TRIPHENYLTIN ACETATE

International Programme on Chemical Safety
Poisons Information Monograph 589
Chemical

1. NAME

1.1 Substance

Triphenyltin acetate

1.2 Group

Organotin compound

1.3 Synonyms

(acetyloxy)triphenylstannan;
acetatotriphenylstannane,
acetoxytriphenylstannane,
acetyloxytriphenyltin,
fentin acetate,
phentin acetate,
stannane, (acetyloxy) triphenyl,
tin triphenyl acetate,
TPTA,
triphenylaceto stannane,

1.4 Identification numbers

1.4.1 CAS number

900-95-8

1.4.2 Other numbers

Hazchem code 2Z
NFPA code 2-0-0
NIOSH number WH 6650000
UN number 2786

1.5 Main brand names, main trade names

1.6 Main manufacturers, main importers

2. SUMMARY

2.1 Main risks and target organs

Triphenyltin has a low toxicity.
Liver damage (usually fairly slight) is seen in humans, and immunotoxicity, fetotoxicity, reproductive toxicity and respiratory toxicity have been observed in animals. Allergic reactions are common, especially after dermal exposure. Respiratory irritation and irritation of the mucosa have been experienced by workers.

2.2 Summary of clinical effects

Malaise,dizziness, vomiting
Headache
Photophobia
Abdominal pain
Temporary loss of consciousness
Allergic reactions (irritation of skin/mucosa/conjunctivae)
Liver enlargement
Glycosuria

2.3 Diagnosis

The following clinical symptoms are characteristic of TPTA poisoning:-

Malaise, dizziness, vomiting
Headache
Abdominal pain
Allergic reactions (irritation of skin/mucosa/conjunctivae)
Liver enlargement

Laboratory analysis:-

Obtaining urine or blood samples is of prime importance. Urinary tin levels have been found to peak 5 or 6 days after poisoning, and provide a specific diagnosis. A decrease in polymorphonuclear leucocyte activity also indicates TPTA poisoning, (Colosio et al., 1991).

2.4 First-aid measures and management principles

EYE: Remove any contact lenses, and wash the eye with flowing water for 10 minutes.

INHALATION: Remove the victim from the area of exposure. If the victim is conscious make the person lie down quietly and give oxygen if available.

INGESTION: Do not induce vomiting. If the victim is conscious give 500ml of water to drink. With usual precautions and contraindications the use of Ipecac Syrup, activated charcoal and a cathartic are indicated in the event of ingestion.

SKIN: Remove any contaminated clothing immediately. Drench the affected area with running water for at least 10 minutes.

GENERAL: The management of triphenyltin poisoning should be primarily directed towards decontamination and supportive care, as there is no specific antidote.

3. PHYSICO-CHEMICAL PROPERTIES

3.1 Origin of the substance

Organotin compounds have been widely used as pesticides over the last 30 years. Triphenyltin acetate was produced in 1954. It's comparatively low phytotoxicity meant that as the

active principle of Brestanâ it was the first practical organotin pesticide.
Triphenyltin acetate is produced by the following pathway, (Bock, 1981).
$SnCl_4 + 4(C_6H_5)MgCl \rightarrow (C_6H_5)_4Sn + 4MgCl_2$
$(C_6H_5)_4Sn + HCl \rightarrow (C_6H_5)_3SnCl + C_6H_6$
$(C_6H_5)_3SnCl + MOH \rightarrow (C_6H_5)_3SnOH + MCL$
Heat the triphenyltin hydroxide with glacial acetic acid.

3.2 Chemical structure

$(C_6H_5)_3SnOOCCH_3$

3.3 Physical properties

3.3.1 Colour

Normal state at room temperature is white odourless crystals.

3.3.2 State/form

3.3.3 Description

MW = 409.6
Solubility @ 20°C: 2.8 mg/100ml in water, (Hayes and Laws, 1991); 0.2% in ethanol, (Bock, 1981); 3.7% in ether, (Bock, 1981).
Melting point: 122-124°C
Vapour pressure: 60 torr @ 230°C (Bock, 1981); 1.33 × 10-6 torr @ 30°C, (Hayes and Laws, 1991).

3.4 Hazardous characteristics

Stability:

Slow decomposition (loss of phenol groups as benzene) of the aqueous solution at room temperature and neutral pH. t1/2 @ 30 mins, (Bock, 1981). Lasts indefinitely with dry storage and normal temperate. Completely decomposes in sunlight after 8 hours. Half life on sugar beet leaves is about 3 days, (Bock,1981). The half life of TPTA in soil is around 140 days, (Duncan, 1980).

Combustion:

TPTA produces an acrid smoke and fumes of CO, CO_2, tin and tin oxides when heated to decomposition.

Care should be taken to ensure there is no adverse phytotoxicity or animal toxicity, especially in aquatic environments as aquatic organisms are very susceptible (Solomon et al., 1989). In some aquatic environments it seems that TPTA persists for long periods of time, (Duncan, 1980).

TPT has been detected in sediments and bivalves of lake Geneva, although tributyltin and dibutyltin were found at higher levels, (Becker et al., 1992).

Contaminants of technical grade TPTA (containing 0-95% TPTA) are approximately 7% tetraphenyltin, 2% diphenyltin and 1% volatiles (Bock, 1981).

The pesticide Brestan 60, in addition to 60% TPTA contains 15% manganese dithiocarbamate (Maneb), which could confuse clinical findings in cases of TPTA poisoning, (NIOSH, 1976).

4. USES/CIRCUMSTANCES OF POISONING

4.1 Uses

4.1.1 Uses

4.1.2 Description

Fungicide, yeasticide, bacteriacide, molluscicide. Organotin compounds are used as stabilizers in PVC and other plastics. Antifoulant in ship paint. 200-360 g/ha, 3 to 5 applications per crop is common (Bock, 1981).
Kills freshwater snails.
Anti-feeding and repellent effects on insects such as caterpillars.

4.2 High risk circumstance of poisoning

People involved with spraying the pesticide and preparing the aqueous solution are particularly at risk of TPTA poisoning.

4.3 Occupationally exposed populations

Crop-duster pilots
Pesticide sprayers
Factory and agricultural workers

5. ROUTES OF ENTRY

5.1 Oral

TPTA may be ingested with vegetable intake. Risk of harmful effects due to normal dietary intake is very low, since residues seldom rise above 1 ppm in crops tested. Compulsory waiting periods allow degradation to levels below legal limits.

5.2 Inhalation

Poisonings have occurred after respiratory exposure during crop spraying and mixing of the powder into water.

5.3 Dermal

Cases have been reported in which contact with skin has caused poisoning. Workers handling the powder are particularly at risk from this type of exposure, (Colosio et al., 1991).

5.4 Eye

Airborne TPTA dust irritates the mucous membranes of the eye.

5.5 Parenteral

No data available

5.6 Others

No data available

6. KINETICS

6.1 Absorption by route of exposure

Absorption through intact skin is poor, (Stoner, 1966). Intraperitoneal absorption is at least 10-fold more efficient than oral in laboratory animals, (Stoner, 1966). TPTA given orally is absorbed well in rats and guinea pigs, but not so well in sheep, (WHO, 1980).

6.2 Distribution by route of exposure

Triphenyltin is rapidly distributed to all tissues, including the brains of rats.
Oral dosing of sheep produces slight accumulation in liver, kidney, lung, pancreas, gall bladder and brain, (Herok and Götte, 1963).

6.3 Biological half-life by route of exposure

Once absorbed, elimination is slow. One study claims a half-life in the rat brain of 3 days, while another study indicated a considerably longer half-life in guinea pigs (World Health Organization, 1980).

6.4 Metabolism

A large proportion of radioactive Sn is found to be converted to inorganic tin in sheep, (Bock, 1981). Phenyltin and diphenyltin are produced in small amounts. TPTA is resistant to biological oxidation by the microsomal monooxygenase reaction (Bock, 1981).

Subacute doses of TPTA to rabbits does not greatly influence the level of liver cytochrome P450, but by some unknown mechanism there appears to be induction of kidney Cyt P450. This induction (2-fold at75 ppm in the diet over a70 day period) could have an effect on the metabolism of endogenous substrates (Dacasto et al., 1991).

The means of metabolism is unknown. Diphenyltin and monophenyltin levels in the waste products increase while triphenytin levels drop, (Freitag and Bock, 1974).
Degradation probably occurs as follows: (C6H5)3Sn+ -> (C6H5)2Sn2+ -> (C6H5)Sn3+ -> Sn4+

6.5 Elimination by route of exposure

Rats dosed with low levels of TPT in their diet excrete 80-90% unchanged in their faeces within7-10 days, (Bock, 1981; Freitag and Bock, 1974).

Sheep given TPTA orally excreted most of it in the faeces within a few days, (Herok and Götte, 1963).

7. TOXICOLOGY

7.1 Mode of Action

Low concentrations of triphenyltin and other organotins inhibit the H+ translocation of the membrane-bound portion of H+ ATPase, (Papa et al., 1982). Some other ATPases and ion channels such as the Na+-K+ and the Ca2+ translocating ATPases are also affected, (Powers and Beavis, 1991). Triphenyltin has been shown to increase the intracellular Ca2+ concentration of mouse thymocytes, and the cytotoxicity could be caused by the resultant disruption of homeostasis. A likely cause for the increase of intracellular Ca2+ is an inhibition of the sequestering of Ca2+ by Ca2+ translocating ATPases, (Oyama et al., 1992).

Organotins may react with thiol groups in proteins, (van der Bend et al., 1985; Byington et al., 1974).

Anti-inflammatory properties may be caused by the prevention of phosphorylation of lipomodulin and the subsequent release of arachidonic acid, (Arakawa and Wada, 1984). Triphenyltin chloride inhibits histamine release from mast cells, (Nishida et al., 1992).

Triphenyltin chloride inhibits superoxide production by human neutrophils, (Matsui et al., 1983; Miura and Matsui, 1991; Matsui et al., 1983).

The hyperglycaemic action of triphenyltin may be due to the inhibition of insulin release, (Manabe and Wada, 1981).

7.2 Toxicity

7.2.1 Human data

7.2.1.1 Adults

No data available.

7.2.1.2 Children

No data available.

7.2.2 Relevant animal data

The toxicity of organotin compounds increases with the number of alkyl groups attached. Of the trialkyltins, triphenyltin is moderately toxic, but less toxic than trialkyltins with shorter alkyl

chains, such as trimethyltin.

Acute oral toxicities:
LD50 in guinea pigs 10-41 mg/kg, (Bock, 1981).
LD50 in rabbits 30-50 mg/kg, (Bock, 1981).
LD50 in mice ranges from 81-93 mg/kg, (Bock, 1981).
LD50 in rats ranges from 136-491 mg/kg, (Bock, 1981; Attahiru et al., 1991).

Acute dermal toxicity:

Rat dermal LD50 of TPTA in oil is around 450 mg/kg, (Bock, 1981).
Dermal LD50 in mice is 350 mg/kg, (Bock, 1981).
Dermal LD50 in rabbits is approximately 125 mg/kg, (Bock, 1981).

Chronic oral toxicity:

In guinea pigs, the cumulative toxic oral dose appears to be of the same order as the acute toxic dose, (Stoner, 1966). The guinea pig dietary NOEL is no more than 5 ppm, (Verschuuren et al., 1966). Rats fed TPTA at levels of 300 ppm become ill and die, (Stoner, 1966). NOEL for dogs is less than 5 ppm, (Stoner, 1966).

7.2.3 Relevant in vitro data

Haemolysis is caused by TPT chloride at low concentrations in animal blood, but not in human blood, (Byington et al., 1974).

7.2.4 Workplace standards

In 1965 the ACGIH set the TLV TWA for all organotins at 0.1 mg/m3, (measured as tin). The paucity of both human and animal toxicity data at low organotin concentrations has meant that this TLV was derived by analogy with mercury, selenium and thallium data. There are no specific standards for individual organotin compounds, although the toxicity of different organotin compounds varies greatly, (NIOSH, 1976).

7.2.5 Acceptable daily intake (ADI)

The ADI is 0.0005 mg/kg, (FAO/WHO, 1971).

7.3 Carcinogenicity

Carcinogenicity tests show no carcinogenic effects in mice, (Innes et al., 1969). Organotin compounds may actually have antitumour activity, (Saxena and Tandon, 1983).

7.4 Teratogenicity

Triphenyltin has no known teratogenic effects. A study by Noda et al., (1991) suggests that TPTA given to maternal rats in their days7-17 phase does not induce foetal

malformations. Embryotoxic effects were, however, seen at doses above 3.0 mg/kg maternal body weight.

7.5 Mutagenicity

Results of the dominant lethal assay suggest that TPTA is not a mouse mutagen, (Duncan, 1980).

7.6 Interactions

No data available

8. TOXICOLOGICAL ANALYSES AND BIOMEDICAL INVESTIGATIONS

8.1 Material sampling plan

8.1.1 Sampling and specimen collection

Toxicological analyses

8.1.1.2 Biomedical analyses

8.1.1.3 Arterial blood gas analysis

8.1.1.4 Haematological analyses

Other (unspecified) analyses

8.1.2 Storage of laboratory samples and specimens

8.1.2.1 Toxicological analyses

8.1.2.2 Biomedical analyses

8.1.2.3 Arterial blood gas analysis

8.1.2.4 Haematological analyses

Other (unspecified) analyses

8.1.3 Transport of laboratory samples and specimens

8.1.3.1 Toxicological analyses

8.1.3.2 Biomedical analyses

8.1.3.3 Arterial blood gas analysis

8.1.3.4 Haematological analyses

8.1.3.5 Other (unspecified) analyses

8.2 Toxicological Analyses and Their Interpretation

8.2.1 Tests on toxic ingredient(s) of material

8.2.1.1 Simple Qualitative Test(s)

8.2.1.2 Advanced Qualitative Confirmation Test(s)

8.2.1.3 Simple Quantitative Method(s)

8.2.1.4 Advanced Quantitative Method(s)

8.2.2 Tests for biological specimens

8.2.2.1 Simple Qualitative Test(s)

8.2.2.2 Advanced Qualitative Confirmation Test(s)

8.2.2.3 Simple Quantitative Method(s)

8.2.2.4 Advanced Quantitative Method(s)

8.2.2.5 Other Dedicated Method(s)

8.2.3 Interpretation of toxicological analyses

8.3 Biomedical investigations and their interpretation

8.3.1 Biochemical analysis

8.3.1.1 Blood, plasma or serum

8.3.1.2 Urine

8.3.1.3 Other fluids

8.3.2 Arterial blood gas analyses

8.3.3 Haematological analyses

8.3.4 Interpretation of biomedical investigations

8.4 Other biomedical (diagnostic) investigations and their interpretation

8.5 Overall Interpretation of all toxicological analyses and toxicological investigations

Sample collection

No data available

Biomedical analysis

Allergological investigations should be made, in order to try to confirm TPTA as the toxic agent. Liver and brain should be tested for signs of damage (Colosio et al., 1991).

Toxicological analysis

High urinary tin excretion is indicative of recent exposure. The normal value ranges from 10-65 ng Sn/ml urine, (Manzo and Richelmi, 1981).

Tin levels in blood above the average range, (10-20ng/ml) are indicative of tin toxicity, (Manzo and Richelmi, 1981).

Other investigations

No data available

8.6 References

9. CLINICAL EFFECTS

9.1 Acute poisoning

9.1.1 Ingestion

After a lethal dose, death in laboratory animals occurs over several days. Signs and symptoms include weakness, anorexia, watery diarrhoea, staggering, reddish tears, coma and death, (Bock, 1981).

9.1.2 Inhalation

Human cases of TPTA inhalation have resulted in malaise, headache, dizziness, loss of consciousness, epigastric pain, dryness of the mouth, photophobia, and liver damage, (Colosio et al., 1991; Manzo and Richelmi, 1981; World Health Organization, 1980).

Animals exposed to TPTA dust showed signs of irritation of mucous membranes, eyes and respiratory tract. Restlessness, grooming and coughing were also seen, but the animals did not die. Post mortem showed respiratory tract irritation, (Bock, 1981).

9.1.3 Skin exposure

Although it has been claimed that TPTA does not readily penetrate intact skin, (Stoner, 1966)there have been cases of human intoxication. TPTA poisonings in man have resulted in allergic reactions, liver damage, malaise, metabolic and enzymatic changes, (Colosio et al., 1991), headaches, epigastric pain, and weakness, (World Health Organization, 1980).

Severe inflammation is produced in rats given TPTA in oil, (Bock, 1981).

Tests of dermal toxicity of triphenyltin fluoride (TPTF) have been conducted on humans, (Andersen and Petri, 1982). In the aromatic solvent it gave rise to burning sensations, erythema and dermal necrosis after 30 mins. It should however be noted that TPTA seems to be more acutely dermally toxic than TPTF.

9.1.4 Eye contact

Triphenyltin is irritating to the eyes, (Bock, 1981; World Health Organization, 1980).

9.1.5 Parenteral exposure

No data available

9.1.6 Other

No data available

9.2 Chronic poisoning

9.2.1 Ingestion

Reduced immune responses and weight loss (mainly related to reduced food intake) have been observed in rats fed with 15 ppm triphenyltin, (Bock, 1981).

Decreased levels of peripheral lymphocytes have been observed in rabbits, (Dacasto et al., 1991).

9.2.2 Inhalation

No data available

9.2.3 Skin exposure

No data available

9.2.4 Eye contact

No data available

9.2.5 Parenteral exposure

No data available

9.2.6 Other

No data available

9.3 Course, prognosis, cause of death

Irritation of the eye, respiratory and nasal mucosa are experienced before airborne TPTA causes signs of toxicity.

Early symptoms of poisoning are malaise, dizziness, vomiting and headache, and it can take between a few hours to days for them to appear. These symptoms can be found in minor poisoning (dermal or inhalation). Allergic symptoms may persist for over a week even after slight poisonings. Moderately poisoned individuals will often experience abdominal pain, with continued nausea/vomiting for up to several days.

Severe acute TPTA toxicity has not been well documented in the literature.

9.4 Systematic description of clinical effects

9.4.1 Cardiovascular

Heart failure is seen in rats given a lethal

oral dose, (Attahiru et al., 1991).

9.4.2 Respiratory

Upper respiratory tract irritation and shortness of breath have been reported in human inhalation cases, (World Health Organization, 1980).

Rats orally given a high lethal dose of TPTA were found to have severe congestion and haemorrhages of the lungs,(Attahiru et al., 1991).

9.4.3 Neurological

9.4.3.1 CNS

Headache, dizziness, photophobia, and occasionally loss of consciousness and epileptic seizures are seen. Cerebral oedema is seen in rats given lethal oral doses, (Attahiru et al., 1991).

Triphenyltin has an excitotoxic effect on neurons at concentrations ranging from 10-5 to 10-7 M, (Oyama and Akaike, 1991; Oyama, 1992).

Triphenyltin has been shown to increase the severity of maximal electroshock seizures in mice at greater than7.17 mg/kg, (Doctor and Fox, 1982).

9.4.3.2 Peripheral nervous system

Spraying with Brestan 60 has been associated with lower limb parasthesia, (Manzo and Richelmi, 1981).

9.4.3.3 Autonomic nervous system

Dry mouth and unquenchable thirst have been reported with poisonings involving Brestan 60 containing dithiocarbamate, (National Institute for Occupational Health and Safety, 1976).

9.4.3.4 Skeletal and smooth muscle

No data available

9.4.4 Gastrointestinal

Nausea and vomiting occur after poisoning, (Manzo and Richelmi, 1981; Colosio et al., 1991). Heartburn and dryness of the mouth seem to precede the more severe symptoms in the case of Brestan 60 poisoning, (National Institute for Occupational Health and Safety, 1976).

9.4.5 Hepatic

Abdominal pain, liver damage, hepatomegaly, and elevated serum enzyme levels have been reported in poisoning cases. A study on hepatotoxicity in rats showed a decrease in certain hepatic enzyme activities and biliary excretion, (Di Nucci et al., 1986).

High doses given orally to rats cause liver congestion, (Attahiru et al., 1991).

9.4.6 Urinary

9.4.6.1 Renal

Lethal doses of TPTA given orally to rats cause renal congestion, (Attahiru et al., 1991).

9.4.6.2 Others

Mild to severe glycosuria has been found in TPTA poisoning cases, (Colosio et al., 1991).

9.4.7 Endocrine and reproductive systems

In female rats, 80 ppm in food caused a slight decrease in litter numbers. 250 ppm caused a distinct reduction of food intake, growth rate and reproductive capability, (Bock, 1981). Female rats fed 20 mg TPTA per kg body weight per day had small ovaries and decreased fertility, (Newton and Hayes, 1968). Male rats fed at 20 mg TPTA/kg body weight per day presented with degenerative changes of the testes, which were shrunken and infertile. It is unknown whether these are primary or secondary effects ofTPTA, (Pate and Hayes, 1968).

9.4.8 Dermatological

Dermatitis has been seen. Irritation of hands, skin and scrotum were seen in workers spraying 20% TPTA, (WHO,1980).

9.4.9 Eye, ears, nose, throat: local effects

Irritation of mucous membranes of the eye and respiratory tract have been observed in laboratory animals treated with airborne TPTA dust, (Bock, 1981).

Identical symptoms were seen in workers spraying 20% TPTA, (WHO, 1980).

9.4.10 Hematological

Haemolysis is caused by TPT chloride at low concentrations in animal blood, but not in human blood, (Byington et al., 1974).

9.4.11 Immunological

Subacute administration of dietary TPTA at levels as low as 15 ppm have been shown to have an immunosuppressive effect on rabbits, (Dacasto et al., 1991).

Triphenyltin is immunotoxic in rats, but it is thought that the triorganotins with shorter alkyl chain lengths are more immunotoxic, (Snoeij et al., 1985).

9.4.12 Metabolic

9.4.12.1 Acid-base disturbances

No data available

9.4.12.2 Fluid and electrolyte disturbances

No data available

9.4.12.3 Others

Kidney cytochrome P450 levels have become elevated in rabbits treated subchronically with 15 ppm TPTA. Transient hyperglycaemia, glycosuria, and an increase in liver transaminase activity can persist for weeks after poisoning in humans, (Colosio et al., 1991).

9.4.13 Allergic reactions

Allergic reactions in the form of dermatitis and erythematous eruptions have been reported in humans, (Colosio et al., 1991).

9.4.14 Other clinical effects

No data available

9.4.15 Special risks

Radiolabelled tin in the milk of pregnant sheep fed at 3.2 ppm was detected at a level of 2 mg/L during the 25 days of feeding, and gradually subsided when doses were stopped, (Bock, 1981).

9.5 Others

No data available

10. MANAGEMENT

10.1 General principles

Generally poisonings are not life-threatening, but vital signs should be watched in severe cases. Treatment must be symptomatic.

10.2 Life supportive procedures and symptomatic treatment

Symptomatic treatment.

10.3 Decontamination

Remove and discard contaminated clothing.
Remove any contact lenses and irrigate exposed eyes with copious amounts of water (or saline).
Induce vomiting, only if the poison does not contain hydrocarbon solvents.
Perform gastric lavage.
Administer a cathartic.

10.4 Elimination

Not applicable

10.5 Antidote treatment

10.5.1 Adults

No data available

10.5.2 Children

No data available

10.6 Management discussion

No data available

11. ILLUSTRATIVE CASES

11.1 Case reports from literature

Case 1. Adult male, dermal occupational exposure. A case has been reported in which a 36 year old man was exposed to an unknown amount of 18.95% TPTA, (Brestan formulation). While handling the powder without gloves he accidentally spilt some on the exposed cutaneous area of his arms. He washed immediately afterwards, but 12 hours later experienced bilateral plantar pain, and after 24 hours severe genital oedema, followed by an erythematous reaction on his torso. Two days after exposure he was experiencing general malaise, dizziness and abdominal pain. Tests showed an impairment of glucose metabolism, slight glycosuria, liver enlargement, a brief increase in IgE, however patch tests with Brestan did not produce any positive results. Steroid treatment did not prevent urticarial recurrences, but antihistamine was found to be helpful, (Colosio et al., 1991).

Case 2. Adult male, occupational exposure by inhalation. A75 year old farmer inhaled some Brestan 60 powder (60% TPTA) while preparing the aqueous solution. A few days later he suffered from episodes of sudden malaise, dizziness and temporary loss of consciousness, followed by a severe headache, vomiting and photophobia. Once admitted to hospital he received 10 mg metoclopramide IM twice a day and 6.5 mg diethylperazine twice a day as a suppository. Despite the treatment, nausea, vomiting and photophobia persisted for 4

days. He underwent a complete recovery in 10 days. All laboratory tests were within normal limit, (Manzo and Richelmi, 1981).

Case 3. Adult male epileptic, occupational exposure. A 53 year old farmer, suffering from primary epilepsy was admitted to hospital 3 hours after spraying Brestan 60 (60% TPTA). He had experienced general malaise, dizziness, headache, asthenia and dryness of the mouth, but the symptoms had mostly disappeared at the time of hospitalization. His liver was slightly enlarged. Symptoms reappeared the next day, and he developed an epileptic seizure which was treated with intravenous diazepam. The following day headache and dizziness were accompanied by lower limb paresthesia. Nine days after admission the patient developed diffuse erythema on the face, which was treated successfully with 25 mg promethazine orally per day, (Manzo and Richelmi, 1981).

12. ADDITIONAL INFORMATION

12.1 Specific preventive measures

Workers exposed to TPTA either in the aqueous form or the powdered form should wear adequate protective clothing. Respirators should be used if there is any chance of inhalation of the powder or the aerosol. Exposed workers would be advised to regularly have their urinary tin levels checked.

12.2 Other

13. REFERENCES

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