# ozone

| CAS number: | 10028-15-6 |
| --- | --- |
| Synonyms: | — |
| Chemical formula: | O3 |

Workplace exposure standard (retained)

| TWA: | **—** |
| --- | --- |
| STEL: | **—** |
| Peak limitation: | **0.1 ppm (0.2 mg/m3)** |
| Notations: | **—** |
| IDLH: | **5 ppm** |
| **Sampling and analysis:** The recommended value is quantifiable through available sampling and analysis techniques. | |

## Recommendation and basis for workplace exposure standard

A peak limitation of 0.1 ppm (0.2 mg/m3) is recommended to protect for adverse pulmonary effects in exposed workers.

## Discussion and conclusions

Ozone is used as a disinfectant for air and water, in industrial waste treatment and for bleaching textiles. It is generated in welding arcs and electrical discharges and by ultraviolet radiation.

Critical effects of exposure are adverse effects on lung function. Studies also suggest potential impacts on mucosa and headaches following exposure.

A significant decrease in lung function parameters was reported in two studies. In these studies, volunteers were exposed for two hours at concentrations of 0.12 ppm with intermittent exercise as well as heavy exercise and at 0.2 ppm with high, moderate and light exercise loads. An increase in coughing was also reported in volunteers exposed to 0.12 ppm and symptoms increased with increases in exposure concentration in this group. Exposure at 0.08 ppm for 6.6 hours also resulted in decreases in pulmonary function. Although, the relationship between exposure and work load has not been clearly quantified, the evidence suggests the heavier the workload, the greater the severity of effects on the lungs (ACGIH, 2018).

The peak limitation of 0.1 ppm (0.2 mg/m3) adopted by SWA is recommended to be retained. Based on the weight of evidence in the primary sources, it is considered to protect for adverse pulmonary effects in 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.

There are insufficient data to recommend a skin notation.

# Appendix

### Primary sources with reports

| Source Year set Standard |
| --- |
| SWA 1991 Peak limitation: 0.1 ppm (0.2 mg/m3) | |
|  |
| ACGIH 2001 TLV-TWA: 0.05 ppm (0.1 mg/m3), Heavy work, TLV-TWA: 0.08 ppm (0.16 mg/m3), Moderate work TLV-TWA: 0.10 ppm (0.2 mg/m3), Light work TLV-TWA (≤ 2 hours): 0.2 ppm (0.39 mg/m3), Heavy, moderate, or light workloads |
| TLV-TWAs recommended to minimise adverse pulmonary response in workers. TWAs incorporate workload and exertion rates.  Summary of data:  Human data:   * Evidence suggests heavier workload, the greater potentiation of effects on the lungs; relationship has not been clearly quantified * No evidence people with asthma or COPD are more susceptible to ozone effects than healthy adults * 2 h at 1.5 ppm (average) resulted in a 20% reduction in timed vital capacity of the lung and other effects; no further details * Pulmonary congestion in welders at <2 ppm; effects ‘*disappeared’* at 0.2 ppm; no further details * Discomforting headache and dryness of the throat and mucous membranes of nose and eyes following short duration exposures less than ‘*a few tenths ppm’*; no further details * 6 young male volunteers exposed for 2.5 h at 0, 0.12, 0.18, 0.24, 0.30, or 0.40 ppm while undergoing intermittent exercise: * 0.12 ppm resulted in significant decreases in FVC and FEV1; increased successively with great concentrations * coughing increased at ≥0.12 ppm * Reported threshold between 0.1–0.15 ppm as measured by spirometry for 2 h exposure with intermittent exercise. No further details * Reported a summary of a collection of human exposure-chamber studies; predominantly exposure times of either 2 or 6.6 h; concentrations ranging from 0.08–1.5 ppm; included exercise described as ranging from resting to very heavy exercise: * >0.2 ppm 2 h exposures showed significant decreases in pulmonary function measures with high, moderate and light exercise loads * ≥0.12 ppm 2 h, very heavy exercise resulted in statistically significant decreases in pulmonary function * 0.08 ppm for 6.6 h exposure resulted in statistically significant decreases in mean pulmonary function of up to 8.4% and 7.0% * No evidence indicating chronic damage to the lung at exposures below those which produce acute effects * Reports US EPA ambient air recommends 0.09 ppm for 8 h; includes sensitive individuals.   Animal data:   * LC50: 4.8 ppm (rat, 4h); pulmonary congestion, oedema and haemorrhage * In nasal mucosa study, Bonnet monkeys exposed at 0.15 and 0.30 ppm 8 h/d for 6 or 90 d resulted in ciliated cell necrosis, shortened cilia and secretory cell hyperplasia * Primate studies up to 18 mo reported bronchiolitis, cell hypertrophy and interstitial and luminal inflammatory cells, together with changes in collagen crosslinking at 0.25 ppm. No further details * Rats and mice exposed at 0.12, 0.5, or 1.0 ppm for 2 yr or at 0.5 or 1.0 ppm for 30 mo; reorganisation of the centriacinar zones with bronchiolar epithelium extending down into the alveolar ducts, evidence for mild progressive fibrosis; not carcinogenic to rats; in mice, there was a marginal increase in alveolar/bronchiolar adenoma and carcinoma (combined).   Insufficient data to recommend a sensitiser or TLV-STEL notation. |
| DFG 1998 Not assigned |
| No MAK due to potential carcinogenicity based on evidence in female mice (cited by ACGIH, 2018).  Summary of additional data:   * Based on evidence of reversible changes in lung function parameters in healthy volunteers exposed at 0.08 ppm (0.16 mg/m3) for 6.6 h (cited by ACGIH, 2018) concluded effects could be avoided if cumulative dose remained <0.5 ppm/h: * derivation: 0.08 ppm x 6.6= ~0.5 ppm * could be met by observance of a concentration of 0.1 ppm for a period of 4 h or 0.05 ppm for 8 h * No further information. |
| SCOEL NA NA |
| No report. |
| OARS/AIHA NA NA |
| No report. |
| HCOTN NA NA |
| No report. |

### Secondary source reports relied upon

NIL.

### Carcinogenicity — non-threshold based genotoxic carcinogens

| Is the chemical mutagenic? | Insufficient data |
| --- | --- |
| Is the chemical carcinogenic with a mutagenic mechanism of action? | Insufficient data |
| **Insufficient data are available to determine if the chemical is a non-threshold based genotoxic carcinogen.** | |

## Notations

| Source | Notations |
| --- | --- |
| SWA | NA |
| HCIS | NA |
| NICNAS | NA |
| EU Annex | NA |
| ECHA | NA |
| ACGIH | Carcinogenicity – A4 |
| DFG | NA |
| SCOEL | NA |
| HCOTN | NA |
| 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 |
| --- |
| Insufficient data to assign a skin notation. |

### IDLH

| Is there a suitable IDLH value available? | Yes |
| --- | --- |

## Additional information

| Molecular weight: | 47.99 |
| --- | --- |
| 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

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*](http://www.acgih.org/tlv-bei-guidelines/policies-procedures-presentations) on the ACGIH website.

Deutsche Forschungsgemeinschaft (DFG) (1998) Ozone – MAK value documentation.

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