化 学 品 安 全 技 术 说 明 书

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MSDS标题

JOHNSON FREEDOM STRIPPER MSDS报告

产品标题

2-苯氧基乙醇;乙二醇单苯醚

CAS号

122-99-6

化学品及企业标识

PRODUCT NAME

JOHNSON FREEDOM STRIPPER

NFPA

Flammability	0
Toxicity	2
Body Contact	4
Reactivity	2
Chronic	2

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

PRODUCT USE

Industrial/institutional floor cleaner. This product is intended to be diluted prior to use.

SYNONYMS

"floor cleaner", detergent

CANADIAN WHMIS SYMBOLS

EMERGENCY OVERVIEW

RISK

May form explosive peroxides.
Harmful if swallowed.
Causes burns.
Risk of serious damage to eyes.

POTENTIAL HEALTH EFFECTS

ACUTE HEALTH EFFECTS

SWALLOWED

The material can produce chemical burns within the oral cavity and gastrointestinal tract following ingestion. Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual. Ingestion of alkaline corrosives may produce burns around the mouth, ulcerations and swellings of the mucous membranes, profuse saliva production, with an inability to speak or swallow. Both the esophagus and stomach may experience burning pain; vomiting and diarrhea may follow. Epiglottal swelling may result in respiratory distress and asphyxia; shock can occur. Narrowing of the esophagus, stomach or stomach valve may occur immediately or after a long delay (weeks to years). Severe exposure can perforate the esophagus or stomach leading to infections of the chest or abdominal cavity, with low chest pain, abdominal stiffness and fever. All of the above can cause death. At sufficiently high doses the material may be neurotoxic(i.e. poisonous to the nervous system). Ingestion of anionic surfactants may produce diarrhea, bloated stomach,and occasional vomiting.

EYE

The material can produce chemical burns to the eye following direct contact. Vapors or mists may be extremely irritating. If applied to the eyes, this material causes severe eye damage. Direct eye contact with corrosive bases can cause pain and burns. There may be swelling, epithelium destruction, clouding of the cornea and inflammation of the iris. Mild cases often resolve; severe cases can be prolonged with complications such as persistent swelling, scarring, permanent cloudiness, bulging of the eye, cataracts, eyelids glued to the eyeball and blindness. The material may produce severe irritation to the eye causing pronounced inflammation.

Repeated or prolonged exposure to irritants may produce conjunctivitis.

SKIN

The material can produce chemical burns following direct contactwith the skin. Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. The material can produce severe chemical burns following direct contactwith the skin. Skin contact with alkaline corrosives may produce severe pain and burns; brownish stains may develop. The corroded area may be soft, gelatinous and necrotic; tissue destruction may be deep. Absorption by skin may readily exceed vapor inhalation exposure. Symptoms for skin absorption are the same as for inhalation. The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.

INHALED

Not normally a hazard due to non-volatile nature of product. The material can cause respiratory irritation in some persons. The body's response to such irritation can cause further lung damage. Inhaling corrosive bases may irritate the respiratory tract. Symptoms include cough, choking, pain and damage to the mucous membrane. In severe cases, lung swelling may develop, sometimes after a delay of hours to days. There may be low blood pressure, a weak and rapid pulse, and crackling sounds. Inhalation of high concentrations of gas/vapor causes lung irritation with coughing and nausea, central nervous depression with headache and dizziness, slowing of reflexes, fatigue and inco-ordination. The material may produce respiratory tract irritation, and result in damage to the lung including reduced lung function.

CHRONIC HEALTH EFFECTS

Repeated or prolonged exposure to corrosives may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis. 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. Ethylene glycol esters and their ethers cause wasting of the testicles, reproductive changes, infertility and changes to kidney function. Shorter chain compounds are more dangerous. They are also associated with the formation of stones in the urine. Prolonged or chronic exposure to alkanolamines may result in liver, kidney or nervous system injury. Repeated inhalation may aggravate asthma and inflammatory or fibrotic pulmonary disease.Results of repeated exposure tests with diethanolamine (DEA) in

laboratory animals include anaemia (rats) and effects on the kidneys (rats and mice) and liver (mice). DEA produces nervous system injury in dogs and rats. Heart and salivary gland lesions have also been seen in mice treated cutaneously with DEA and in mice receiving DEA in drinking water. Rats given high doses of DEA developed anaemia and testicular lesions.Exaggerated doses of DEA produced heart and nervous system effects in other animals. Changes in other organs were judged to be secondary due to the poor health of animals subjected to extremely high doses of DEA. Rats, rabbits and guinea pigs exposed to high vapour concentrations of volatile monoethanolamine (MEA) (up to 1250 ppm) for periods of up to 5 weeks developed pulmonary, hepatic and renal lesions. Dogs, rats and guinea pigs exposed to 100 ppm MEA for 30 days, became apathetic and developed poor appetites. Animal tests also indicate that inhalation exposure to MEA may result in nervous system injury. All species exposed to airborne MEA experienced dermal effects, varying from ulceration to hair loss probably resulting from contact with the cage.An increased incidence of skeletal variations, suggestive of a slight developmental delay was seen in the foetuses of rats given 1500 mg/kg/day DEA cutaneously; this also produced significant maternal toxicity. No foetal malformations, however, were seen in rats nor in rabbits receiving identical treatment. The foetus of rats given high doses of MEA by gavage, showed an increased rate of embryofoetal death, growth retardation, and some malformations including hydronephrosis and hydroureter. The high doses required to produce these effects bring into question the relevance of this finding to humans. There is some evidence that embryofoetotoxicity and teratogenicity does not occur in rats when MEA is administered by dermal application to the mother.The National Toxicology Program (NTP) concluded that there is clear evidence of liver tumours and some evidence of kidney tumours in mice exposed dermally to DEA over their lifetime. Chronic skin painting studies in mice of both sexes produced liver tumours and an increased incidence of kidney tumours in male mice. The significance of these findings to humans is unclear as DEA is neither genotoxic, mutagenic nor clastogenic, and did not induce tumours in rats or transgenic mice similarly treated. Alkanolamines (especially those containing a secondary amine moiety) may react with nitrites or other nitrosating agents to form carcinogenic N-nitrosamines. Alkanolamines are metabolised by biosynthetic routes to ethanolamine and choline and incorporated into phospholipids. They are excreted predominantly unchanged with a half-life of approximately one week. In the absence of sodium nitrite, no conversion to carcinogenic N-nitrosamines was observed.Diethanolamine competitively inhibits the cellular uptake of choline, in vitro, and hepatic changes in choline homeostasis, consistent with choline deficiency, are observed in vivo.Many amines are potent skin and respiratory sensitisers and certain individuals especially those described as "atopic" (i.e. those predisposed to asthma and other allergic responses) may show allergic reactions when chronically exposed to alkanolamines.In a study with coconut diethanolamide, the National Toxicology Program (Technical Report Series 479), showed clear evidence of carcinogenic activity in male B6C3F1 mice based on increased incidences of hepatic and renal tubule neoplasms and in female B6C3F1 mice based on increased incidences of hepatic neoplasms. There was equivocal evidence of carcinogenic activity in female F344/N rats based on a marginal increase in the incidence of renal tube neoplasms. These increases were associated with the

concentration of free diethanolamine present as a contaminant in the diethanolamine condensate. Exposure to rats to coconut oil diethanolamine condensate by dermal application in ethanol for 2 years resulted in epidermal hyperplasia, sebaceous gland hyperplasia, hyperkeratosis and parakeratosis in males and females and ulcer in females at the site of application. There were increases in the incidences of chronic inflammation, epithelial hyperplasia, and epithelial ulcer in the forestomach of female rats. The severity of nephropathy in dosed female rats were increased. Exposure of mice to coconut oil diethanolamine condensate by dermal application for 2 years resulted in increased incidences of eosinophilic foci of the liver in males. Increased incidences of epidermal hyperplasia, sebaceous gland hyperplasia, and hyperkeratosis in males and females, ulcer in males, and parakeratosis and inflammation in females at the site of application and of follicular cell hyperplasia in the thyroid gland of males and females, were chemical related. 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.