

化学品安全技术说明书

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**MSDS标题**

HACH OXYSCAV REAGENT 1 POWDER MSDS报告

**产品标题**

乙氨酸;氨基醋酸;氨基乙酸

**CAS号**

56-40-6

**化学品及企业标识**

**PRODUCT NAME**

HACH OXYSCAV REAGENT 1 POWDER

**NFPA**

Flammability	1
Toxicity	2
Body Contact	0
Reactivity	1
Chronic	2

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

**PRODUCT USE**

Laboratory reagent.

# CANADIAN WHMIS SYMBOLS

## EMERGENCY OVERVIEW

### RISK

## POTENTIAL HEALTH EFFECTS

### ACUTE HEALTH EFFECTS

#### SWALLOWED

Accidental ingestion of the material may be damaging to the health of the individual. 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 anaesthesia) 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.

## **EYE**

Although the material is not thought to be an irritant, direct contact with the eye may cause transient discomfort characterized by tearing or conjunctival redness (as with windburn). Slight abrasive damage may also result. The material may produce foreign body irritation in certain individuals.

## **SKIN**

The material is not thought to produce adverse health effects or skin irritation following contact (as classified using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting. 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.

## **INHALED**

The material is not thought to produce either adverse health effects or irritation of the respiratory tract following inhalation (as classified using animal models). Nevertheless, adverse effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.

## **CHRONIC HEALTH EFFECTS**

There is some evidence that inhaling this product is more likely to cause a sensitization reaction in some persons compared to the general population. Long term exposure to high dust concentrations may cause changes in lung function i.e. pneumoconiosis; caused by particles less than 0.5 micron penetrating and remaining in the lung. Prime symptom is breathlessness; lung shadows show on X-ray. Exposure to Sulfonates can cause an imbalance in cellular salts and therefore cellular function. Airborne sulfonates may be responsible for respiratory allergies and, in some instances, minor dermal allergies.