Pentachlorophenol

CAS: 87-86-5
MF: C_6HCl_5O
FW: 266.4
Insoluble in water; well soluble in the organic solvents and oils.

Major uses
Pentachlorophenol (PCP) has a broad application as a very effective biocide, e.g. fungicide, insecticide, herbicide and bactericide. It is the main active ingredient in the wood preservatives, and it is added to products such as stains and paints. PCR is also used in the textile industry. Other applications of PCP include health-care products and disinfectants [1].

Human toxicity
Characteristic features of an acute poisoning are dermatitis, strong irritation in eyes, weakness, seizures, acute lung injury, hepatotoxicity, heart failure, cerebral edema, and rapidly progressing coma in severe cases [1].

Chronic poisoning may produce anorexia, weight loss, weakness, dizziness, headache and anxiety. Prolonged or frequent contact with either solution or dust may cause dermatitis or systemic symptoms including damage to the circulatory system and the heart.

PCP has moderate acute toxicity by ingestion and by skin contact and high toxicity by inhalation. The acute lethal oral dose is approximately 30 mg/kg body weight [1]. The mean lethal dose is 2 g in adults [2].

Signs of systemic poisoning occur at the PCP blood concentrations > 40 mg/l.
In several cases with fatal outcome blood concentrations ranged 46-173 mg/l (reviewed in [3]). The mean lethal blood concentration, based on the data from several handbooks, is 100 mg/l [2].

Carcinogenicity: according to US EPA, PCP belongs to chemical group B2, and it is a probable human carcinogen in humans. Occupational exposure with PCP may occur in the workplaces; there are also some unique suicide cases [1].

Kinetic data
Absorption: PCP is effectively absorbed through the respiratory and gastro-intestinal tract, as well as through the skin. In human volunteers, the observed *half-life for oral absorption* was about 1.3 h [3].

Plasma protein binding of PCP is very high, > 96%, that could explain the long retention time in humans [2].

Elimination half-life in plasma for PCP varies between 30 h [3] and 17 days [4].
Elimination half-life for PCP glucuronide was estimated as 13 h [3].

Metabolism and excretion

PCP is not extensively metabolized, but some metabolism does occur in the liver via conjugation to form glucuronide and via oxidative dechlorination to form tetrachloro-p-hydroquinone [1]. These metabolites are excreted largely in urine in both free (74%) and conjugated (12%) forms over a 7 day period, with an additional 4% found in feces [5].

Toxicological mechanisms

PCP is an inhibitor of oxidative phosphorylation; it binds to mitochondrial protein and inhibits mitochondrial ATPase activity. Thus, both the formation of ATP and the release of energy to the cell from the breakdown of ATP to ADP are prevented. As consequence, basal metabolic rate increases, this leads to increase of body temperature (hyperthermia). Even very low concentration (10^{-6} M) of PCP may inhibit oxidative phosphorylation [6].

Target organs: heart, vascular system (blood vessels/capillaries), CNS, liver and kidney. Pentachlorophenol is accumulated in liver and kidney, where histopathological organ lesions were observed [2].

References

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3. WHO (1987) IPCS – Environmental Health Criteria, No 71. Pentachlorophenol, Geneva.
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5. Baselt, R.C. & Cravey, R.H. (1995) Disposition of Toxic Drugs and Chemicals in Man, 4th edn., pp. 590-593. Foster City, CA, USA: Chemical Toxicology Institute.
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