Yangzhou Sunchem Co.,Ltd.
Material Safety Data Sheet
Zinc pyrithione (ZnPT)

Section 1 - CHEMICAL PRODUCT AND COMPANY IDENTIFICATION

PRODUCT NAME

ZINC PYRITHIONE

NFPA

Flammability	1
Toxicity	3
Body Contact	3
Reactivity	1
Chronic	3

SCALE: Min/Nil=0 Low=1 Moderate=2 High=3 Extreme=4

PRODUCT USE

Operators should be trained in procedures for safe use of this material. Active fungicide and bactericide for topical application. Regeant

SYNONYMS

C10-H8-N2-O2-S2-Zn, "zinc, bis(2-pyridylthio)-, N, N-dioxide", "zinc, bis(2-pyridylthio)-, N, N-dioxide", bis(1-hydroxy-2(1H)-pyridinethionato)zinc, "omadine zinc", "2-pyridinethiol-1-oxide, zinc salt", "2-pyridinethiol-1-oxide, zinc salt", "pyrithione zinc", "zinc, bis(1-hydroxy-2(1H)-pyridinethionato)-", "zinc, bis(1-hydroxy-2(1H)-pyridinethionato-O, S)-, (T-4)-", "zinc, bis(2-pyridylthio)-, 1, 1'-dioxide", "zinc, bis(2-pyridylthio)-, 1, 1'-dioxide", "zinc omadine", zincpolyanemaine, "zinc PT", "zinc pyridine-2-thiol-1-oxide", "zinc pyridine-2-thiol-1-oxide", "zinc 2-pyridinethiol-1-oxide", "zinc 2-pyridinethiol-1-oxide", "zinc pyridinethione", "zinc pyrion", ZnPt, ZPT, OM-1563, "Vancide P"

Section 2 - HAZARDS IDENTIFICATION

CANADIAN WHMIS SYMBOLS

None

EMERGENCY OVERVIEW

RISK

Irritating to skin.
Risk of serious damage to eyes.
Toxic by inhalation, in contact with skin and if swallowed.
Very toxic to aquatic organisms, may cause long- term adverse effects in the aquatic environment.

POTENTIAL HEALTH EFFECTS

ACUTE HEALTH EFFECTS

SWALLOWED

Accidental ingestion of the material may be seriously damaging to the health of the individual; animal experiments indicate that ingestion of less than 40 gram may be fatal. Pyridinethione is a neurotoxin which appears to interfere with the fast axonal transport systems. Rats, rabbits and guinea pigs all develop a distal axonopathy when zinc pyridinethione is a contaminant of their food; this effect is not produced by zinc salts. The earliest signs of myopathy are diminished grip strength and electrophysiological changes of the axon termial with normal conduction along the proximal axon in early stages of exposure. Ultimately, the functional consequence of axonal degeneration is a peripheral neuropathy. Pyridinethione impairs the turnaround of rapidly transported vesicles and slows the retrograde transport of these vesicles. This aberration of the fast axonal transport systems probably produces an accumulation of tubular and vesicular structures in the distal axon. As the materials accumulate, the axon swells. As in many other distal axonmyopathies, the axon degenerates in its more distal regions beyond the accumulated structures. Severe pyrithione ingestion exposure is unlikely because of strong emetic effect. Single doses of sodium pyrithione, independent of the route of administration, produced neurological symptoms such as paralysis of the rear extremities and convulsions in rats, mice and rabbits. When given by mouth in 2% concentration of the zinc pyrithione, as a commercial shampoo, emesis occurs in dogs above 0.05 gm shampoo per kg. Repeated administration of zinc pyrithione produced hind leg paralysis in rabbits and retinal detachment in dogs. These changes do not occur in monkeys. Soluble zinc salts produces irritation and corrosion of the alimentary tract with pain, and vomiting. Death can occur due to insufficiency of food intake due to severe narrowing of the esophagus and pylorus.

EYE

If applied to the eyes, this material causes severe eye damage. Instillation of a 40% (1 ml) aqueous solution of the sodium pyrithione into the eyes of rabbits produced a mild, transient clouding of the cornea in 2/6 animals, 24 hours after the application. In all animals a mild reddening in the conjunctiva developed and in 2/6 animals had not regressed after 72 hours. Conjunctival reddening was seen after application of the powder form.

SKIN

Skin contact with the material may produce toxic effects; systemic effectsmay result following absorption. This material can cause inflammation of the skin oncontact in some persons. The material may accentuate any pre-existing dermatitis condition. Peripheral neuritis with paraesthesia and muscle weakness in a patient were associated with prolonged use of a shampoo (Head and Shoulders) containing 2% zinc pyrithione. Muscle weakness disappeared 3 months

after stopping the use of the shampoo and paraesthesia improved by about 75% in about 2 years. A burning sensation on the skin was reported by 30 of 50 subjects after treatment with a drop of an aqueous solution in concentrations of 1.2%, 1.4% or 2% (pH 9.43) of the sodium salt, applied to the cheek, neck and back of the hand. A 1% solution produced reactions in 5 of 50 individuals; lower concentrations produced no effects. The erythema which developed on 4% to 32% of persons treated with solutions of 1% strength and greater, regressed by the following day. Occlusive application of a 1% aqueous solution of the sodium salt to the healthy skin of 100 volunteers did not produce skin changes. After repeated occlusive applications of a 1% solution to the skin of the upper arms of 100 subjects (24 hours, 15 times during 30 days), one person showed a reaction after the 10th application and one after the 14th (marked erythema, oedema). A solution (0.2 ml, solvent and concentration unspecified) applied occlusively for 24 hours to the healthy skin of 55 persons, 12 times in 3 weeks, produced mild erythema, and in isolated cases, oedema, during the course of the study in 14 individuals. The solution was classified as a weak cumulative irritant. A 1% aqueous solution of the sodium salt applied to the skin of 100 healthy persons in a patch test produced no sensitisation reactions. Neither irritation nor phototoxic reactions nor photosensitisation were seen in 12 men and 13 women treated with a 2% aqueous solution. No reaction was seen in any of 40 workers who handled metal- working fluids and suffered from acute dermatitis on the hands and forearms, following a 48 hr occlusive application of a 1% aqueous solution of sodium pyrithione. Patch tests carried out on 230 metal industry workers with dermatitis on the hands and forearms produced no reactions to a 1% aqueous solution. After dermal application of the sodium salt to rats, muscle damage and paralysis of the hind limbs were observed. The no- observed-effect-level (NOEL) was given as 5 mg/kg body weight. A single dose of sodium pyrithione applied occlusively to the skin of rabbits for 4 hours as a 40% aqueous solution produced mild erythema and oedema; these had regressed after 72 hours. In the Magnusson-Kligman test with guinea pigs, mild to severe erythema developed after intradermal and topical induction with a 4% solution of sodium salt; 24 and 48 hours after provocation with a 2% solution, 2/10 and 3/10 animals reacted with mild erythema. Following application of a 40% solution of sodium pyrithione on 10 occasions, the skin of 12 guinea pigs was irradiated with UVA and UVB light. After the second application, minimal erythema or erythema and oedema was seen in 10 animals. Fourteen days after the last application, provocation was carried out with 4%, 8% and 40% solutions of the sodium salt, followed immediately by irradiation with UVA light. At each concentration one animal reacted with minimal erythema after both 24 and 48 hours. Open cuts, abraded or irritated skin should not be exposed to this material. Entry into the blood-stream, through, for example, cuts, abrasions or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected. This material is a photosensitizer. Certain individuals working with this substance may show allergic reaction of the skin under sunlight. This results in sensitivity to sunburn (may be severe) unless protective covering and 15+PF sunscreen are used. Responses may vary from sunburn-like effects to swelling and blistering lesions.

INHALED

Inhalation of dusts, generated by the material, during the course of normal handling, may produce toxic effects. There is some evidence to suggest that the material can cause respiratory irritation in some persons. The body's response to such irritation can cause further lung

damage. Persons with impaired respiratory function, airway diseases and conditions such as emphysema or chronic bronchitis, may incur further disability if excessive concentrations of particulate are inhaled. Inhalation of pyrithione dust may cause headache, nausea and vomiting. Animals exposed by inhalation to the sodium salt showed reduced body weight gain, reddened lungs and red material around the eyes and noses. Other symptoms included lethargy, tremor, coordination disorders, convulsions and paralysis of the back legs. Animals died 10-14 days after exposure.

CHRONIC HEALTH EFFECTS

There has been some concern that this material can cause cancer or mutations but there is not enough data to make an assessment. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. There is some evidence that inhaling this product is more likely to cause a sensitization reaction in some persons compared to the general population. There is limited evidence that, skin contact with this product is more likely to cause a sensitization reaction in some persons compared to the general population. There is some evidence that human exposure to the material may result in developmental toxicity. This evidence is based on animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects. Medical conditions possibly aggravated by exposure: Diseases of muscles and nerves. [Arch Chemicals]. Prolonged contact may induce allergic dermatitis in sensitive people followed by photosensitivity. [Van Waters] Chronic exposure may result in skeletal muscle atrophy and peripheral nerve damage characterised by general muscle weakness. Diseases of muscle and nerve, e.g. muscular dystrophy, may be aggravated by exposure. Although the mechanism of the neurotoxicity has not been studied, investigations with the zinc salt show that biocide activity is probably due to the inhibition of alcohol dehydrogenase, disturbance of proton gradients in cell membranes, the action of the substance as an antimetabolite of pyridoxal, and production of oxygen radicals during formation of iron complexes. Occasional vomiting was seen in monkeys given oral sodium pyrithione for one year (5, 25 or 75 mg/kg body weight and day). Neurotoxic effects were not seen. In a 90 day study with rats, muscle weakness and histopathological changes in peripheral nerves developed at much lower doses (2 mg/kg body weight and day). These effects were also seen in a two year study with rats especially at 3.5 mg/kg/day, but also at lower doses. Irreversible eye damage and blindness were seen only in dogs, a species with tapetum lucidum (the iridescent pigment which allows the eyes to shine in the dark). Administration of the conjugated acid, pyrithione, in a dose of 25 mg/kg body weight and day for 2 weeks to dogs caused, from the second day of treatment, retinal haemorrhage, oedema and detachment necrosis of the tapetum, and in a few cases, atrophy of the ocular nerve. Oral doses of sodium pyrithione (3.5 mg/kg body weight/day) produced maternal toxicity during and after gestation, caused impaired fertility in males and had adverse effects on mating behaviour. There were no effects on duration of pregnancy, litter size or reproductive organs. Dose-dependent muscle atrophy developed in the upper hind limbs of the parents. Significant malformations were not seen in the off-spring. Earlier studies however showed a reduced number of pregnancies, increased resorptions and embryotoxicity in rats given questionably maternally toxic doses and malformed ribs after maternally toxic doses. Crooked ribs and extremities were seen in the off-spring of rats given sodium pyrithione by epicutaneous

application; this effect was thought to result from maternal toxicity as the dams showed a considerably reduced weight gain. Epicutaneous treatment of rabbits did not produce embryotoxicity or teratogenicity. Welding or flame cutting of metals with zinc or zinc dust coatings may result in inhalation of zinc oxide fume; high concentrations of zinc oxide fume may result in "metal fume fever"; also known as "brass chills", an industrial disease of short duration. [I.L.O] Symptoms include malaise, fever, weakness, nausea and may appear quickly if operations occur in enclosed or poorly ventilated areas. Data from experimental studies indicate that pyridines represent a potential cause of cancer in man. They have also been shown to cross the placental barrier in rats and cause premature delivery, miscarriages and stillbirths. PAs are passed through breast milk. Pyridine has been implicated in the formation of liver cancers. Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).

Section 3 - COMPOSITION / INFORMATION ON INGREDIENTS

NAME	CAS RN	%
zinc pyrithione	13463-41-7	>98

Section 4 - FIRST AID MEASURES

SWALLOWED

· Give a slurry of activated charcoal in water to drink. NEVER GIVE AN UNCONSCIOUS PATIENT WATER TO DRINK.
· At least 3 tablespoons in a glass of water should be given.
· Although induction of vomiting may be recommended (IN CONSCIOUS PERSONS ONLY), such a
first aid measure is dissuaded because to the risk of aspiration of stomach contents. (i) It is better to take the patient to a doctor who can decide on the necessity and method of emptying the stomach. (ii) Special circumstances may however exist; these include non-availability of charcoal and the ready availability of the doctor.
NOTE: If vomiting is induced, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.
NOTE: Wear protective gloves when inducing vomiting.
· REFER FOR MEDICAL ATTENTION WITHOUT DELAY.
· In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition.
· If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the MSDS should be provided. Further action will be the responsibility of the medical specialist.
· If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the MSDS.
(ICSC20305/20307).

EYE

If this product comes in contact with the eyes:
· Immediately hold eyelids apart and flush the eye continuously with running water.
· Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.
· Continue flushing until advised to stop by the Poisons Information Center or a doctor, or for at least 15 minutes.
· Transport to hospital or doctor without delay.
· Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.

SKIN

If skin or hair contact occurs:
· Quickly but gently, wipe material off skin with a dry, clean cloth.
· Immediately remove all contaminated clothing, including footwear.
· Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Center.
· Transport to hospital, or doctor.

INHALED

· If fumes or combustion products are inhaled remove from contaminated area.
· Lay patient down. Keep warm and rested.
· Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.
· Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.
· Transport to hospital, or doctor, without delay.

NOTES TO PHYSICIAN

Treat symptomatically. · Absorption of zinc compounds occurs in the small intestine. · The metal is heavily protein bound. · Elimination results primarily from fecal excretion · The usual measures for decontamination (Ipecac Syrup, lavage, charcoal or cathartics) may be administered, although patients usually have sufficient vomiting not to require them. · CaNa2EDTA has been used successfully to normalize zinc levels and is the agent of choice. [Ellenhorn and Barceloux: Medical Toxicology]. for poisons (where specific treatment regime is absent):

BASIC TREATMENT

· Establish a patent airway with suction where necessary. · Watch for signs of respiratory insufficiency and assist ventilation as necessary. · Administer oxygen by non-rebreather mask at 10 to 15 l/min. · Monitor and treat, where necessary, for pulmonary edema . · Monitor and treat, where necessary, for shock. · Anticipate seizures . · DO NOT use emetics. Where ingestion is

suspected rinse mouth and give up to 200 ml water (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.

ADVANCED TREATMENT

· Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred. · Positive-pressure ventilation using a bag-valve mask might be of use. · Monitor and treat, where necessary, for arrhythmias. · Start an IV D5W TKO. If signs of hypovolemia are present use lactated Ringers solution. Fluid overload might create complications. · Drug therapy should be considered for pulmonary edema. · Hypotension with signs of hypovolemia requires the cautious administration of fluids. Fluid overload might create complications. · Treat seizures with diazepam. · Proparacaine hydrochloride should be used to assist eye irrigation. BRONSTEIN, A.C. and CURRANCE, P.L. EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994.

Section 15 - REGULATORY INFORMATION

REGULATIONS

zinc pyrithione (CAS: 13463-41-7) is found on the following regulatory lists;
Canada Domestic Substances List (DSL)
Canada Ingredient Disclosure List (SOR/88-64)
Canada National Pollutant Release Inventory (NPRI)
Canada Toxicological Index Service - Workplace Hazardous Materials Information System - WHMIS
US - California Air Toxics "Hot Spots" List (Assembly Bill 2588) Substances for which emissions must be
quantified
US - California Environmental Health Standards for the Management of Hazardous Waste - List of Inorganic
Persistent and Bioaccumulative Toxic Substances and Their STLC & TTLC Values
US - California Occupational Safety and Health Regulations (CAL/OSHA) - Hazardous Substances List
US - California OEHHA/ARB - Chronic Reference Exposure Levels and Target Organs (CRELs)
US - California Toxic Air Contaminant List Category II
US ACGIH Biological Exposure Indices (BEI)
US CERCLA List of Hazardous Substances and Reportable Quantities
US CWA (Clean Water Act) - Priority Pollutants
US CWA (Clean Water Act) - Toxic Pollutants
US EPA Carcinogens Listing
US EPA High Production Volume Chemicals 1994 List of Additions
US EPCRA Section 313 Chemical List
US RCRA (Resource Conservation & Recovery Act) - Appendix IX to Part 264 Ground-Water

Monitoring List 1
US RCRA (Resource Conservation & Recovery Act) - List of Hazardous Inorganic and Organic Constituents 1
US Toxic Substances Control Act (TSCA) - Inventory

Section 16 - OTHER INFORMATION

LIMITED EVIDENCE

Cumulative effects may result following exposure*.
May produce discomfort of the respiratory system*.
Limited evidence of a carcinogenic effect*.
Possible respiratory and skin sensitizer*.
May be harmful to the fetus/ embryo*.
* (limited evidence).