TOXICOLOGICAL PROFILE FOR PICRIC ACID

Criteria and Standards Division
Office of Drinking Water
U.S. Environmental Protection Agency
Washington, DC 20460

June, 1989

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FOR

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PICRIC ACID

A. GENERAL

1. CAS Number: 88-89-1

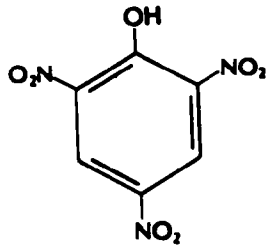
2. RTECS Number: TJ7877000

3. General Name/Synonyms: Ammonium picrate
   Carbazotic acid
   Picronitric acid
   2,4,6-Trinitrophenol

4. Molecular Formula: $C_6H_3N_3O_7$

5. Molecular Weight: 229.11

6. Structure:



B. PHYSICAL AND CHEMICAL PROPERTIES

1. State:	Pale yellow, odorless, intensely bitter crystals	Windholz et al. (1983)
2. Vapor Pressure:	<1 mmHg at 20°C	NIOSH/OSHA (1978)
3. Melting Point:	122-123°C	Windholz et al. (1983)
4. Boiling Point:	Explodes above 300°C	Windholz et al. (1983)
5. Specific Gravity:	1.763	Windholz et al. (1983)

6. Solubility: 1 g dissolves in 78 mL water; 15 mL boiling water, 12 mL alcohol 10 mL benzene; 35 mL chloroform; and 65 mL ether Windholz et al. (1983)

7. Log K_{ow}: No information was found.

8. UV Absorption: No information was found.

C. PHYSICAL/CHEMICAL EQUILIBRIUM FACTORS

1. Bioconcentration Factors (BCF): For the American oysters (Crassostrea virginica), the 42-day BCF was 65.5 for exposure to 0.45 mg/L picric acid and 16.5 for exposure to 0.05 mg/L picric acid (Burton et al., 1983). Cooper et al. (1984) found that the BCF was less than 1 in the epaxial muscle of rainbow trout (Salmo gairdneri) exposed to picric acid for 42-days in a continuous-flow system.

2. K_{wa}: No information was found.

3. K_{oc}: No information was found.

D. ENVIRONMENTAL FATE

1. Photolysis: No information was found.

2. Leaching: No information was found.

3. Route of Water Contamination: No information was found.

4. Hydrolysis: No information was found.

5. Plant Uptake: No information was found.

6. Microbial Degradation: In vitro studies showed that the concentration of picric acid was reduced by 22% (from 240 to 187 μg/mL) during 30 days of incubation with Pseudomonas aeruginosa (Wyman et al., 1979). Approximately 2% was converted to picramic acid; the remaining 20% loss of picric acid was attributed to adsorption onto bacterial surfaces, uptake and accumulation by bacteria, and the formation of unidentified degradation products. In contrast, picric acid (as a 0.1% aqueous solution) was not degraded when incubated for 3 months under aerobic or anaerobic conditions with mixed cultures obtained from soil, compost, activated sewage sludge, and estuarine sediment (Wyman et al., 1979).

7. Persistence in Soil/Water: No information was found.

8. Byproducts: No information was found.

9. Vaporization: No information was found.

E. ACUTE TOXICITY IN MAMMALS

No information was found.

F. SKIN AND EYE IRRITATION AND SENSITIZATION IN MAMMALS

No information was found.

G. SUBCHRONIC TOXICITY IN MAMMALS

No information was found.

H. REPRODUCTION AND TERATOGENICITY IN MAMMALS

No information was found.

I. MUTAGENICITY/GENOTOXICITY

Data are presented in tabular form on page 6.

J. CHRONIC TOXICITY/CARCINOGENICITY STUDIES IN MAMMALS

Data are presented in tabular form on page 6.

K. PHARMACOKINETICS IN MAMMALS

No information was found.

L. HUMAN HEALTH EFFECTS

Gleason et al. (1969) rated picric acid as an extremely toxic compound with a probable oral lethal dose of 5 to 50 mg/kg in humans. Ingestion of 1 to 2 g causes severe poisoning accompanied by headache, progressive stupor, coma, and death. Other effects may include severe gastroenteritis, intravascular hemolysis, hemorrhagic nephritis, and occasionally hepatitis.

Arena and Drew (1986) reported that the local irritant properties of picric acid can cause severe conjunctivitis, palpebral edema, keratitis, and yellow vision. Contact with skin may cause intense pruritic dermatitis with vesicles and weeping lesions. Ingestion can cause acute gastroenteritis with severe abdominal pain, nausea, vomiting, and diarrhea; the vomitus is yellow. The target organ of toxicity is the kidney, and the renal involvement may in

part be due to the hemolytic action of picric acid on red blood corpuscles; acute nephritis is not uncommon when kidneys are affected. In severe acute poisoning, nervous system symptoms are prominent and consist of headache, progressive depression, and finally coma and death.

M. EXISTING STANDARDS/CRITERIA

Type	Standards/Criteria	Proponent	Reference
TLV-TWA[a]	0.1 mg/m^3	ACGIH	ACGIH (1986)
STEL[b]	0.3 mg/m^3 (skin)	ACGIH	ACGIH (1988)
PEL[c]	0.1 mg/m^3	OSHA	CFR (1988)
IDLH[d]	100 mg/m^3	NIOSH/OSHA	NIOSH/OSHA (1978)

[a]Threshold Limit Value-Time Weighted Average.
[b]Short-Term Exposure Limit.
[c]Permissible Exposure Limit.
[d]Immediately Dangerous to Life or Health.

I. MUTAGENICITY/GENOTOXICITY

Test	Strain	Activation	Dose/concentration	Toxic effects	Reference
Ames (reverse mutation)	Salmonella typhimurium TA98, TA100	± S9 (rat liver)	10 µg/plate	Positive only with activation	Wyman et al. (1979)
Ames	Salmonella typhimurium TA 98, TA 100	None	10 µL/plate	Negative	Chiu et al. (1978)
Sex-linked recessive lethal mutations and reciprocal translocation	Drosophila melanogaster	NA	Injected: 400 ppm Feeding: 450 ppm	Positive for sex-linked recessive lethal mutations; negative for reciprocal translocation	Woodruff et al. (1985)

J. CHRONIC/CARCINOGENICITY STUDIES IN MAMMALS

Animal/strain/sex	Route	Dose	Duration	Effects	Reference
Rat/Wistar/M,F	Oral (dietary)	500 ppm	Up to 2.5 years	None	Van Esch et al. (1957, as cited in PHS, 1969)

N. REFERENCES

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