.02 mg/m3 corresponding to 0.015 mg/m3 respirable EC), epidemiological data suggest significant cancer risks at and below these exposure levels; therefore, protective occupational exposure limit could not be derived * STEL not applicable due to length of time for lung toxicity to develop * No reproductive/developmental effects * Not consider an allergen; may exacerbate allergenic and asthma-like responses. |
| OARS/AIHA NA NA |
| No report. |
| HCOTN 2019 Not assigned |
| Summary of additional data:   * Occupational exposure associated with respiratory inflammation and adverse cardiovascular effects * Exposure can induce lung cancer; components present in the particulate matter induce lung cancer by a stochastic genotoxic mode of action; other components may induce cancer *via* non-genotoxic mechanisms * 3 epidemiological studies (1998–2013) in trucking and mining industry workers show statistically significant positive associations and trends between cumulative respirable EC and lung cancer mortality * considered uncertainties on actual historical exposure levels, smoking or co-exposure to known carcinogenic substances adequately addressed * Derived risk-based exposure levels with lung cancer as critical effect based on meta-analysis of the 3 epidemiological studies * respirable EC is the exposure marker/parameter as a specific and sensitive indicator of DEEE exposure * carcinogenic components primarily found in the particulate matter * based on predicted exposure-response curve calculated in the meta-analysis * concentrations of respirable EC correspond to RR calculated using a log-linear model equation * TWA of 0.011 µg/m3 corresponds to 4 extra lung cancer death cases per 100,000; 40 yr occupational exposure * TWA of 1.03 µg/m3 corresponds to 4 extra lung cancer death cases per 1,000; 40 yr occupational exposure * Not necessary to derive STEL due to long lung clearance duration * No substantial dermal absorption expected. |

### Secondary source reports relied upon

| Source |  | Year | Additional information |
| --- | --- | --- | --- |
| AIOH |  | 2017 | * Acknowledges increase in lung cancer associated with exposure * Recommends a TWA of 0.05 mg/m3 diesel particulate matter (DPM) as action level that triggers investigation * Recommends ALARP below a TWA ≤0.1 mg/m3 DPM * EC is a suitable surrogate for DPM for monitoring * Critical of the degree of potency assigned to diesel particulates by some authorities * Reported a cumulative exposure of 2.5 mg/m3 respirable EC to be protective of increased lung cancer risk * corresponds to 0.05 mg/m3 respirable EC for 45 yr working life. |
| IARC |  | 2014 | * Causes cancer of lungs in humans (Group 1) * positive link between DEEE exposure and cancer of bladder * inadequate evidence for link between DEEE exposure and pancreatic cancer * Exhaust, exhaust particulates and particulate extracts induced DNA damage (e.g. oxidative lesions and bulky adducts), gene mutations, DNA strand breaks *in vivo* and *in vitro* in a wide range of experimental systems * Strong mechanistic evidence DEEE and many of its components, can induce lung cancer in humans through genotoxic mechanisms that include DNA damage * Inflammatory response possible in carcinogenicity |
| US EPA |  | 2002A | * Association of risk of lung cancer and diesel emission exposure observed in >30 epidemiological studies * Demonstrated mutagenic effects of DE and its organic constituents. |
| US EPA |  | 2002B | * Limit data available for short-term exposures; likely to cause acute irritation, neurophysiological symptoms (e.g., light-headedness, nausea), and respiratory symptoms (cough, phlegm) * No further additional information. |
| US EPA |  | 2002C | * PAH Inhalation Unit Risk Factor 6 x 104 |

### Carcinogenicity — non-threshold based genotoxic carcinogens

| Is the chemical mutagenic? | Yes |
| --- | --- |
| Is the chemical carcinogenic with a mutagenic mechanism of action? | Yes |
| **The chemical is a non-threshold based genotoxic carcinogen.** |  |
| Is a cancer slope factor or inhalation unit risk value available? | No |

## Notations

| Source | Notations |
| --- | --- |
| SWA | NA |
| HCIS | NA |
| NICNAS | NA |
| EU Annex | NA |
| ECHA | NA |
| ACGIH | NA |
| DFG | Carcinogenicity – 2 |
| SCOEL | Cat. B/C |
| HCOTN | — |
| IARC | Group 1 |
| 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 |
| --- |
| Insufficient evidence to assign a skin notation. |

### IDLH

| Is there a suitable IDLH value available? | No, the chemical is a genotoxic carcinogen |
| --- | --- |

## Additional information

| Molecular weight: | — |
| --- | --- |
| Conversion factors at 25°C and 101.3 kPa: | 1 ppm = Number mg/m3; 1 mg/m3 = Number ppm |
| This chemical is used as a pesticide: |  |
| This chemical is a biological product: |  |
| This chemical is a by-product of a process: |  |
| A biological exposure index has been recommended by these agencies: | ACGIH  DFG  SCOEL |

## Workplace exposure standard history

| Year | Standard |
| --- | --- |
| Click here to enter year |  |

## References

Australian Institute of Occupational Hygienists (AIOH) (2017) Position paper (revised) - Diesel particulate matter and occupational health issues

Deutsche Forschungsgemeinschaft (DFG) (2014) Diesel engine emissions – MAK value documentation.

EU Scientific Committee on Occupational Exposure Limits (SCOEL) (2016) Opinion from the Scientific Committee on Occupational Exposure Limits for Diesel Engine Exhaust. SCOEL/OPIN/403.

Health Council of the Netherlands (HCOTN) (2019) Diesel engine exhaust. Health-based recommended occupational exposure limit. The Hague: Health Council of the Netherlands; publication no. 2019/02.

International Agency for Research on Cancer (IARC) Diesel and gasoline engine exhaust and some nitroarenes. IARC Monographs – 105 (2014).

US Environmental Protection Agency (US EPA) (2002 A) Diesel engine exhaust, IRIS Chemical Assessment Summary

US Environmental Protection Agency (US EPA) (2002 B) Health Assessment Document for Diesel engine exhaust

US Environmental Protection Agency (US EPA) (2002 C) Polycyclic Aromatic Hydrocarbons, IRIS Chemical Assessment Summary