西亚试剂® Xiya Reagent 说明书

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化 学 品 安 全 技 术 说 明 书

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MSDS标题

HARMALINE MSDS报告

产品标题

3, 4-二氢骆驼蓬碱;O-甲基骆驼蓬酚

CAS号

304-21-2

化学品及企业标识

PRODUCT NAME

HARMALINE

NFPA

Flammability	1
Toxicity	2
Body Contact	2
Reactivity	1
Chronic	2

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

PRODUCT USE

Alkaloid obtained from peganum, the dried seeds of Peganum harmala (Zygophyllaceae). Found together with harmine in the South American hallucinogenic drink, " caapi" . Produces CNS stimulation. May act through NMDA receptors.

SYNONYMS

C13-H14-N2-O, armalin, dihydroharmine, "3, 4-dihydroharmine", "3, 4-dihydroharmine", "3, 4-dihydro-7-methoxy-1-methyl-3H-pyrido[3, 4-b]indole", "3, 4-dihydro-7-methoxy-1-methyl-3H-pyrido[3, 4-b]indole", "4, 9-dihydro-7-methoxy-1-methyl-3H-pyrido[3, 4-b]indole", "4, 9-dihydro-7-methoxy-1-methyl-3H-pyrido[3, 4-b]indole", harmidine, "harmine, dihydro-", "harmalol methyl ether", o-methylharmalol, o-methylharmalol, "1-methyl-7-methoxy-3, 4-dihydro-beta-carboline", "1-methyl-7-methoxy-3, 4-dihydro-beta-carboline", "hallucinogenic alkaloid"

CANADIAN WHMIS SYMBOLS

EMERGENCY OVERVIEW

RISK

Danger of cumulative effects.

POTENTIAL HEALTH EFFECTS

ACUTE HEALTH EFFECTS

SWALLOWED

Although ingestion is not thought to produce harmful effects, the material may still be damaging to the health of the individual following ingestion, especially where pre- existing organ (e.g. liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality (death) rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern. Adverse effects associated with the administration of central nervous system stimulants include labored breathing, coughing, narrowed airways, chest tightness, and throat spasm. Muscular involvement may produce contraction small localized muscle fibers (visible through the skin) or seizures. Headache, dizziness, fever and confusion may also result. Other symptoms may include nausea, vomiting, diarrhea and difficulty in urination, alterations in blood pressure and irregular heart beat. Morphine and other analgesics cause nausea, vomiting, constipation, drowsiness and confusion. Urination can be difficult, and the bowel and bile ducts can spasm. They also cause dry mouth, pin point pupils, sweating, flushing, vertigo, slow and shallow breathing, weak pulse, blue-gray skin (cyanosis), palpitations, low blood pressure, low temperature, restlessness, and mood changes. Acute toxic effects include lung swelling, spasticity, muscle twitching and unconsciousness. Increased pressure in the head may occur. Larger doses can cause depression of breathing and low blood pressure, with failure of circulation and deepening coma. Failure of breathing can cause death. As the analgesia (loss of sensation) wears off, sensitivity to pain is increased. Higher doses produce stiffening of the

muscles and depression of the central nervous system; this can progress to stupor, sedation, unconsciousness and coma. The blood vessels may dilate, causing flushing of the face, neck and upper chest, and lowering of the blood pressure, resulting in fainting. Serious effects due to toxicity to the heart include high blood pressure, irregular heart rhythms, shock, acute heart failure and stoppage. Hypersensitive reactions can occur, producing rashes, itch, bleeding, and blistering. Digestive effects include constipation, impaction of the bowel with feces and cramps. Urine movements may become less frequent. There may be liver abnormalities, and the liver may be enlarged and tender to touch. The material may bind to the N-methyl-D-aspartate (NMDA) neuroreceptor. The NMDA receptor is an ionotropic glutamate receptor found on post-synaptic neurons and is a membrane channel that regulates the flow of sodium and calcium ions, flowing into the neuron, while potassium ions flow out. The NMDA receptor, therefore, tightly regulates "ion channel conductance". NMDA agonists (receptor activators), such as the glutamates, can, however, be highly toxic to the neuron. Excessive amounts of glutamate or its congeners, can be highly toxic to neurons and may contribute to neuron damage/death in stroke, epilepsy and neurodegenerative diseases. The decreased supply of oxygen (hypoxia) in stroke has been shown to result in excess glutamate release.Overactivation by glutamates, other excitatory amino-acids (EAAs) such as the cysteines and homocysteines, and its congeners (excitotoxins), causes an excessive influx of calcium, into neurons, triggering nervous tissue damage. Glutamate is the major excitatory neurotransmitter in the central nervous system. When concentrations of glutamate and excitotoxins rise above a certain level, in the extracellular fluid, the neuron begins to fire abnormally. At higher concentrations, the cells of the neuron undergo a specialised process of delayed cell death known as excitotoxicity. Although the effects of excitotoxins are generally not dramatic, certain individuals may be especially sensitive and may develop severe symptoms as a result of cardiac irritability.Excess calcium can activate pathways that are potentially harmful to the cell. For example, kinases, phospholipase A2, calpains, NO synthase, endonucleases and other enzymes can be activated. Phospholipase A2 stimulates arachidonic acid production while NO synthase produces nitric oxide. The production of both species ultimately results in free radical damage. Calpain activation may cause breakdown of the cytoskeleton and also contributes to free radical production and lipid peroxidation. Endonucleases damage neuronal DNA, as do free radicals. In addition, high internal calcium ion concentrations create large osmotic forces that drive water into the cell causing swelling and possibly, rupture. Rupture, in turn, causes the release of even more glutamate, inducing excitotoxicity in neighbouring cells. When brain cells are injured, they also release large amounts of glutamate from surrounding astrocytes and this glutamate can produce further damage in adjacent normal neuronal cells. This appears to be the case in strokes, seizures and brain trauma.Activation of calcium-dependent enzymes is thought to produce changes in neuronal function that are long-lasting, persisting for weeks or months; it has been suggested that such activation is responsible for memory. Blockade (antagonism) of the receptor by several chemical agents produces amnesia in laboratory animals.NMDA antagonists have been used as neuroprotective agents counteracting the effects of overactivation of the receptor; however such antagonists may also be harmful, at high doses, as the

neuron also needs calcium for normal function. Very high doses may produce irreversible damage (including the psychomimetic effects caused by PCP - "angel dust"- abuse). Certain NMDA antagonists (notably those used to produce anaesthesis) induce arousal and even seizures. This class of drug has also produced a model psychosis indistinguishable from schizophrenia.Large doses of calcium channel blocking agents may produce nausea, weakness, dizziness, drowsiness, confusion and slurred speech. Marked and prolonged hypotension and bradycardia may result from second or third degree atrioventricular block, decreased cardiac output and junctional rhythms; death may ensue.Certain NMDA receptor antagonists may produce lightheadedness, ataxia, mood elevation and muscle incoordination. Side-effects of uptake of these antagonists (such as the isoxazole derivative, ibotenic acid, isolated from hallucinogenic mushrooms), by neurones, include dizziness, ataxia, euphoria, muscle twitches, and initial psychic stimulations followed by dream-filled sleep. More severe ingestions may produce visual disturbances, fever, confusion, myoclonus, mydriasis, seizures and coma. Residual headache may persist for several days. Ibotenic acid binds to NMDA neurotransmitter and inhibits (antagonises) its action. The congener muscimol (also isolated from mushrooms) which is structurally related to ibotenic acid and glutamic acid, by contrast, binds to another neuroreceptor, the so-called GABA receptor. This receptor, when activated inhibits the firing of some central neurones by causing influx of anions (e.g. chloride) into the cell. Muscimol is a GABA receptor agonist and produces a similar effect and almost identical clinical outcome to that of ibotenic acid. Systemic administration of ibotenic acid and muscimol to laboratory animals produces central inhibition of motor activity with little change to peripheral autonomic activity. Both compounds induce EEG changes in cats, rabbits and rats and thus within the central nervous system both compounds behave as false inhibitory neurotransmitters.There are at least five different NMDA receptor sites that determine whether or not the channel opens. Two important ligands, glutamate and glycine (both amino-acids), are required to bind their respective NMDA sites for the channel to open. At low micromolar concentrations, polyamines, such as dopamine or cholinergic agents (binding to polyamine sites), increase the probability that glutamate and glycine will open the channel; high concentrations of polyamine, in contrast, produce the reverse effect. Two other regulatory ions, magnesium and zinc inhibit the action of amino- acids by binding to sites in the inner pore region of the NMDA channel. Many beta-carbolines are neurotoxic; several are mutagenic. Tetrahydro-beta-carbolines (THBCs) may produce symptoms resembling Parkinson's disease. They may occur naturally or may be produced by the metabolic conversion of synthetic substances such as the heroin contaminant, MPTP. . Almost all cooked or prepared foods contain beta-carbolines (BCs) (9H-pyrido[3,4-b]- indoles) which are analogues of tryptophan or tryptamine. These substances may produce neurological effects. They appear to influence benzodiazepine, serotonine and dopamine receptors in the brain and may modify the effects of other neurotransmitters. Such influence may result in increased secretion and decomposition of dopamine (mimicking physical stress) thus enhancing aggressive behaviour (3-methoxycarbonyl- beta-carboline produces such effects) . Other BCs (such as 3-ethoxycarbonyl- beta-carboline) are hypnotic and anaesthetic and diminish sexual appetite. Sleep may be disturbed (3-hydroxymethyl-beta-carboline). Certain BCs (notably 3-N-methylcarboxamide-

beta-carboline) promote reckless behaviour while others (notably 3-methylcarbonyl-6,7-dimethoxy-4-ethyl- beta-carboline) produce anxiety and suppress immune system activity. Still other BCs may produce sedation (notably 3-ethylcarbonyl-6-benzyloxy-4-methoxymethyl-beta-carboline) beta-Carbolines (heterocyclic amines) may further react with endogenous amines, such as aniline, to produce mutagenic species. Mutagenic activity is partly dependent on how much nitrogen the BC contains.

EYE

Although the material is not thought to be an irritant, direct contact with the eye may produce transient discomfort characterized by tearing or conjunctival redness (as with windburn). The dust may produce eye discomfort causing smarting, pain and redness.

SKIN

Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. The material is not thought to be a skin irritant (as classified using animal models). Temporary discomfort, however, may result from prolonged dermal exposures. Good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting. Toxic effects may result from skin absorption. Contact dermatitis has been reported with morphine and other narcoticanalgesics.

INHALED

Inhalation may produce health damage*. The material is not thought to produce respiratory irritation (as classified using animal models). Nevertheless inhalation of the material, especially for prolonged periods, may produce respiratory discomfort and occasionally, distress. Not normally a hazard due to non-volatile nature of product. 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.

CHRONIC HEALTH EFFECTS

Principal routes of exposure are by accidental skin and eye contact andinhalation of generated dusts. Chronic morphine poisoning or addiction causes pin-point pupils, rapid mood changes and poor social adaptation. As dependence and tolerance occurs, there is an overwhelming need to continue taking the drug or similar drugs and to increase the dose. Prolonged therapy or abuse may cause abnormal lung function, increased body temperature, and kidney failure. Withdrawal symptoms can last for months. Abrupt withdrawal of the opiates may produce yawning, dilated pupils, tears, runny nose, sneezing, muscle tremor, headache, weakness, sweating, anxiety, irritability, disturbed sleep or insomnia, restlessness, orgasm, loss of appetite, nausea, vomiting, loss of weight, diarrhea, dehydration, increase in the number of white blood

cells, bone pain, abdominal and muscle cramps, increase in heart rate, breathing rate and blood pressure, rise in temperature and gooseflesh and blood vessel dilation or constriction.