

DATA EVALUATION RECORD

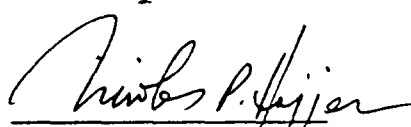
TRICHLORFON

ACUTE TOXICITY

CITATION: Kazakevich RL, Shamray LM. 1977. The state of the nervous system following acute trichlorfon poisoning. Zh. Nevropatol. Psikhiatr. im. S.S. Korsakova 77(2):207-210 [English translation].

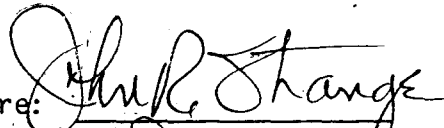
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Date: July 25, 1983

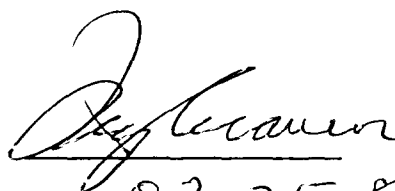
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DATA EVALUATION RECORD

STUDY TYPE: Acute toxicity, case studies in man.

CITATION: Kazakevich RL, Shamray LM. 1977. The state of the nervous system following acute trichlorfon poisoning. Zh. Nevropatol. Psikhiatr. im. S.S. Korsakova 77(2):207-210 [English translation].

ACCESSION NUMBER: Not available.

MRID NUMBER: Not available.

LABORATORY: All-Union Scientific Research Institute of the Hygiene and Toxicology of Pesticides. Polymers, and Plastics, Kiyev, USSR.

TEST MATERIAL: Trichlorfon solution (source, purity, and concentration unspecified).

PROTOCOL:

1. Trichlorfon was the insecticide investigated.
2. The effects on 4 human patients were reported: two 23-year-old women, one 47-year-old man, and one 44-year-old person, sex not specified.
3. In all 4 cases, subjects drank 150-200 ml of a trichlorfon solution (concentration and purity unspecified) by mistake.
4. Upon admission to the treatment facility, patients were examined and the effects were investigated.

RESULTS:

Acute exposure resulted in signs of poisoning and loss of consciousness. Two to three weeks later, pain, paresthesia, and limb weakness developed. Toxic myeloradiculopolyneuritis and encephalopolyneuritis developed.

CONCLUSIONS:

Development of disease occurred 2-3 weeks following acute exposure. Initial effects were pain and paresthesia in the extremities. Delayed effects consisted of polyneuritic surface sensitivity, motor disturbances (legs), pain, toxic hepatitis, and toxic nephritis. The authors reported a direct dependence between the severity of the initial reaction and the severity of the neurological pathology. They therefore postulated a self-allergic mechanism of action persisting as a result of damage to myelinic membranes and peripheral nerve axons. In the opinion of this reviewer, the report provided an adequate description of the symptomatology associated with acute exposure. However, the discussion on mechanism is only of academic interest, because it is neither supported nor refuted by the data.

CORE CLASSIFICATION: Supplementary data.

The study provides a useful description of acute effects of trichlorfon exposure in humans. However, more useful information could be gained if the authors estimated the dose level received by each patient. An estimate of the maximum possible dose received can be made as follows: a volume of 200 ml of water would contain about 24 g of trichlorfon (solubility limits), and if the average weight of the patients was 70 kg, then the maximum possible dose received would be 34.3 mg/kg.