



SODIUM AZIDE

CAS number: 26628-22-8

Synonyms: Azide, azium, Nemazyd®, Smite®

Chemical formula: N_3Na

Structural formula: —

Workplace exposure standard (interim)

TWA: 0.04 ppm (0.1 mg/m³)

STEL: —

Peak limitation: 0.11 ppm (0.3 mg/m³)

Notations: —

IDLH: —

Sampling and analysis: The recommended value is quantifiable through available sampling and analysis techniques.

Recommendation and basis for workplace exposure standard

A TWA of 0.04 ppm (0.1 mg/m³) is recommended to protect for headaches in exposed workers.

A peak limitation of 0.11 ppm (0.3 mg/m³) is recommended to protect for acute irritation and adverse cardiovascular and central nervous system (CNS) effects in exposed workers.

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

Discussion and conclusions

Sodium azide forms volatile hydrazoic acid in aqueous solution and is used in chemical synthesis, explosives, rubber, beer production, as a propellant and pesticide. It was formerly used as a vasodilator and disinfectant in medical applications.

Critical effects of exposure are eye and mucous membrane irritation and acute cardiovascular and CNS toxicity.

Headaches and mucous membrane irritation are reported at 0.3 to 0.5 ppm in occupationally exposed workers (ACGIH, 2018; SCOEL, 2009), the incidence of which decreased when exposed to 0.2 ppm. However, a NOAEC could not be determined (SCOEL, 2009). Decreased blood pressure is associated with exposure at 0.11 ppm in acutely exposed production workers (SCOEL, 2009). Hypertensive patients had transient headaches at oral doses up to 3.9 mg/day, whereas doses above 50 mg/day caused tachycardia and collapse (ACGIH, 2018; DFG, 2003).

The available occupational data indicate the onset of adverse cardiovascular effects at 0.11 ppm, which cause headaches at 0.2 ppm; with a steep dose-response relationship observed in orally dosed patients. In view of this information, the peak limitation of 0.11 ppm is retained and considered protective of acute cardiovascular and CNS effects. In the absence of a NOAEC for these effects, the interim TWA of 0.04 ppm by SCOEL (2009) is recommended to be adopted.

Further assessment of additional source material regarding a threshold concentration for headaches is recommended during 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).

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

A skin notation is not recommended based on the available evidence.

DRAFT

APPENDIX

Primary sources with reports

Source	Year set	Standard
SWA	1991	Peak Limitation: 0.11 ppm (0.3 mg/m³)
ACGIH	2001	TLV-Ceiling: 0.29 mg/m³ as N₃Na; 0.11 ppm as hydrazoic acid
<p>TLV-Ceiling intended to protect for acute cardiovascular, CNS and pulmonary toxicity and eye irritation, headache, hypertension and bronchitis. Hydrazoic acid considered in evaluation due to its facile formation in aqueous solution from N₃Na, and common presence when handling and use in its production.</p> <p>Not classifiable as a human carcinogen (A4) based on absence of carcinogenicity in chronically exposed rodents.</p> <p>Summary of information:</p> <p>Toxicity related to inhibition of haem-enzymes. Potent vasodilator rapidly causes dyspnoea, tachycardia and headache. Based on symptoms of laboratory workers exposed at 0.5 ppm, maximal intake of 0.75 mg/d is estimated, which equates to an air concentration of 0.1 mg/m³ (0.04 ppm) assuming a respiratory volume of 10 m³ and 75% retention during an 8 h shift. To provide a margin of safety for the acute effects, a TLV-Ceiling of 0.29 mg/m³ is recommended; derivation of this value is not discussed or presented.</p> <p>Human data:</p> <ul style="list-style-type: none"> Decreased BP and transient throbbing headache in patients treated for hypertension with 0.65–3.9 mg/kg (n=30, up to 2.5 yr): <ul style="list-style-type: none"> 20/30 patients sensitised to these effects and dosage was reduced Transient headache at 5–10 mg: <ul style="list-style-type: none"> hypotension, tachycardia and collapse at 50–60 mg (no further details provided) Headaches and mucosal irritation in laboratory workers chronically exposed at 0.5 ppm hydrazoic acid. <p>Animal data:</p> <ul style="list-style-type: none"> Lethargy and mortality at 40 and 80 mg/kg in repeat oral dose study (rats, 5 d/wk, 2 wk): <ul style="list-style-type: none"> 2/5 females died and increased liver weight in males at 20 mg/kg LD₅₀: 20 mg/kg (rabbits, dermal) Cardiovascular collapse and brain necrosis at 20 mg/kg/d in sub-chronic gavage study with dose groups 0, 1.25, 2.5, 5, 10, and 20 mg/kg/d (rats, 5 d/wk, 13 wk): <ul style="list-style-type: none"> 18/20 rats died at 20 mg/kg/d, survivors had pulmonary congestion, haemorrhage, oedema and brain necrosis clinical signs of intoxication and increased liver weights at all doses (females) and 10 mg/kg (males), but no other symptoms; LOAEL: 1.25 mg/kg/d Intoxication and reduced body weight at 10 mg/kg/d in chronic gavage study by NTP with dose groups 0, 5, and 10 mg/kg/d (rats, 2 yr): <ul style="list-style-type: none"> brain necrosis, convulsions and coma ≥5 mg/kg/d no evidence for carcinogenicity Sterility produced in male mice (no further details) No changes to oestrus cycle at 5 or 10 mg/kg/d (rats, 5 d/wk, 1 yr) Mutagenic <i>in vitro</i> in bacteria, cereal crops, and yeast, weakly mutagenic, but not clastogenic in Chinese hamster ovarian and rat epithelial cells, or human lymphocytes; no <i>in vivo</i> data presented. 		



Source	Year set	Standard
Insufficient data to recommend notations for skin absorption or sensitisation.		
DFG	1981	MAK: 0.2 mg/m³
<p>Summary of additional information:</p> <p>Previous MAK of 0.2 mg/m³ retained based on report of headache and mucosal irritation complaints in laboratory workers chronically exposed at 0.5 ppm (also cited in ACGIH, 2018) and no signs of toxicity in hypertensive patients treated with up to 3.9 mg/d (also cited in ACGIH, 2018); agency notes the latter corresponds to an air concentration of 0.39 mg/m³ assuming a respiratory volume of 10 m³ during an 8-h shift.</p> <p>Human data:</p> <ul style="list-style-type: none"> Complaints of headaches, palpitations, weakness and unsteady gait in factory workers exposed at 0.3–3.9 ppm hydrazoic acid (n=10, 1–16 yr): <ul style="list-style-type: none"> medical examination showed decreased blood pressure and increased heart rate during exposures no effects on ECG, field of vision or liver/kidney function. <p>Animal data:</p> <ul style="list-style-type: none"> Oral LD₅₀: 45 mg/kg (rats); 27–40 mg/kg (mice): <ul style="list-style-type: none"> symptoms included convulsions, respiratory stress and increased heart rate, no MetHb formation observed Non-irritating as semi-occlusive or occlusive patch (rabbits, 1 h): <ul style="list-style-type: none"> caused corrosion and mortality in 3/6 animals after 4 h, unclear if mortality due to substance penetration through damaged skin Positive mutagenicity results <i>in vitro</i> in bacteria not considered relevant to humans due to bacteria-specific metabolism of the substance to genotoxic metabolites: <ul style="list-style-type: none"> weak mutagenicity in yeast was observed at cytotoxic concentrations and therefore also not considered relevant to human genotoxicity; no <i>in vivo</i> studies available. <p>Insufficient data to recommend notations for carcinogenicity, skin absorption or sensitisation.</p>		
SCOEL	2009	8-hour TWA: 0.1 mg/m³
<p>Summary of additional information:</p> <p>OEL based on available occupational exposure data, which suggest a LOAEL near 0.11 ppm (0.3 mg/m³) for blood pressure decrease from chronic exposure, and 0.19 ppm (0.5 mg/m³) for acute headaches in exposed workers; 0.04 ppm (0.1 mg/m³) expected to be protective of these systemic effects and mucous membrane irritation caused by hydrazoic acid.</p> <p>Human data:</p> <ul style="list-style-type: none"> Accidental occupational overexposure during cleaning caused permanent reactive airway dysfunction syndrome in 2 workers (exposure data not presented) Increased incidence of self-reported eye irritation in workers exposed at 0.23 mg/m³, range 0–0.93 mg/m³ (n=41) Headaches (10/11), palpitation (9/11) and low blood pressure (9/11) in production workers at static air concentrations of 0.3 ppm (0.69 mg/m³) N₃Na and 0.07 ppm hydrazoic acid (n=11, 6 mo): <ul style="list-style-type: none"> breathing zone measurements were up to 1.7 mg/m³ and 0.1 ppm, respectively, workplace OEL was 0.11 ppm (0.3 mg/m³) for N₃Na, but was frequently exceeded Headaches reported in 12/65 workers at air concentrations of 0.38–2.82 ppm (1–7.5 mg/m³): 		



Source	Year set	Standard
<ul style="list-style-type: none"> mean BP in workers acutely exposed ≥ 0.11 ppm (0.3 mg/m^3) lower than in workers exposed ≤ 0.11 ppm (0.3 mg/m^3) reported in follow-up study of these 12 workers (15 min) incidence of headache complaints reduced to 1/65 when exposure controlled to 0.19 ppm (0.5 mg/m^3). <p>Animal data:</p> <ul style="list-style-type: none"> Conflicting mutagenicity <i>in vivo</i> in 2 separate sex-linked recessive lethality (SLRL) tests with <i>Drosophila melanogaster</i>, positive mutagenicity in dominant lethal test in <i>Musca domestica</i>: <ul style="list-style-type: none"> no mammalian <i>in vivo</i> studies available. <p>Insufficient data to recommend notations for carcinogenicity, skin absorption or sensitisation.</p>		
OARS/AIHA	NA	NA
No report.		
HCOTN	NA	NA
No report.		

Secondary source reports relied upon

Source	Year	Additional information
US EPA	✓ 1987	<ul style="list-style-type: none"> Sub-chronic gavage study with dose groups 0, 1.25, 2.5, 5, 10, and 20 mg/kg/d (rats, 5 d/wk, 13 wk, also cited in ACGIH, 2018) used principally to derive RfD: <ul style="list-style-type: none"> agency notes statistical significance of increased liver weights in all female rats was not reported 5 mg/kg/d considered NOAEL based on reduced weight gain in males and females Inhalation RfC and carcinogenic potential not assessed.
ECHA	✓ 2020	<ul style="list-style-type: none"> DNEL based on chronic gavage study with dose groups 0, 5, and 10 mg/kg/d (rats, 5 d/wk, 2 yr, also cited in ACGIH, 2018): <ul style="list-style-type: none"> 5 mg/kg/d considered NOAEL due to low incidence of brain lesions (3/60) compared with controls (0/60) overall UF of 37.5 applied to account for dose-response relationship, exposure duration, inter- and intraspecies differences, and exposure route to afford long-term systemic DNEL: 0.164 mg/m^3.

Carcinogenicity — non-threshold based genotoxic carcinogens

Is the chemical mutagenic? Insufficient data

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	NA
EU Annex	—
ECHA	—
ACGIH	Carcinogenicity – A4
DFG	—
SCOEL	—
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

Adverse effects in human case study:

Dermal LD₅₀ ≤ 1000 mg/kg: **yes**

Dermal repeat-dose NOAEL ≤ 200 mg/kg:

Dermal LD₅₀/Inhalation LD₅₀ < 10:

In vivo dermal absorption rate > 10%:

Estimated dermal exposure at WES > 10%:

consider assigning a skin notation

IDLH

Is there a suitable IDLH value available? No

Additional information

Molecular weight:	65.01
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:	✓
This chemical is a biological product:	<input type="checkbox"/>



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
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[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.

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European Chemicals Agency (ECHA) (2020) Sodium fluoroacetate – REACH assessment.

US Environmental Protection Authority (US EPA) (1991) Integrated Risk Information System (IRIS) Chemical Assessment Summary – Sodium fluoroacetate.