# Phosphorus (yellow)

| CAS number: | 7723-14-0 |
| --- | --- |
| Synonyms: | White phosphorus, yellow phosphorus, phosphorus |
| Chemical formula: | P4 |
| Structural formula: | — |

Workplace exposure standard (amended)

| TWA: | **0.01 mg/m3** |
| --- | --- |
| STEL: | **—** |
| Peak limitation: | **—** |
| Notations: | **—** |
| IDLH: | **5 mg/m3** |
| **Sampling and analysis:** The recommended value is quantifiable through available sampling and analysis techniques. | |

## Recommendation and basis for workplace exposure standard

A TWA of 0.01 mg/m3 is recommended to protect for adverse effects on the liver, kidney and bones and respiratory tract irritation in exposed workers.

## Discussion and conclusions

Yellow phosphorus is used in the chemical manufacture of pyrotechnics, explosives and fertilisers.

Critical effects of exposure are liver, kidney and bone damage and respiratory tract irritation. It is corrosive upon dermal contact.

Inhalational exposure data in humans and animals are limited. Oxidation products, such as phosphorus pentoxide and phosphoric acid, cause respiratory tract irritation in humans and animals (DFG, 2007). Accidental ingestion of 1 mg/kg was fatal in humans (ACGIH, 2018). A NOAEL of 0.015 mg/kg/day for histopathological changes to liver, kidneys and bones with a corresponding LOAEL of 0.075 mg/kg/day are reported in two repeat gavage studies in rats (DFG, 2007). The systemic NOAEL of 0.015 mg/kg/day noted above is approximately equivalent to an air concentration of 0.105 mg/m3 (DFG, 2007).

Based on the NOAEL of 0.015 mg/kg/day in rats, a TWA 0.01 mg/m3 by DFG (2012) is recommended, which accounts for conversion from rats to humans and translation from experimental to workplace conditions. The recommended TWA is expected to protect for the adverse effects on the liver, kidney and bone and irritation 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 TWA: 0.1 mg/m3 | |
|  |
| ACGIH 2001 TLV-TWA: 0.1 mg/m3 |
| TLV-TWA intended to protect respiratory irritation and acute intoxication including liver cirrhosis, electrolyte imbalance, myocardial disruption and kidney necrosis; margin of safety to protect for potential chronic effects such as jawbone necrosis is unknown due to limited dataset. Derivation of the TLV-TWA is not reported.  Summary of information:  TLV-TWA of 0.02 ppm (0.1 mg/m3) intended to be measured as particulates and vapour due to the relatively high vapour pressure under standard conditions.  Human data:   * Fatal accidental ingestions (as low as 1 mg/kg) caused gastroenteritis, hepatic insufficiency and cardiovascular collapse (no further details provided) * Dermal contact causes 2nd and 3rd degree burns; sudden death after dermal contact reported (no further details provided) * Symptoms of acute poisoning include itching skin, nausea, vomiting, bloody diarrhoea, jaundice, acute hepatic necrosis/cirrhosis and cardiovascular collapse * Necrosis of the jawbone reported in exposed fireworks production workers * No evidence for jawbone necrosis from dental record survey (40 yr) of fertiliser production workers (no further study details provided) * Changes in blood Ca2+ levels and hypophosphataemia associated with chronic exposures (no further details provided).   Animal data:   * Severe respiratory irritation and high mortality due to pulmonary oedema at 20 ppm ≡101 mg/m3 (rats, 7 h/d, 5 d/wk) * No change in growth at 13–16 ppm ≡66–81 mg/m3 compared to controls (species not specified, 7 h/d, 5 d/wk, 4 mo): * prolonged exposure caused bone changes and severe liver and kidney damage (no further details provided) * Bone changes (not specified) at 0.05 mg/kg/d (total 50 mg over study period) in repeat subcutaneous injection study (rats) * Liver damage at 0.1 mg/kg/d in repeat subcutaneous injection study (dogs, duration not specified) * Burned and damaged skin when applied dermally (rabbits); absorption through damaged skin caused phosphorus/calcium electrolyte imbalance * Inhalational dose distributed in descending order of concentration to lungs, bone, liver and kidney: * soft tissue concentration dropped rapidly on exposure cessation * clearance from bones was slow (no further details provided) * Oral dose retained primarily in liver, skeletal muscle, GIT, blood and kidneys after 5 d (rats):   + converted to phosphates and excreted through urine, peak concentrations of phosphate in liver reached 2–3 h following oral dose   Insufficient data available to recommend notations for carcinogenicity, skin absorption or sensitisation. |
| DFG 2011 MAK: 0.01 mg/m3 |
| Summary of additional information:  Respiratory irritant, presumably due to the formation of phosphoric acid and phosphorus pentoxide on contact with air and moisture; systemic endpoints are liver, kidney, and bone damage.  A NOAEL of 0.015 mg/kg/d for histopathological changes to liver, kidneys, and bones in sub‑chronically exposed rats is equivalent to an air concentration of 0.105 mg/m3 in humans, assuming a respiratory volume of 10 m3 for a 70 kg worker during an 8-h shift. Factors of 7/5 and 4 are applied to account for translation from continuous exposure to workplace conditions and allometric scaling from rats to humans, respectively, to arrive at the MAK of 0.01 mg/m3.  Co-exposure to phosphorus oxidation products, e.g. phosphorus pentoxide and phosphoric acid, is considered in combination with the MAK for phosphorus. The MAKs for phosphoric acid and phosphorus pentoxide are 2 mg/m3; the MAK for phosphorus is therefore considered protective of potential irritation effects caused by these oxidation products.  A skin notation is not warranted due to the high reactivity of elemental phosphorus.  Human data:   * Ingestion causes fatty liver degeneration due to impairment of transport lipoprotein synthesis and inhibits bone growth by inhibiting calcified cartilage absorption * Jawbone necrosis in 2/44 workers exposed for 1 yr, and 13/27 exposed for >1 yr reported in workplace study of 3 fireworks factories (n=71); some workers also had irritant cough (no further details) * Mechanism for jawbone necrosis unclear, but possibly due to direct reaction of substance with mouth tissue; unclear if systemic availability contributes to this effect:   + chronically exposed workers likely co-exposed to phosphoric acid due to oxidation of particulate phosphorus * Modelled dermal flux of saturated aqueous solution: 0.0002 mg/cm2/h * No quantitative inhalational exposure studies available.   Animal data:   * Oral LD50: 3.03–3.76 mg/kg (rats), 4.82–4.85 mg/kg (mice) * NOAEL: 0.015 mg/kg/d for histopathological changes to liver, kidney and bones and decreased fertility reported in 2 repeat gavage reproductive studies (rats, 145 or 204 d); LOAEL: 0.075 mg/kg, mortality also observed in this dose group * No carcinogenic activity relative to controls reported in 2 chronic feeding and subcutaneous injection studies with respective exposure ranges 0.2–1.6 mg/kg/d (rats, lifetime ≈420 d) and 0.5–3.2 mg/kg (rats, 2 d/wk, lifetime); agency considers design of both studies inadequate to draw conclusions on carcinogenicity * Non-mutagenic *in vitro* in bacteria as aqueous solution with or without metabolic activation.   Insufficient data to recommend notations for carcinogenicity or sensitisation. |
| SCOEL NA NA |
| No report. |
| OARS/AIHA NA NA |
| No report. |
| HCOTN NA NA |
| No report. |

### Secondary source reports relied upon

| Source |  | Year | Additional information |
| --- | --- | --- | --- |
| US EPA |  | 1990 | * Not considered carcinogenic based on negative results from bacterial mutagenicity and chronic subcutaneous injection studies with rats (also cited in DFG, 2007). |
| US NIOSH |  | 1994 | * IDLH based on acute oral toxicity data in humans and animals. |

### 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 | — |
| HCIS | — |
| NICNAS | NA |
| EU Annex | — |
| ECHA | — |
| ACGIH | — |
| DFG | — |
| 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: | 123.89 |
| --- | --- |
| Conversion factors at 25°C and 101.3 kPa: | 1 ppm = 5.97 mg/m3; 1 mg/m3 = 0.197 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) (2007) Phosphorus, white/yellow – MAK value documentation.

Deutsche Forschungsgemeinschaft (DFG) (2012) Phosphorus, white/yellow – MAK value documentation.

European Chemicals Agency (ECHA) (2020) Phosphorus (yellow) – REACH assessment.

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

US Environmental Protection Authority (US EPA) (1990) Integrated Risk Information System (IRIS) Chemical Assessment Summary – White phosphorus.