# Maggo

## Troy Laboratories Pty Ltd

Chemwatch Hazard Alert Code: **3**

Chemwatch: **5445-39**
Version No: **2.1.1.1**
Safety Data Sheet according to WHS and ADG requirements

Issue Date: **19/12/2020**
Print Date: **08/04/2021**
L.GHS.AUS.EN

## SECTION 1 Identification of the substance / mixture and of the company / undertaking

### Product Identifier

| | |
|---|---|
| **Product name** | Maggo |
| **Chemical Name** | Not Applicable |
| **Synonyms** | Maggo; ACVM number A005679 |
| **Proper shipping name** | ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains 1,4-dichlorobenzene) |
| **Chemical formula** | Not Applicable |
| **Other means of identification** | Not Available |

### Relevant identified uses of the substance or mixture and uses advised against

| | |
|---|---|
| **Relevant identified uses** | To be used as directed on product label. |

### Details of the supplier of the safety data sheet

| | |
|---|---|
| **Registered company name** | **Troy Laboratories Pty Ltd** |
| **Address** | 37 Glendenning Road Glendenning NSW 2761 Australia |
| **Telephone** | 02 8808 3600 |
| **Fax** | 02 9677 9300 |
| **Website** | www.Troylab.com.au |
| **Email** | admin@troylab.com.au |

### Emergency telephone number

| | |
|---|---|
| **Association / Organisation** | **Troy Laboratories Pty Ltd** |
| **Emergency telephone numbers** | 02 8808 3600 (Office hours (8am – 4pm, Monday to Friday) |
| **Other emergency telephone numbers** | 0800 734 607 (24 hours) |

## SECTION 2 Hazards identification

### Classification of the substance or mixture

COMBUSTIBLE LIQUID, regulated for storage purposes only

| | |
|---|---|
| **Poisons Schedule** | S6 |
| **Classification [1]** | Flammable Liquid Category 4, Acute Toxicity (Oral) Category 4, Aspiration Hazard Category 1, Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 1, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Carcinogenicity Category 2, Acute Aquatic Hazard Category 1, Chronic Aquatic Hazard Category 1 |
| ***Legend:*** | *1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI* |

### Label elements

| | |
|---|---|
| **Hazard pictogram(s)** |  |
| **Signal word** | **Danger** |

### Hazard statement(s)

| | |
|---|---|
| **H227** | Combustible liquid. |
| **H302** | Harmful if swallowed. |
| **H304** | May be fatal if swallowed and enters airways. |
| **H315** | Causes skin irritation. |
| **H318** | Causes serious eye damage. |
| **H336** | May cause drowsiness or dizziness. |
| **H351** | Suspected of causing cancer. |
| **H410** | Very toxic to aquatic life with long lasting effects. |

### Precautionary statement(s) Prevention

| | |
|---|---|
| **P201** | Obtain special instructions before use. |
| **P210** | Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking. |
| **P271** | Use only a well-ventilated area. |
| **P280** | Wear protective gloves/protective clothing/eye protection/face protection/hearing protection/... |
| **P261** | Avoid breathing mist/vapours/spray. |
| **P270** | Do not eat, drink or smoke when using this product. |
| **P273** | Avoid release to the environment. |

### Precautionary statement(s) Response

| | |
|---|---|
| **P301+P310** | IF SWALLOWED: Immediately call a POISON CENTER/doctor/... |
| **P305+P351+P338** | IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing. |
| **P308+P313** | IF exposed or concerned: Get medical advice/attention. |
| **P331** | Do NOT induce vomiting. |
| **P370+P378** | In case of fire: Use alcohol resistant foam or normal protein foam to extinguish. |
| **P391** | Collect spillage. |
| **P301+P312** | IF SWALLOWED: Call a POISON CENTER/doctor/... if you feel unwell |
| **P302+P352** | IF ON SKIN: Wash with plenty of water. |
| **P304+P340** | IF INHALED: Remove person to fresh air and keep comfortable for breathing. |
| **P330** | Rinse mouth. |
| **P332+P313** | If skin irritation occurs: Get medical advice/attention. |
| **P362+P364** | Take off contaminated clothing and wash it before reuse. |

### Precautionary statement(s) Storage

| | |
|---|---|
| **P405** | Store locked up. |
| **P403+P233** | Store in a well-ventilated place. Keep container tightly closed. |

### Precautionary statement(s) Disposal

| | |
|---|---|
| **P501** | Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation. |

## SECTION 3 Composition / information on ingredients

### Substances

See section below for composition of Mixtures

### Mixtures

| CAS No | %[weight] | Name |
|---|---|---|
| 64742-94-5 | 30-60 | solvent naphtha petroleum, heavy aromatic |
| 106-46-7 | 30-60 | 1,4-dichlorobenzene |
| 127087-87-0 | 10-30 | 4-nonylphenol, branched, ethoxylated |
| 31218-83-4 | 1-10 | propetamphos |

## SECTION 4 First aid measures

### Description of first aid measures

| | |
|---|---|
| **Eye Contact** | If this product comes in contact with the eyes:<br>Immediately hold eyelids apart and flush the eye continuously with running water.<br>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.<br>Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes.<br>Transport to hospital or doctor without delay.<br>Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. |
| **Skin Contact** | If skin contact occurs:<br>Immediately remove all contaminated clothing, including footwear.<br>Flush skin and hair with running water (and soap if available).<br>Seek medical attention in event of irritation. |
| **Inhalation** | If fumes or combustion products are inhaled remove from contaminated area.<br>Lay patient down. Keep warm and rested.<br>Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.<br>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.<br>Transport to hospital, or doctor. |
| **Ingestion** | If swallowed do **NOT** induce vomiting.<br>If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.<br>Observe the patient carefully.<br>Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.<br>Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.<br>Seek medical advice.<br>Avoid giving milk or oils.<br>Avoid giving alcohol.<br>If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus. |

### Indication of any immediate medical attention and special treatment needed

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.
Atropine sulfate, usually in doses of 600 microgram may be given intravenously, intramuscularly, or subcutaneously to control the muscarinic effects of choline esterase inhibitors. Supportive treatment may be required.
MARTINDALE: The Extra Pharmacopoeia, Twenty-ninth Edition
While other antimuscarinic agents (e.g., scopolamine) can counteract the effects of cholinesterase inhibitors, their inherent toxic effects in patients who do not have cholinesterase inhibitor poisoning have led to their rejection in favor of atropine. Glycopyrrolate in doses of 1-2 mg, I.V., (0.025 mg/kg in children) has been suggested as an alternative to atropine, and is said to have fewer CNS side effects. However, its use has not been extensively evaluated.
Atropine works by competitively occupying muscarinic receptor sites, thus reducing the effects of excessive acetylcholine on these sites brought about by cholinesterase inhibition.
Atropine is not thought to have significant effect on nicotinic receptors, and thus does not counteract fasciculations, weakness, or flaccid paralysis. Thus, even when given sufficient doses of atropine, patients may need artificial ventilation, sometimes for weeks.
A number of authors have recommended the “atropine challenge” as an aid to diagnosis.
When given to a normal person who has not been exposed to cholinesterase inhibitors, a 2 mg dose of atropine (0.025-0.050/kg in pediatric cases) causes:

- A dry mouth.
- An increase in heart rate of about 35 beats/minute (which is usually not noticed by the recipient) within 3-5 minutes of an I.V. dose, and a maximal increase in heart rate of about 35-45 beats/minute with I.M. or autoinjector administration, respectively, within about 35-45 minutes (the longer being with I.M. injection).
- Blurred near-vision.
- Dry, hot skin.
- Mydriasis (pupillary dilation).

Most of these effects will dissipate within 4-6 hours, except blurred near-vision which may persist for 24 hours.
It has been suggested that when these physiological changes do not occur with this dose (sometimes referred to as an atropine challenge), this is indicative of cholinesterase inhibitor toxicity.

**Cautions**

If miosis (pupillary constriction) is due to direct conjunctival vapor exposure, it is relatively unresponsive to parenteral atropine. Although, it does respond to topical administration).

In 2-13% of cases of cholinesterase inhibitor toxicity, mydriasis (pupillary dilation) --- rather than miosis (pupillary constriction), and tachycardia --- rather than bradycardia (3-77% of cases),.may be a presenting signs.

One author points out that this strategy has never been empirically tested and may not be very sensitive or specific (Parenteral atropine is not generally recommended for those whose sole manifestation of toxicity is miosis (pupillary constriction).

Some cases of mild to moderate poisonings may improve with these doses of atropine. Thus, signs of atropinization do not always exclude the presence of cholinesterase inhibitor toxicity.

In approximate order of preference, the following routes of administration can be used for the administration of atropine

1. Intravenous: bolus, followed by I.V. drip. .
1. Intraosseous: (American Heart Association 2005) bolus, followed by continuous infusion.
1. Military MARK I atropine autoinjector: Although intravenous injection is the preferred route of administration, use of the autoinjector may be more practical in the field, where it can be rapidly administered even through clothing.) Blood levels are achieved more rapidly than by other forms of IM injection. Note that each MARK I kit contains an atropine autoinjector, containing 2 mg of atropine plus another autoinjector containing 600 mg of 2-PAM. Paediatric atropine autoinjector syringes are available in 0.5 mg and 1 mg sizes.
1. Intramuscular: Research for this Case Study did not turn up any comparisons of intramuscular with inhalation routes of atropine administration.
1. Inhalation: by nebulised inhalation or via the intratracheal route. The intratracheal route can be used, but absorption is notably less complete and less reliable than the intravenous or intraosseous routes, which are preferred. The optimal intratracheal dose is unknown, but is typically administered in an amount 2-2½ times the intravenous dose. The American Heart Association recommends that the dose be diluted in 5-10 ml water or normal saline. American Heart Association 2005; American Heart Association 2005)
1. Oral: use has been reported after I.V. administration became unnecessary.
1. Ophthalmic: Anticholinergic eye drops (e.g., atropine or homatropine) have been recommended for severe eye pain caused by miosis (pupillary constriction), and secondary reflex nausea and vomiting, but may result in blurred vision. However, one author questions whether there is enough evidence to recommend this practice.

Tachycardia should not be used as an end-point, because it sometimes is a nicotinic manifestation of toxicity.
Resolution of miosis [Miosis has been defined as pupillary diameter of <3 mm in the dark, along with sluggish or absent response to light] should not be used as an end-point, because:

Miosis (pupillary constriction) from systemic exposure may be a late finding.

When miosis pupillary constriction) is present, it may be resistant to systemic atropine therapy.

Miosis (pupillary constriction) may reflect only localized ophthalmic exposure to vapor without systemic effects.

Pupils are of normal size in a significant minority of poisoned patients (20% in one series).

Toxic patients may present with mydriasis (pupillary dilation) due to occasional dominance of nicotinic effects from cholinesterase inhibitors.

Case Studies in Environmental Medicine (CSEM) Cholinesterase Inhibitors Including Insecticides and Chemical Warfare Nerve Agents Part 4: The Cholinergic Toxidrome; Section 11: Management of the Cholinergic Toxidrome Management Strategy 3: Medications Atropine Agency for Toxic Substance and Disease Registry ATSDR (USA)
Treat symptomatically.
For petroleum distillates

- In case of ingestion, gastric lavage with activated charcoal can be used promptly to prevent absorption - decontamination (induced emesis or lavage) is controversial and should be considered on the merits of each individual case; of course the usual precautions of an endotracheal tube should be considered prior to lavage, to prevent aspiration.
- Individuals intoxicated by petroleum distillates should be hospitalized immediately, with acute and continuing attention to neurologic and cardiopulmonary function.
- Positive pressure ventilation may be necessary.
- Acute central nervous system signs and symptoms may result from large ingestions of aspiration-induced hypoxia.
- After the initial episode,individuals should be followed for changes in blood variables and the delayed appearance of pulmonary oedema and chemical pneumonitis. Such patients should be followed for several days or weeks for delayed effects, including bone marrow toxicity, hepatic and renal impairment Individuals with chronic pulmonary disease will be more seriously impaired, and recovery from inhalation exposure may be complicated.
- Gastrointestinal symptoms are usually minor and pathological changes of the liver and kidneys are reported to be uncommon in acute intoxications.
- Chlorinated and non-chlorinated hydrocarbons may sensitize the heart to epinephrine and other circulating catecholamines so that arrhythmias may occur.Careful consideration of this potential adverse effect should precede administration of epinephrine or other cardiac stimulants and the selection of bronchodilators.

BP America Product Safety & Toxicology Department
Chlorobenzenes are readily adsorbed from the gastrointestinal tract; they are distributed into highly perfused tissues and accumulate in lipid tissues. Lipid accumulation is greatest for the more highly chlorinated chlorobenzene compounds. Chlorobenzenes are metabolised by microsomal oxidation to form arene oxide intermediates and then further to their corresponding chlorophenols which are excreted in the urine as mercapturic acids after conjugation with glutathione or as glucuronic acid or sulfate conjugates. A small percentage are eliminated unchanged in expired air or faeces.
The material may induce methaemoglobinaemia following exposure.

Initial attention should be directed at oxygen delivery and assisted ventilation if necessary. Hyperbaric oxygen has not demonstrated substantial benefits.

Hypotension should respond to Trendelenburg's position and intravenous fluids; otherwise dopamine may be needed.

Symptomatic patients with methaemoglobin levels over 30% should receive methylene blue. (Cyanosis, alone, is not an indication for treatment). The usual dose is 1-2 mg/kg of a 1% solution (10 mg/ml) IV over 50 minutes; repeat, using the same dose, if symptoms of hypoxia fail to subside within 1 hour.

Thorough cleansing of the entire contaminated area of the body, including the scalp and nails, is of utmost importance.

BIOLOGICAL EXPOSURE INDEX - BEI

These represent the determinants observed in specimens collected from a healthy worker exposed at the Exposure Standard (ES or TLV):

| Determinant | Index | Sampling Time | Comment |
|---|---|---|---|
| 1. Methaemoglobin in blood | 1.5% of haemoglobin | During or end of shift | B, NS, SQ |

B: Background levels occur in specimens collected from subjects **NOT** exposed
NS: Non-specific determinant; also observed after exposure to other materials
SQ: Semi-quantitative determinant - Interpretation may be ambiguous; should be used as a screening test or confirmatory test.

## SECTION 5 Firefighting measures

### Extinguishing media

Foam.
Dry chemical powder.
BCF (where regulations permit).
Carbon dioxide.
Water spray or fog - Large fires only.

### Special hazards arising from the substrate or mixture

| | |
|---|---|
| **Fire Incompatibility** | Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result |

### Advice for firefighters

| | |
|---|---|
| **Fire Fighting** | Alert Fire Brigade and tell them location and nature of hazard.<br>Wear full body protective clothing with breathing apparatus.<br>Prevent, by any means available, spillage from entering drains or water course.<br>Use water delivered as a fine spray to control fire and cool adjacent area.<br>Avoid spraying water onto liquid pools.<br>**DO NOT** approach containers suspected to be hot.<br>Cool fire exposed containers with water spray from a protected location.<br>If safe to do so, remove containers from path of fire. |
| **Fire/Explosion Hazard** | Combustible.<br>Slight fire hazard when exposed to heat or flame.<br>Heating may cause expansion or decomposition leading to violent rupture of containers.<br>On combustion, may emit toxic fumes of carbon monoxide (CO).<br>May emit acrid smoke.<br>Mists containing combustible materials may be explosive.<br>Combustion products include:<br>carbon dioxide ($CO_2$)<br>hydrogen chloride<br>phosgene<br>phosphorus oxides (POx)<br>sulfur oxides (SOx)<br>other pyrolysis products typical of burning organic material. |
| **HAZCHEM** | •3Z |

## SECTION 6 Accidental release measures

### Personal precautions, protective equipment and emergency procedures

See section 8

### Environmental precautions

See section 12

### Methods and material for containment and cleaning up

| | |
|---|---|
| **Minor Spills** | Environmental hazard - contain spillage.<br>Clean up all spills immediately.<br>Avoid breathing vapours and contact with skin and eyes.<br>Control personal contact with the substance, by using protective equipment.<br>Contain and absorb spill with sand, earth, inert material or vermiculite.<br>Wipe up.<br>Place in a suitable, labelled container for waste disposal. |
| **Major Spills** | Environmental hazard - contain spillage.<br>Moderate hazard.<br>Clear area of personnel and move upwind. |

| | |
|---|---|
| | Alert Fire Brigade and tell them location and nature of hazard.<br>Wear breathing apparatus plus protective gloves.<br>Prevent, by any means available, spillage from entering drains or water course.<br>No smoking, naked lights or ignition sources.<br>Increase ventilation.<br>Stop leak if safe to do so.<br>Contain spill with sand, earth or vermiculite.<br>Collect recoverable product into labelled containers for recycling.<br>Absorb remaining product with sand, earth or vermiculite.<br>Collect solid residues and seal in labelled drums for disposal.<br>Wash area and prevent runoff into drains.<br>If contamination of drains or waterways occurs, advise emergency services. |

Personal Protective Equipment advice is contained in Section 8 of the SDS.

## SECTION 7 Handling and storage

### Precautions for safe handling

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|---|---|
| **Safe handling** | The conductivity of this material may make it a static accumulator., A liquid is typically considered nonconductive if its conductivity is below 100 pS/m and is considered semi-conductive if its conductivity is below 10 000 pS/m., Whether a liquid is nonconductive or semi-conductive, the precautions are the same., A number of factors, for example liquid temperature, presence of contaminants, and anti-static additives can greatly influence the conductivity of a liquid.<br>Containers, even those that have been emptied, may contain explosive vapours.<br>Do NOT cut, drill, grind, weld or perform similar operations on or near containers.<br>**DO NOT** allow clothing wet with material to stay in contact with skin<br>Avoid all personal contact, including inhalation.<br>Wear protective clothing when risk of exposure occurs.<br>Use in a well-ventilated area.<br>Prevent concentration in hollows and sumps.<br>**DO NOT** enter confined spaces until atmosphere has been checked.<br>Avoid smoking, naked lights or ignition sources.<br>Avoid contact with incompatible materials.<br>When handling, **DO NOT** eat, drink or smoke.<br>Keep containers securely sealed when not in use.<br>Avoid physical damage to containers.<br>Always wash hands with soap and water after handling.<br>Work clothes should be laundered separately.<br>Use good occupational work practice.<br>Observe manufacturer's storage and handling recommendations contained within this SDS.<br>Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions. |
| **Other information** | Store in original containers.<br>Keep containers securely sealed.<br>Store in a cool, dry, well-ventilated area.<br>Store away from incompatible materials and foodstuff containers.<br>Protect containers against physical damage and check regularly for leaks.<br>Observe manufacturer's storage and handling recommendations contained within this SDS. |

### Conditions for safe storage, including any incompatibilities

| | |
|---|---|
| **Suitable container** | Avoid contact with aluminium and its alloys (including storage containers). Formation of aluminium chloride may catalyse further self-accelerating attack on the metal (Friedel-Crafts reaction) leading to violent explosion.<br>**DO NOT** use aluminium or galvanised containers<br>Metal can or drum<br>Packaging as recommended by manufacturer.<br>Check all containers are clearly labelled and free from leaks. |
| **Storage incompatibility** | For alkyl aromatics:<br>The alkyl side chain of aromatic rings can undergo oxidation by several mechanisms. The most common and dominant one is the attack by oxidation at benzylic carbon as the intermediate formed is stabilised by resonance structure of the ring.<br>Following reaction with oxygen and under the influence of sunlight, a hydroperoxide at the alpha-position to the aromatic ring, is the primary oxidation product formed (provided a hydrogen atom is initially available at this position) - this product is often short-lived but may be stable dependent on the nature of the aromatic substitution; a secondary C-H bond is more easily attacked than a primary C-H bond whilst a tertiary C-H bond is even more susceptible to attack by oxygen<br>Monoalkylbenzenes may subsequently form monocarboxylic acids; alkyl naphthalenes mainly produce the corresponding naphthalene carboxylic acids.<br>Oxidation in the presence of transition metal salts not only accelerates but also selectively decomposes the hydroperoxides.<br>Hock-rearrangement by the influence of strong acids converts the hydroperoxides to hemiacetals. Peresters formed from the hydroperoxides undergo Criegee rearrangement easily.<br>Alkali metals accelerate the oxidation while CO2 as co-oxidant enhances the selectivity. |

Microwave conditions give improved yields of the oxidation products.
Photo-oxidation products may occur following reaction with hydroxyl radicals and NOx - these may be components of photochemical smogs.
Oxidation of Alkylaromatics: T.S.S Rao and Shubhra Awasthi: E-Journal of Chemistry Vol 4, No. 1, pp 1-13 January 2007
Vigorous reactions, sometimes amounting to explosions, can result from the contact between aromatic rings and strong oxidising agents.
Aromatics can react exothermically with bases and with diazo compounds.
Haloaryl compounds (halogenated aromatics), though normally not very reactive, may be sufficiently activated by other substituents or by a few specific reaction conditions, to undergo violent reactions.
BRETHERICK L.: Handbook of Reactive Chemical Hazards
Avoid contact with aluminium and its alloys (including storage containers). Formation of aluminium chloride may catalyse further self-accelerating attack on the metal (Friedel-Crafts reaction) leading to violent explosion.

## SECTION 8 Exposure controls / personal protection

### Control parameters

**Occupational Exposure Limits (OEL)**

**INGREDIENT DATA**

| Source | Ingredient | Material name | TWA | STEL | Peak | Notes |
|---|---|---|---|---|---|---|
| Australia Exposure Standards | 1,4-dichlorobenzene | p-Dichlorobenzene | 25 ppm / 150 mg/m3 | 300 mg/m3 / 50 ppm | Not Available | Not Available |

**Emergency Limits**

| Ingredient | TEEL-1 | TEEL-2 | TEEL-3 |
|---|---|---|---|
| 1,4-dichlorobenzene | 30 ppm | 170 ppm | 1,000 ppm |
| 4-nonylphenol, branched, ethoxylated | 30 mg/m3 | 330 mg/m3 | 2,000 mg/m3 |
| 4-nonylphenol, branched, ethoxylated | 30 mg/m3 | 330 mg/m3 | 2,000 mg/m3 |

| Ingredient | Original IDLH | Revised IDLH |
|---|---|---|
| solvent naphtha petroleum, heavy aromatic | Not Available | Not Available |
| 1,4-dichlorobenzene | 150 ppm | Not Available |
| 4-nonylphenol, branched, ethoxylated | Not Available | Not Available |
| propetamphos | Not Available | Not Available |

**Occupational Exposure Banding**

| Ingredient | Occupational Exposure Band Rating | Occupational Exposure Band Limit |
|---|---|---|
| 4-nonylphenol, branched, ethoxylated | E | ≤ 0.1 ppm |
| propetamphos | E | ≤ 0.1 ppm |
| ***Notes:*** | *Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.* | |

**MATERIAL DATA**

### Exposure controls

| | |
|---|---|
| **Appropriate engineering controls** | Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.<br>The basic types of engineering controls are:<br>Process controls which involve changing the way a job activity or process is done to reduce the risk.<br>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.<br>Employers may need to use multiple types of controls to prevent employee overexposure. |

Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.
An approved self contained breathing apparatus (SCBA) may be required in some situations.
Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

| Type of Contaminant: | Air Speed: |
|---|---|
| solvent, vapours, degreasing etc., evaporating from tank (in still air). | 0.25-0.5 m/s (50-100 f/min.) |
| aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation) | 0.5-1 m/s (100-200 f/min.) |
| direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion) | 1-2.5 m/s (200-500 f/min.) |
| grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion). | 2.5-10 m/s (500-2000 f/min.) |

Within each range the appropriate value depends on:

| Lower end of the range | Upper end of the range |
|---|---|
| 1: Room air currents minimal or favourable to capture | 1: Disturbing room air currents |
| 2: Contaminants of low toxicity or of nuisance value only. | 2: Contaminants of high toxicity |
| 3: Intermittent, low production. | 3: High production, heavy use |
| 4: Large hood or large air mass in motion | 4: Small hood-local control only |

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

**Personal protection**

**Eye and face protection**

Safety glasses with side shields.
Chemical goggles.
Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]

**Skin protection**

See Hand protection below

**Hands/feet protection**

Wear chemical protective gloves, e.g. PVC.
Wear safety footwear or safety gumboots, e.g. Rubber

**NOTE:**

The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.
Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:

- frequency and duration of contact,
- chemical resistance of glove material,
- glove thickness and

| | |
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| | · dexterity<br>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).<br>· When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.<br>· When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.<br>· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.<br>· Contaminated gloves should be replaced.<br>As defined in ASTM F-739-96 in any application, gloves are rated as:<br>· Excellent when breakthrough time > 480 min<br>· Good when breakthrough time > 20 min<br>· Fair when breakthrough time < 20 min<br>· Poor when glove material degrades<br>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.<br>It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.<br>Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task.<br>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:<br>· Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.<br>· Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential<br>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. |
| **Body protection** | See Other protection below |
| **Other protection** | Overalls.<br>P.V.C apron.<br>Barrier cream.<br>Skin cleansing cream.<br>Eye wash unit. |

### Recommended material(s)

**GLOVE SELECTION INDEX**

Glove selection is based on a modified presentation of the:
***"Forsberg Clothing Performance Index".***
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
Maggo

| Material | CPI |
|---|---|
| NEOPRENE | B |
| NITRILE | C |
| PVC | C |

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
**NOTE**: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

### Respiratory protection

Type A-P Filter of sufficient capacity. (AS/NZS 1716 & 1715, EN 143:2000 & 149:2001, ANSI Z88 or national equivalent)

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required. Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

| Required Minimum Protection Factor | Half-Face Respirator | Full-Face Respirator | Powered Air Respirator |
|---|---|---|---|
| up to 5 x ES | A-AUS / Class 1 P2 | - | A-PAPR-AUS / Class 1 P2 |
| up to 25 x ES | Air-line* | A-2 P2 | A-PAPR-2 P2 |
| up to 50 x ES | - | A-3 P2 | - |
| 50+ x ES | - | Air-line** | - |

^ - Full-face
A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the

humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

## SECTION 9 Physical and chemical properties

### Information on basic physical and chemical properties

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| **Appearance** | Straw coloured liquid; emulsifies in water. |

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| **Physical state** | Liquid | **Relative density (Agua= 1)** | Not Available |
| **Odour** | Not Available | **Partition coefficient n-octanol / water** | Not Available |
| **Odour threshold** | Not Available | **Auto-ignition temperature (°C)** | Not Available |
| **pH (as supplied)** | Not Available | **Decomposition temperature** | Not Available |
| **Melting point / freezing point (°C)** | Not Available | **Viscosity (cSt)** | Not Available |
| **Initial boiling point and boiling range (°C)** | Not Available | **Molecular weight (g/mol)** | Not Applicable |
| **Flash point (°C)** | 70 | **Taste** | Not Available |
| **Evaporation rate** | Not Available | **Explosive properties** | Not Available |
| **Flammability** | Combustible. | **Oxidising properties** | Not Available |
| **Upper Explosive Limit (%)** | Not Available | **Surface Tension (dyn/cm or mN/m)** | Not Available |
| **Lower Explosive Limit (%)** | Not Available | **Volatile Component (%vol)** | Not Available |
| **Vapour pressure (kPa)** | Not Available | **Gas group** | Not Available |
| **Solubility in water** | Partly miscible | **pH as a solution (1%)** | Not Available |
| **Vapour density (Air = 1)** | Not Available | **VOC g/L** | Not Available |

## SECTION 10 Stability and reactivity

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| **Reactivity** | See section 7 |
| **Chemical stability** | Unstable in the presence of incompatible materials.<br>Product is considered stable.<br>Hazardous polymerisation will not occur. |
| **Possibility of hazardous reactions** | See section 7 |
| **Conditions to avoid** | See section 7 |
| **Incompatible materials** | See section 7 |
| **Hazardous decomposition products** | See section 5 |

## SECTION 11 Toxicological information

### Information on toxicological effects

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| **Inhaled** | Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.<br>Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.<br><br>High inhaled concentrations of mixed hydrocarbons may produce narcosis characterised by nausea, vomiting and lightheadedness. Inhalation of aerosols may produce severe pulmonary oedema, pneumonitis and pulmonary haemorrhage. Inhalation of petroleum hydrocarbons consisting substantially of low molecular weight species (typically C2-C12) may produce irritation of mucous membranes, incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, |

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| | tremors and anaesthetic stupor. Massive exposures may produce central nervous system depression with sudden collapse and deep coma; fatalities have been recorded. Irritation of the brain and/or apnoeic anoxia may produce convulsions. Although recovery following overexposure is generally complete, cerebral micro-haemorrhage of focal post-inflammatory scarring may produce epileptiform seizures some months after the exposure. Pulmonary episodes may include chemical pneumonitis with oedema and haemorrhage. The lighter hydrocarbons may produce kidney and neurotoxic effects. Pulmonary irritancy increases with carbon chain length for paraffins and olefins. Alkenes produce pulmonary oedema at high concentrations. Liquid paraffins may produce anaesthesia and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce polyneuropathy. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue and vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons are cardiac sensitisers and may cause ventricular fibrillations.<br>Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.<br>The physiological response to 1,4-dichlorobenzene (DCB )is primarily injury to the liver and secondarily to the kidneys. Central nervous system depression will occur at concentrations that are extremely objectionable to the eyes and nose.<br>Individuals exposed to higher concentrations may show weakness, dizziness and weight loss. Vomiting may occur. Acute haemolytic anaemia with methaemoglobinaemia has been reported.<br>Prolonged inhalation exposure may cause dizziness, headache nausea, vomiting, central nervous system depression and damage to liver and kidneys.<br>In two human fatalities believed to be caused by 1,4-DCB inhalation, the subjects died of massive hepatic (liver) necrosis; the exposure concentrations are not known. A 3 year-old child who had been playing with crystals containing 1,4-DCB for 4-5 days was jaundiced with pale mucous membranes, indicative of liver damage. A case of pulmonary granulomatosis was reported to have occurred in a 53-year-old woman who for 12-15 years had been inhaling 1,4-DCB crystals that were scattered on a weekly basis on the carpets and furniture of her home. A lung biopsy revealed the presence of 1,4-DCB crystals with the surrounding lung parenchyma being distorted (by fibrosis, thickening of the alveolar walls, and marked infiltrates of lymphocytes and mononuclear phagocytes). These effects are most likely related to the physical interaction of 1,4-DCB crystals (or any crystals when inhaled) with lung tissue, rather than to chemical toxicity. A health survey of 58 men occupationally exposed to 1,4-DCB for 8 hours/day, 5 days/week for 8 months to 25 years (average, 4.75 years) found the odor to be faint at 15-30 ppm and strong at 30-60 ppm, with painful irritation of the nose and eyes usually occurring at concentrations ranging from 80 to 160 ppm. At levels >160 ppm, the air was considered not breathable for unacclimated persons.<br>Rabbits exposed 8 hours/day for a total of 62 exposures in 83 days at 770-800 ppm exhibited tremors, weakness, and death along with oedema of the cornea and opacity of the lens.<br>In male mice exposed to 1,2-DCB in mean concentrations of 0, 64, or 163 ppm for 6 hours/day,<br>5 days/week for 4, 9, or 14 days, histopathologic lesions were observed in the olfactory epithelium of the nasal cavity at >64 ppm. The olfactory epithelial lesions were graded as very severe following the 4-day exposure and moderate after the 14 day exposure, indicating to the study authors that repair may occur despite continued exposure. The more severe cases were characterized by a complete loss of olfactory epithelium, which left only partially denuded basement membrane. No histological alterations were<br>observed in the respiratory epithelium of the nasal cavity, or in the trachea or lungs.<br>Mouse exposed to the saturated vapour (calculated as between 2000 and 3000 ppm) showed prompt narcosis, followed by central respiratory depression and cyanosis - death occurred within 24 hours. 8000 ppm produced sedation in dogs exposed for 1 hour. Rats exposed at a concentration of 450 ppm, 6 hours/day for up to 13 days showed pale, discoloured kidneys<br>Rats survived inhalation exposure for 2 hours at 977 ppm but died after 7 hour exposure. Rats surviving a 7 hour exposure at 539 ppm showed liver necrosis and kidney tubule damage. Liver damage was evident in other rats exposed from 50 to 800 ppm and during exposures lasting 0.5 and 1 hour at 390 ppm.<br>Following a single or multiple 3-hour inhalation exposures of radiolabelled 1,4-DCB in rats, label was detected in all evaluated tissues (liver, kidneys, lungs, muscle, fat, and blood plasma), indicating that considerable absorption had occurred. Levels of label in tissues did not appreciably increase with increasing the number of exposures beyond one. Similarly, following a single 24-hour inhalation exposure in rats, 1,4-DCB levels in the liver, kidney, fat, and blood increased sharply during the first 6-hour evaluation period, then rose slowly but steadily for the remainder of the exposure period, indicating an initial rapid absorption, followed by a slower total absorption as equilibration of body and blood levels is approached.<br>The acute toxicity of inhaled alkylbenzenes is best described by central nervous system depression. As a rule, these compounds may also act as general anaesthetics.<br>Systemic poisoning produced by general anaesthesia is characterised by lightheadedness, nervousness, apprehension, euphoria, confusion, dizziness, drowsiness, tinnitus, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness and respiratory depression and arrest. Cardiac arrest may result from cardiovascular collapse. Bradycardia, and hypotension may also be produced.<br>Inhaled alkylbenzene vapours cause death in animals at air levels that are relatively similar (typically LC50s are in the range 5000 -8000 ppm for 4 to 8 hour exposures). It is likely that acute inhalation exposure to alkylbenzenes resembles that to general anaesthetics.<br>Alkylbenzenes are not generally toxic other than at high levels of exposure. This may be because their metabolites have a low order of toxicity and are easily excreted. There is little or no evidence to suggest that metabolic pathways can become saturated leading to spillover to alternate pathways. Nor is there evidence that toxic reactive intermediates, which may produce subsequent toxic or mutagenic effects, are formed<br><br>Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual. |
| **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. |

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| | Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result.<br>Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).<br>Nonionic surfactants may produce localised irritation of the oral or gastrointestinal mucosa and induce vomiting and mild diarrhoea.<br>The substance and/or its metabolites may bind to haemoglobin inhibiting normal uptake of oxygen. This condition, known as "methaemoglobinemia", is a form of oxygen starvation (anoxia).<br>Symptoms include cyanosis (a bluish discolouration skin and mucous membranes) and breathing difficulties. Symptoms may not be evident until several hours after exposure.<br>At about 15% concentration of blood methaemoglobin there is observable cyanosis of the lips, nose and earlobes. Symptoms may be absent although euphoria, flushed face and headache are commonly experienced. At 25-40%, cyanosis is marked but little disability occurs other than that produced on physical exertion. At 40-60%, symptoms include weakness, dizziness, lightheadedness, increasingly severe headache, ataxia, rapid shallow respiration, drowsiness, nausea, vomiting, confusion, lethargy and stupor. Above 60% symptoms include dyspnea, respiratory depression, tachycardia or bradycardia, and convulsions. Levels exceeding 70% may be fatal.<br>Ingestion of petroleum hydrocarbons may produce irritation of the pharynx, oesophagus, stomach and small intestine with oedema and mucosal ulceration resulting; symptoms include a burning sensation in the mouth and throat. Large amounts may produce narcosis with nausea and vomiting, weakness or dizziness, slow and shallow respiration, swelling of the abdomen, unconsciousness and convulsions. Myocardial injury may produce arrhythmias, ventricular fibrillation and electrocardiographic changes. Central nervous system depression may also occur. Light aromatic hydrocarbons produce a warm, sharp, tingling sensation on contact with taste buds and may anaesthetise the tongue. Aspiration into the lungs may produce coughing, gagging and a chemical pneumonitis with pulmonary oedema and haemorrhage.<br>Adverse effects of choline esterase inhibitors include nausea, vomiting, abdominal pain, flushing, sweating, salivation, lachrymation, rhinorrhoea, eruction, involuntary defecation, and urination, bradycardia, and peripheral vasodilation leading to hypotension, transient heart block, bronchioconstriction and a feeling of constriction beneath the chest.<br>Acute-, intermediate,- and chronic-duration inhalation and oral studies of dichlorobenzenes (DCBs) clearly identify the liver as a sensitive target of oral exposure, inducing increases in liver weight at low levels of exposure and histological changes such as cloudy swelling and centrilobular degeneration and necrosis at higher levels in rats and mice. In rats exposed to 455 mg/kg/day for 15 days 1,2-DCB, severe liver damage, characterised by intense necrosis and fatty changes and porphyria, were reported. Large doses have caused tremor in exposed animals; insects exhibit symptoms resembling DDT poisoning.<br>1,2-DCB is a strong central nervous system depressant. 1,2-DCB is quickly and extensively absorbed through both the gastrointestinal tract and the respiratory tract; studies measuring the absorption of 1,2-DCB following dermal exposure are not available. Following absorption, 1,2-dichlorobenzene (1,2-DCB) is distributed throughout the body, but tends to be found in greatest levels in the fat, kidney, and liver. Metabolism is believed to occur mainly in the liver, but may occur at lower levels in other tissues, such as the kidney or lung. Elimination of 1,2-DCB from the body is rapid, with the majority of a single dose being removed within the first 75 hours postexposure; elimination occurs primarily in the urine as metabolites<br>Information on the oral toxicity of 1,3-DCB in animals is available from one 90-day systemic toxicity study and one developmental toxicity study. The intermediate-duration study found effects in the thyroid, pituitary, and liver of rats, with thyroid lesions occurring at dose levels lower than those inducing pituitary and liver effects.<br>Hepatic porphyria was produced in rats following seven consecutive doses of 770 mg 1,4-DCB/kg. Slight to moderate corneal opacity was noted in rabbits following 3 weeks of daily dosing with 5000 mg/kg 1,4-DCB. Rats receiving a daily dose of 500 mg/kg 1,4-DCB for 20 days showed cloudy swelling and necrosis in the central areas of the liver lobules and swelling of the renal tubular epithelium. 100 mg/kg daily doses did not reproduce this finding. Pale and mottled kidneys were seen in rats given oral doses of 70 to 428 mg/kg/day, 1,4-DCB for 28 days. Rats given 1200 mg/kg for 13 weeks showed degeneration and necrosis of hepatocytes, hypoplasia of the bone marrow, lymphoid depletion of the spleen and thymus, and epithelial necrosis of the nasal turbinates and small intestinal mucosa. At doses of 300 mg/kg 1,4-DCB male rats showed kidney damage characterised by degeneration or necrosis of the renal cortical tubular epithelial cells. Female rats did not show these lesions even at doses of 1500 mg/kg<br>Oral doses of 500 mg 1,2-DCB given over 13- weeks to mice and rats produced necrosis and hepatocellular degeneration and depletion of lymphocytes in both the spleen and thymus and renal tubular degeneration in rats. Multifocal mineralisation of the myocardial fibers of the heart and skeletal muscle was seen in mice. Necrosis of individual hepatocytes was seen in female mice given 250 mg/kg. At 125 mg/kg a few rats exhibited minimal hepatocellular necrosis. |
| **Skin Contact** | Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.<br>The material may accentuate any pre-existing dermatitis condition<br>Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.<br>Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.<br>One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been established that there is a connection between the potential of surfactants to denature protein in vitro and their effect on the skin. Nonionic surfactants do not carry any net charge and, therefore, they can only form hydrophobic bonds with proteins. For this reason, proteins are not deactivated by nonionic surfactants, and proteins with poor solubility are not solubilized by nonionic surfactants<br>1,2-dichlorobenzene (DCB) was irritating when applied to the skin of human subjects for 15-60 minutes. One worker developed a dermatitis following hand contact that was reported as sensitisation after a follow-up patch test. Two subjects reported a burning |

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| | sensation during a 1 hour exposure. A diffuse redness of the treated area progressed to a darker red colour with blister formation within 24 hours. A brown pigment formed at the site which was apparent 3 months postexposure<br>Open cuts, abraded or irritated skin should not be exposed to this material<br>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds 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.<br><br>Absorption by skin may readily exceed vapour inhalation exposure. Symptoms for skin absorption are the same as for inhalation.<br>Aromatic hydrocarbons may produce skin irritation, vasodilation with erythema and changes in endothelial cell permeability.<br>Systemic intoxication, resulting from contact with the light aromatics, is unlikely due to the slow rate of permeation. Branching of the side chain appears to increase percutaneous absorption. |
| **Eye** | When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.<br><br>Some nonionic surfactants may produce a localised anaesthetic effect on the cornea; this may effectively eliminate the warning discomfort produced by other substances and lead to corneal injury. Irritant effects range from minimal to severe dependent on the nature of the surfactant, its concentration and the duration of contact. Pain and corneal damage represent the most severe manifestation of irritation.<br>Undiluted 1,2-dichlorobenzene (DCB) applied to rabbit eye caused pain and slight conjunctival irritation. Irritation cleared within 5 days without residual injury.<br>Vapours from heated 1,4-DCB may cause mild corneal damage. Solid particles of 1,4-CB in the eye are reported to be very painful. At workplace concentrations ranging from 50-170 ppm 1,4-DCB, periodic medical examination found no evidence of adverse effects in workers with particular reference to ocular lesions including cataracts. Painful irritation of eyes and nose has been recorded at 80-160 ppm 1,4-DCB<br><br>Petroleum hydrocarbons may produce pain after direct contact with the eyes. Slight, but transient disturbances of the corneal epithelium may also result. The aromatic fraction may produce irritation and lachrymation. |
| **Chronic** | On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.<br>Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.<br>There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals.<br>Repeated or prolonged exposures to cholinesterase inhibitors produce symptoms similar to acute effects. In addition workers exposed repeatedly to these substances may exhibit impaired memory and loss of concentration, severe depression and acute psychosis, irritability, confusion, apathy, emotional lability, speech difficulties, headache, spatial disorientation, delayed reaction times, sleepwalking, drowsiness or insomnia. An influenza-like condition with nausea, weakness, anorexia and malaise has been described. There is a growing body of evidence from epidemiological studies and from experimental laboratory studies that short-term exposure to some cholinesterase-inhibiting insecticides may produce behavioural or neuro-chemical changes lasting for days or months, presumably outlasting the cholinesterase inhibition. Although the number of adverse effects following humans poisonings subsides, there are still effects in some workers months after cholinesterase activity returns to normal. These long-lasting effects include blurred vision, headache, weakness, and anorexia. The neurochemistry of animals exposed to chlorpyrifos or fenthion is reported to be altered permanently after a single exposure. These effects may be more severe in developing animals where both acetyl- and butyrylcholinesterase may play an integral part in the development of the nervous system.<br><br>*Padilla S., The Neurotoxicity of Cholinesterase-Inhibiting Insecticides: Past and Present Evidence Demonstrating Persistent Effects. Inhalation Toxicology 7:903-907, 1995*<br>Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/or memory loss, tremor in the fingers and tongue, vertigo, olfactory disorders, constriction of visual field, paraesthesias of the extremities, weight loss and anaemia and degenerative changes in the liver and kidney. Chronic exposure by petroleum workers, to the lighter hydrocarbons, has been associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness and paraesthesias), psychological and neurophysiological deficits, bone marrow toxicities (including hypoplasia possibly due to benzene) and hepatic and renal involvement. Chronic dermal exposure to petroleum hydrocarbons may result in defatting which produces localised dermatoses. Surface cracking and erosion may also increase susceptibility to infection by microorganisms. One epidemiological study of petroleum refinery workers has reported elevations in standard mortality ratios for skin cancer along with a dose-response relationship indicating an association between routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have been unable to confirm this finding.<br>Hydrocarbon solvents are liquid hydrocarbon fractions derived from petroleum processing streams, containing only carbon and hydrogen atoms, with carbon numbers ranging from approximately C5-C20 and boiling between approximately 35-370 deg C. Many of the hydrocarbon solvents have complex and variable compositions with constituents of 4 types, alkanes (normal paraffins, isoparaffins, and cycloparaffins) and aromatics (primarily alkylated one- and two-ring species). Despite the compositional complexity, most hydrocarbon solvent constituents have similar toxicological properties, and the overall toxicological hazards can be characterized in generic terms. Hydrocarbon solvents can cause chemical pneumonitis if aspirated into the lung, and those that are volatile can cause acute CNS effects and/or ocular and respiratory irritation at exposure levels exceeding occupational recommendations. Otherwise, there are few toxicologically important effects. The exceptions, n-hexane and naphthalene, have unique toxicological properties<br><br>Animal studies:<br>No deaths or treatment related signs of toxicity were observed in rats exposed to light alkylate naphtha (paraffinic hydrocarbons) at concentrations of 668, 2220 and 6646 ppm for 6 hrs/day, 5 days/wk for 13 weeks. Increased liver weights and kidney toxicity |

(male rats) was observed in high dose animals. Exposure to pregnant rats at concentrations of 137, 3425 and 6850 ppm did not adversely affect reproduction or cause maternal or foetal toxicity. Lifetime skin painting studies in mice with similar naphthas have shown weak or no carcinogenic activity following prolonged and repeated exposure. Similar naphthas/distillates, when tested at nonirritating dose levels, did not show any significant carcinogenic activity indicating that this tumorigenic response is likely related to chronic irritation and not to dose. The mutagenic potential of naphthas has been reported to be largely negative in a variety of mutagenicity tests. The exact relationship between these results and human health is not known. Some components of this product have been shown to produce a species specific, sex hormonal dependent kidney lesion in male rats from repeated oral or inhalation exposure. Subsequent research has shown that the kidney damage develops via the formation of a alpha-2u-globulin, a mechanism unique to the male rat. Humans do not form alpha-2u-globulin, therefore, the kidney effects resulting from this mechanism are not relevant in human.
Chronic inhalation exposure to dichlorobenzenes (DCBs) may cause changes to liver and kidney and haematological (blood) disorders. There is some evidence to suggest a link between leukaemia and exposure to dichlorobenzenes. [NIOSHTIC].
Workers who were chronically exposed to 1,4-DCB vapor experienced irritation of the nose and eyes and case reports of people who inhaled or ingested 1,4-DCB suggest that the liver, nervous system, and haematopoietic system are systemic targets in humans. The available limited information on these systemic effects in humans is consistent with findings in animals exposed to 1,4-DCB.
In individuals exposed chronically to 1,4-DCB, liver effects including jaundice, cirrhosis, and possible death may occur. Chronic exposure may also produce weakness, headache, rhinitis, twitching of the facial muscles. A woman who consumed 4 to 5 moth ball pellets daily for 2.5 years developed unsteady gait, tremors of the hand and general mental sluggishness which disappeared 4 months after exposure ceased. Eight workers manufacturing 1,4-DCB based moth-proofing agents for 1 to 7 months developed neural disorders including intensified muscle reflexes, mild clonus of the ankle and tremors of the fingers. They reported loss of appetite and haemopoietic changes.
An evaluation of 953 adult participants in the Third National Health and Nutrition Examination Survey of the general U.S. population found that exposure to 1,4-DCB may possibly contribute to decreases in lung function.
Little human data is available about developmental effects. A 21-year-old woman who had eaten 1-2 blocks of 1,4-DCB toilet freshener per week for the first 38 weeks of pregnancy gave birth to an apparently normal child.
Rats treated 1,4-DCB for 2 years, by gastric intubation, showed kidney lesion and in the male, hyperplasia of the thyroid at dose rates of 150 mg/kg.
Mice treated with 300 mg/kg 1,4-DCB, in a similar 2 year gavage study, showed liver changes characterised by hepatocellular degeneration. Thyroid follicular cell hyperplasia was increased in male but not female mice. Nephropathy consisting primarily of degeneration of the cortical tubular epithelium was seen and was more pronounced in males.
Rats, guinea pigs, rabbits, mice and monkeys exposed by inhalation to 1,4-DCB, 7 hours/day, 5 days/week for 140 exposures at 800 ppm exhibited tremor, weight loss and liver changes, including swelling and central necrosis in female rats, and swelling of the kidney epithelium.
A 2 year study with rats and mice treated with oral doses of 1,2-DCB at either 60 or 120 mg 5 days/ week produced a lower survival time of male rats receiving the higher dose. An increase in the incidence of tubular regeneration in the male mouse kidney was the only compound-related, non-neoplastic,
histologic lesion observed and no evidence of carcinogenicity was seen during the study
In rabbits exposed to 300 ppm, but not those exposed to 800 ppm, there was a significant increase in the number of resorptions and the percentages of resorbed implantations per litter; the fact that the effect did not occur in the rabbits exposed to the higher exposure level suggests that it was not treatment-related. A 2-generation oral study in rats found toxicity in the offspring at doses .90 mg/kg/day; effects included reduced birth weight in F1 pups, increased mortality on postnatal day 4 in F1 and F2 pups, clinical manifestations of dry and scaly skin (until approximately postnatal day 7) in F1 and F2 pups, and reduced neurobehavioral performance (draw-up reflex evaluated at weaning) in F2 pups. No exposure-related changes occurred at 30 mg/kg/day. Other evaluations of developmental effects of 1,4-DCB following oral exposure have been negative.
Data on the carcinogenic effects of 1,4-DCB in humans are not available. Four cases involving cancer and exposure to 1,2-DCB have been reported. These involved the development of peripheral leukoblastosis, chronic lymphoid leukaemia and myeloblastic leukaemia.
1,4-DCB has been shown to be carcinogenic in chronic animal studies by both the inhalation and oral routes. Following lifetime oral exposure, hepatic tumors (hepatocellular adenomas and carcinomas and
histiocytic sarcomas) were increased in mice of both sexes, but not in either sex of rats. The oral bioassay also found that the male rats exposed to 1,4-DCB developed renal tubular cell adenocarcinomas, but these are believed to be the result of interaction with a2u-globulin, a renal protein not present in humans. Data on the possible carcinogenic effects of 1,4-DCB following dermal exposure are not available.
An increase in liver tumours (e.g. renal tubular cell adenocarcinomas) was seen in male rats treated with 1,4-DCB, by gastric intubation doses of 150 mg/kg for 2 years. No evidence of carcinogenicity was seen in female rats. An increase incidence of hepatocellular carcinomas and adenomas was seen in
mice treated with gavage doses of 300 mg/kg/day for 2 years. A positive dose-trend for adrenal gland pheochromocytomas in male mice was also reported.
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Prolonged or repeated skin contact may cause degreasing with drying, cracking and dermatitis following.
Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

**Maggo**

| TOXICITY | IRRITATION |
|---|---|
| Not Available | Not Available |

**solvent naphtha petroleum, heavy aromatic**

| TOXICITY | IRRITATION |
|---|---|
| Dermal (rabbit) LD50: >2000 mg/kg[2] | Eye (rabbit): Irritating |

| | | |
|---|---|---|
| | Inhalation(Rat) LC50; >0.003 mg/L4h[1] | Eye: no adverse effect observed (not irritating)[1] |
| | Oral(Rat) LD50; 512 mg/kg[1] | Skin: adverse effect observed (irritating)[1] |

| 1,4-dichlorobenzene | TOXICITY | IRRITATION |
|---|---|---|
| | dermal (rat) LD50: >2000 mg/kg[1] | Eye (human): 80 ppm |
| | Inhalation(Rat) LC50; >5.07 mg/l4h[1] | |
| | Oral(Rat) LD50; >2000 mg/kg[1] | |

| 4-nonylphenol, branched, ethoxylated | TOXICITY | IRRITATION |
|---|---|---|
| | Oral(Mouse) LD50; 150 mg/kg[2] | Eye (rabbit): SEVERE |
| | | Eye: adverse effect observed (irritating)[1] |
| | | Eye: no adverse effect observed (not irritating)[1] |
| | | Skin (rabbit): Mild |
| | | Skin: no adverse effect observed (not irritating)[1] |

| propetamphos | TOXICITY | IRRITATION |
|---|---|---|
| | dermal (rat) LD50: 564 mg/kg[2] | Not Available |
| | Oral(Rat) LD50; 0.067 mg/kg[2] | |

| | |
|---|---|
| ***Legend:*** | *1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances* |

| | |
|---|---|
| **SOLVENT NAPHTHA PETROLEUM, HEAVY AROMATIC** | Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length,with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent that iso- or cyclo-paraffins.<br>The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption,is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.<br>for petroleum:<br>Altered mental state, drowsiness, peripheral motor neuropathy, irreversible brain damage (so-called Petrol Sniffer's Encephalopathy), delirium, seizures, and sudden death have been reported from repeated overexposure to some hydrocarbon solvents, naphthas, and gasoline<br>This product may contain benzene which is known to cause acute myeloid leukaemia and n-hexane which has been shown to metabolize to compounds which are neuropathic.<br>This product contains toluene. There are indications from animal studies that prolonged exposure to high concentrations of toluene may lead to hearing loss.<br>This product contains ethyl benzene and naphthalene from which there is evidence of tumours in rodents<br>**Carcinogenicity:** Inhalation exposure to mice causes liver tumours, which are not considered relevant to humans. Inhalation exposure to rats causes kidney tumours which are not considered relevant to humans.<br>**Mutagenicity:** There is a large database of mutagenicity studies on gasoline and gasoline blending streams, which use a wide variety of endpoints and give predominantly negative results. All in vivo studies in animals and recent studies in exposed humans (e.g. petrol service station attendants) have shown negative results in mutagenicity assays.<br>**Reproductive Toxicity:** Repeated exposure of pregnant rats to high concentrations of toluene (around or exceeding 1000 ppm) can cause developmental effects, such as lower birth weight and developmental neurotoxicity, on the foetus. However, in a two-generation reproductive study in rats exposed to gasoline vapour condensate, no adverse effects on the foetus were observed.<br>**Human Effects:** Prolonged/ repeated contact may cause defatting of the skin which can lead to dermatitis and may make the skin more susceptible to irritation and penetration by other materials.<br>Lifetime exposure of rodents to gasoline produces carcinogenicity although the relevance to humans has been questioned. Gasoline induces kidney cancer in male rats as a consequence of accumulation of the alpha2-microglobulin protein in hyaline droplets in the male (but not female) rat kidney. Such abnormal accumulation represents lysosomal overload and leads to chronic renal tubular cell degeneration, accumulation of cell debris, mineralisation of renal medullary tubules and necrosis. A sustained regenerative proliferation occurs in epithelial cells with subsequent neoplastic transformation with continued exposure. The |

| | |
|---|---|
| | alpha2-microglobulin is produced under the influence of hormonal controls in male rats but not in females and, more importantly, not in humans. |
| **1,4-DICHLOROBENZENE** | During the manufacture and use of chlorobenzenes, clinical symptoms and signs of excessive exposure include: central nervous system effects and irritation of the eyes and upper respiratory tract (MCB); haematological disorders (1,2-DCB); and central nervous system effects, hardening of the skin, and haematological disorders including anaemia (1,4-DCB).<br>All chlorobenzenes appear to be absorbed readily from the gastrointestinal and respiratory tracts in humans and experimental animals, with absorption influenced by the position of the chlorine in different isomers of the same congener. The chlorobenzenes are less readily absorbed through the skin. After rapid distribution to highly perfused organs in experimental animals, absorbed chlorobenzenes accumulate primarily in the fatty tissue, with smaller amounts in the liver and other organs. Chlorobenzenes have been shown to cross the placenta, and have been found in the foetal brain. In general, accumulation is greater for the more highly chlorinated congeners. There is considerable variation, however, in the accumulation of different isomers of the same congener. In both humans and experimental animals, the metabolism of chlorobenzenes proceeds via microsomal oxidation to the corresponding chlorophenol. These chlorophenols can be excreted in the urine as mercapturic acids, or as glucuronic acid or sulfate conjugates. Tetrachlorobenzenes (TeCB) and pentachlorobenzene (PeCB) are metabolized at a slower rate and remain in the tissues for longer periods than the monochloro- to trichloro- congeners. Some of the chlorobenzenes induce a wide range of enzyme systems including those involved in oxidative, reductive, conjugation, and hydrolytic pathways. In general, elimination of the higher chlorinated benzenes is slower than that of the MCB and DCB congeners, and a greater proportion of the tri- to penta-congeners are eliminated unchanged in the faeces.<br>With few exceptions, the chlorobenzenes are only moderately toxic for experimental animals, on an acute basis, and, generally, have oral LD50s greater than 1000 mg/kg body weight; from the limited data available, dermal LD50s are higher. The ingestion of a lethal dose leads to respiratory paralysis, while the inhalation of high doses causes local irritation and depression of the central nervous system. Acute exposures to non-lethal doses of chlorobenzenes induce toxic effects on the liver, kidneys, adrenal glands, mucous membranes, and brain, and effects on metabolizing enzymes. Studies on skin and eye irritation caused by chlorobenzenes have been restricted to 1,2,4-TCB and 1,2-DCB. Both produce severe discomfort, but no permanent damage was noted after direct application to the rabbit eye. 1,2,4-TCB is mildly irritating to the skin and may lead to dermatitis after repeated or prolonged contact. No evidence of sensitization was found. Short-term exposures (5-21 days) of rats and mice to MCB and DCBs at hundreds of mg/kg body weight resulted in liver damage and haematological changes indicative of bone marrow damage. Liver damage was also the major adverse effect noted after the short-term exposure of rats or rabbits to other chlorobenzenes (TCB-PeCB), at doses slightly lower than those for MCB and DCBs. Several of the chlorobenzene isomers studied induced porphyria, the isomers with *para* chlorine atoms being the most active (i.e., 1,4-DCB, 1,2,4-TCB, 1,2,3,,4-TeCB, and PeCB). The general order of toxicity noted for TeCBs and PeCB after short-term exposure was: 1,2,4,5-TeCB >PeCB>1,2,3,4- and 1,2,3,5-TeCB, which correlated well with the levels found in fat and liver.<br>Long-term exposure studies (up to 6 months) on several species of experimental animals indicated a trend for the toxicity of chlorobenzenes to increase with increased ring chlorination. However, there was considerable variation in the long-term toxicities of different isomers of the same congener. For example, 1,4-DCB appeared to be much less toxic than 1,2-DCB. There was a good correlation between toxicity and the degree of accumulation of the compound in the body tissues, female animals being less sensitive than males. Major target organs were the liver and kidney; at higher doses, effects on the haematopoietic system were reported and thyroid toxicity was noted in studies on 1,2,4,5-TeCB and PeCB.<br>There has been no evidence that chlorobenzenes are teratogenic in rats and rabbits. High doses produce embryotoxic and fetotoxic effects. However, such doses were clearly toxic to the mother. Although there is some evidence that TCBs, TeCBs, and PeCB are embryotoxic and fetotoxic at doses that are not toxic for the mother, available data are inconsistent.<br>1,2-DCB is quickly and extensively absorbed through both the gastrointestinal tract and the respiratory tract; studies describing the absorption of 1,2-DCB following dermal exposure are not available. Following absorption, 1,2-DCB is distributed throughout the body, but tends to be found in greatest levels in the fat, kidney, and liver. 1,2-DCB is initially metabolized by cytochrome P-450 enzymes, specifically P4502E1, to an active epoxide followed by hydrolysis to 2,3-dichlorophenol or 3,4-dichlorophenol. The dichlorophenols may be further oxidised or, more often, be conjugated to glutathione, sulfate, or to form the glucuronide; conjugation occurs extensively, with virtually no unconjugated metabolites reported in the available studies. Metabolism is believed to occur mainly in the liver, but may occur at lower levels in other tissues, such as the kidney or lung. Elimination of 1,2-DCB from the body is rapid, with the majority of a single dose being removed within the first 75 hours postexposure; elimination occurs primarily in the urine as metabolites.<br>Absorption of 1,3-DCB can be inferred from studies that have detected 1,3-DCB or metabolites in the breast milk, blood, and fat of humans and in the bile and urine of exposed animals. Distribution is believed to be similar to the other DCB isomers. Similar to the other DCB isomers, 1,3-DCB is initially metabolised by cytochrome P-450 enzymes, followed by extensive conjugation, primarily to glutathione, has been reported. 1,3-DCB is eliminated mainly in the urine, similar to the other DCB isomers.<br>Absorption of 1,4-DCB is rapid and essentially complete following inhalation or oral exposure. Dermal absorption is believed to be very low, based on a very high (>6 g/kg) dermal LD50 for 1,4-DCB in rats, and on a lack of systemic effects in humans who held solid 1,4-DCB in their hands. Similar to the other dichlorobenzene isomers, 1,4-DCB is distributed throughout the body, but tends to be found in greatest levels in fat, liver, and kidney. Metabolism of 1,4-DCB is similar to that of 1,2-DCB, with an initial oxidation to an epoxide, followed by hydrolysis to 2,5-dichlorophenol. Extensive phase II metabolism occurs subsequently, with eliminated metabolites found mainly as the sulfate, glucuronide, or mercapturic acid. 1,4-DCB is eliminated almost exclusively in the urine, primarily as conjugates of 2,5-dichlorophenol.<br><br>**WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.<br>Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen<br>[*National Toxicology Program: U.S. Dep. of Health & Human Services 2002*]<br>Eye effects, respiratory tract changes, diarrhoea, specific developmental effects (cardiovascular system) recorded. |
| **4-NONYLPHENOL, BRANCHED, ETHOXYLATED** | For nonylphenol and its compounds:<br>Alkylphenols like nonylphenol and bisphenol A have estrogenic effects in the body. They are known as xenoestrogens. Estrogenic substances and other endocrine disruptors are compounds that have hormone-like effects in both wildlife and humans. Xenoestrogens usually function by binding to estrogen receptors and acting competitively against natural estrogens. |

Nonylphenol has been found to act as an agonist of GPER (G protein-coupled estrogen receptor),. Nonylphenol has been shown to mimic the natural hormone 17beta-estradiol, and it competes with the endogeous hormone for binding with the estrogen receptors ERalpha and ERbeta.

Effects in pregnant women.

Subcutaneous injections of nonylphenol in late pregnancy causes the expression of certain placental and uterine proteins, namely CaBP-9k, which suggest it can be transferred through the placenta to the fetus. It has also been shown to have a higher potency on the first trimester placenta than the endogenous estrogen 17beta-estradiol. In addition, early prenatal exposure to low doses of nonylphenol cause an increase in apoptosis (programmed cell death) in placental cells. These “low doses” ranged from 10-13-10-9 M, which is lower than what is generally found in the environment.

Nonylphenol has also been shown to affect cytokine signaling molecule secretions in the human placenta. In vitro cell cultures of human placenta during the first trimester were treated with nonylphenol, which increase the secretion of cytokines including interferon gamma, interleukin 4, and interleukin 10, and reduced the secretion of tumor necrosis factor alpha. This unbalanced cytokine profile at this part of pregnancy has been documented to result in implantation failure, pregnancy loss, and other complications.

Effects on metabolism

Nonylphenol has been shown to act as an obesity enhancing chemical or obesogen, though it has paradoxically been shown to have anti-obesity properties. Growing embryos and newborns are particularly vulnerable when exposed to nonylphenol because low-doses can disrupt sensitive processes that occur during these important developmental periods. Prenatal and perinatal exposure to nonylphenol has been linked with developmental abnormalities in adipose tissue and therefore in metabolic hormone synthesis and release. Specifically, by acting as an estrogen mimic, nonylphenol has generally been shown to interfere with hypothalamic appetite control. The hypothalamus responds to the hormone leptin, which signals the feeling of fullness after eating, and nonylphenol has been shown to both increase and decrease eating behavior by interfering with leptin signaling in the midbrain. Nonylphenol has been shown mimic the action of leptin on neuropeptide Y and anorectic POMC neurons, which has an anti-obesity effect by decreasing eating behavior. This was seen when estrogen or estrogen mimics were injected into the ventromedial hypothalamus. On the other hand, nonylphenol has been shown to increase food intake and have obesity enhancing properties by lowering the expression of these anorexigenic neurons in the brain. Additionally, nonylphenol affects the expression of ghrelin: an enzyme produced by the stomach that stimulates appetite. Ghrelin expression is positively regulated by estrogen signaling in the stomach, and it is also important in guiding the differentiation of stem cells into adipocytes (fat cells). Thus, acting as an estrogen mimic, prenatal and perinatal exposure to nonylphenol has been shown to increase appetite and encourage the body to store fat later in life. Finally, long-term exposure to nonylphenol has been shown to affect insulin signaling in the liver of adult male rats.

Cancer

Nonylphenol exposure has also been associated with breast cancer. It has been shown to promote the proliferation of breast cancer cells, due to its agonistic activity on ERalpha (estrogen receptor alpha) in estrogen-dependent and estrogen-independent breast cancer cells. Some argue that nonylphenol's suggested estrogenic effect coupled with its widespread human exposure could potentially influence hormone-dependent breast cancer disease

Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.

Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol ) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture .

On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing.

Allergic Contact Dermatitis—Formation, Structural Requirements,and Reactivity of Skin Sensitizers.

Ann-Therese Karlberg et al; Chem. Res. Toxicol.2008,21,53-69

Polyethylene glycols (PEGs) have a wide variety of PEG-derived mixtures due to their readily linkable terminal primary hydroxyl groups in combination with many possible compounds and complexes such as ethers, fatty acids, castor oils, amines, propylene glycols, among other derivatives. PEGs and their derivatives are broadly utilized in cosmetic products as surfactants, emulsifiers, cleansing agents, humectants, and skin conditioners.

PEGs and PEG derivatives were generally regulated as safe for use in cosmetics, with the conditions that impurities and by-products, such as ethylene oxides and 1,4-dioxane, which are known carcinogenic materials, should be removed before they are mixed in cosmetic formulations.

Most PEGs are commonly available commercially as mixtures of different oligomer sizes in broadly- or narrowly-defined molecular weight (MW) ranges. For instance, PEG-10,000 typically designates a mixture of PEG molecules (n = 195 to 265) having an average MW of 10,000. PEG is also known as polyethylene oxide (PEO) or polyoxyethylene (POE), with the three names being chemical synonyms. However, PEGs mainly refer to oligomers and polymers with molecular masses below 20,000 g/mol, while PEOs are polymers with molecular masses above 20,000 g/mol, and POEs are polymers of any molecular mass. Relatively small molecular weight PEGs are produced by the chemical reaction between ethylene oxide and water or ethylene glycol (or other ethylene glycol oligomers), as catalyzed by acidic or basic catalysts. To produce PEO or high-molecular weight PEGs, synthesis is performed by suspension polymerization. It is necessary to hold the growing polymer chain in solution during the course of the poly-condensation process. The reaction is catalyzed by magnesium-, aluminum-, or calcium-organoelement compounds. To prevent coagulation of polymer chains in the solution, chelating additives such as dimethylglyoxime are used

Safety Evaluation of Polyethyene Glycol (PEG) Compounds for Cosmetic Use: Toxicol Res 2015; 31:105-136 The Korean Society of Toxicology

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Human beings have regular contact with alcohol ethoxylates through a variety of industrial and consumer products such as soaps, detergents, and other cleaning products . Exposure to these chemicals can occur through ingestion, inhalation, or contact with the skin or eyes. Studies of acute toxicity show that volumes well above a reasonable intake level would have to occur to produce any toxic response. Moreover, no fatal case of poisoning with alcohol ethoxylates has ever been reported. Multiple studies investigating the acute toxicity of alcohol ethoxylates have shown that the use of these compounds is of low concern in terms of oral and dermal toxicity .
Clinical animal studies indicate these chemicals may produce gastrointestinal irritation such as ulcerations of the stomach, pilo-erection, diarrhea, and lethargy. Similarly, slight to severe irritation of the skin or eye was generated when undiluted alcohol ethoxylates were applied to the skin and eyes of rabbits and rats. The chemical shows no indication of being a genotoxin, carcinogen, or mutagen (HERA 2007). No information was available on levels at which these effects might occur, though toxicity is thought to be substantially lower than that of nonylphenol ethoxylates.
Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.
Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol ) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture .
On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficultto diagnose ACD to these compounds by patch testing.
Alcohol ethoxylates are according to CESIO (2000) classified as Irritant or Harmful depending on the number of EO-units:
EO < 5 gives Irritant (Xi) with R38 (Irritating to skin) and R41 (Risk of serious damage to eyes)
EO > 5-15 gives Harmful (Xn) with R22 (Harmful if swallowed) - R38/41
EO > 15-20 gives Harmful (Xn) with R22-41
>20 EO is not classified (CESIO 2000)
Oxo-AE, C13 EO10 and C13 EO15, are Irritating (Xi) with R36/38 (Irritating to eyes and skin) .
AE are not included in Annex 1 of the list of dangerous substances of the Council Directive 67/548/EEC

In general, alcohol ethoxylates (AE) are readily absorbed through the skin of guinea pigs and rats and through the gastrointestinal mucosa of rats. AE are quickly eliminated from the body through the urine, faeces, and expired air (CO2).Orally dosed AE was absorbed rapidly and extensively in rats, and more than 75% of the dose was absorbed. When applied to the skin of humans, the doses were absorbed slowly and incompletely (50% absorbed in 72 hours). Half of the absorbed surfactant was excreted promptly in the urine and smaller amounts of AE appeared in the faeces and expired air (CO2) ). The metabolism of C12 AE yields PEG, carboxylic acids, and CO2 as metabolites. The LD50 values after oral administration to rats range from about 1-15 g/kg body weight indicating a low to moderate acute toxicity.

The ability of nonionic surfactants to cause a swelling of the stratum corneum of guinea pig skin has been studied. The swelling mechanism of the skin involves a combination of ionic binding of the hydrophilic group as well as hydrophobic interactions of the alkyl chain with the substrate. One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been established that there is a connection between the potential of surfactants to denature protein in vitro and their effect on the skin. Nonionic surfactants do not carry any net charge and, therefore, they can only form hydrophobic bonds with proteins. For this reason, proteins are not deactivated by nonionic surfactants, and proteins with poor solubility are not solubilized by nonionic surfactants. A substantial amount of toxicological data and information in vivo and in vitro demonstrates that there is no evidence for alcohol ethoxylates (AEs) being genotoxic, mutagenic or carcinogenic. No adverse reproductive or developmental effects were observed. The majority of available toxicity studies revealed NOAELs in excess of 100 mg/kg bw/d but the lowest NOAEL for an individual AE was established to be 50 mg/kg bw/day. This value was subsequently considered as a conservative, representative value in the risk assessment of AE. The effects were restricted to changes in organ weights with no histopathological organ changes with the exception of liver hypertrophy (indicative of an adaptive response to metabolism rather than a toxic effect). It is noteworthy that there was practically no difference in the NOAEL in oral studies of 90-day or 2 years of duration in rats. A comparison of the aggregate consumer exposure and the systemic NOAEL (taking into account an oral absorption value of 75%) results in a Margin of Exposure of 5,800. Taking into account the conservatism in the exposure assessment and the assigned systemic NOAEL, this margin of exposure is considered more than adequate to account for the inherent uncertainty and variability of the hazard database and inter and intra-species extrapolations.

AEs are not contact sensitisers. Neat AE are irritating to eyes and skin. The irritation potential of aqueous solutions of AEs depends on concentrations. Local dermal effects due to direct or indirect skin contact in certain use scenarios where the products are diluted are not of concern as AEs are not expected to be irritating to the skin at in-use concentrations. Potential irritation of the respiratory tract is not a concern given the very low levels of airborne AE generated as a consequence of spray cleaner aerosols or laundry powder detergent dust.

In summary, the human health risk assessment has demonstrated that the use of AE in household laundry and cleaning detergents is safe and does not cause concern with regard to consumer use.
For high boiling ethylene glycol ethers (typically triethylene- and tetraethylene glycol ethers):
**Skin absorption:** Available skin absorption data for triethylene glycol ether (TGBE), triethylene glycol methyl ether (TGME), and triethylene glycol ethylene ether (TGEE) suggest that the rate of absorption in skin of these three glycol ethers is 22 to 34 micrograms/cm2/hr, with the methyl ether having the highest permeation constant and the butyl ether having the lowest. The rates of absorption of TGBE, TGEE and TGME are at least 100-fold less than EGME, EGEE, and EGBE, their ethylene glycol monoalkyl ether counterparts, which have absorption rates that range from 214 to 2890 micrograms/ cm2/hr . Therefore, an

increase in either the chain length of the alkyl substituent or the number of ethylene glycol moieties appears to lead to a decreased rate of percutaneous absorption. However, since the ratio of the change in values of the ethylene glycol to the diethylene glycol series is larger than that
of the diethylene glycol to triethylene glycol series , the effect of the length of the chain and number of ethylene glycol moieties on absorption diminishes with an increased number of ethylene glycol moieties. Therefore, although tetraethylene glycol methyl; ether (TetraME) and tetraethylene glycol butyl ether (TetraBE) are expected to be less permeable to skin than TGME and TGBE, the differences in permeation between these molecules may only be slight.
**Metabolism:** The main metabolic pathway for metabolism of ethylene glycol monoalkyl ethers (EGME, EGEE, and EGBE) is oxidation via alcohol and aldehyde dehydrogenases (ALD/ADH) that leads to the formation of an alkoxy acids. Alkoxy acids are the only toxicologically significant metabolites of glycol ethers that have been detected *in vivo.* The principal metabolite of TGME is believed to be 2-[2-(2-methoxyethoxy)ethoxy] acetic acid . Although ethylene glycol, a known kidney toxicant, has been identified as an impurity or a minor metabolite of glycol ethers in animal studies it does not appear to contribute to the toxicity of glycol ethers.
The metabolites of category members are not likely to be metabolized to any large extent to toxic molecules such as ethylene glycol or the mono alkoxy acids because metabolic breakdown of the ether linkages also has to occur
**Acute toxicity:** Category members generally display low acute toxicity by the oral, inhalation and dermal routes of exposure. Signs of toxicity in animals receiving lethal oral doses of TGBE included loss of righting reflex and flaccid muscle tone, coma, and heavy breathing. Animals administered lethal oral doses of TGEE exhibited lethargy, ataxia, blood in the urogenital area and piloerection before death.
**Irritation:** The data indicate that the glycol ethers may cause mild to moderate skin irritation. TGEE and TGBE are highly irritating to the eyes. Other category members show low eye irritation.
**Repeat dose toxicity**: Results of these studies suggest that repeated exposure to moderate to high doses of the glycol ethers in this category is required to produce systemic toxicity
In a 21-day dermal study, TGME, TGEE, and TGBE were administered to rabbits at 1,000 mg/kg/day. Erythema and oedema were observed. In addition, testicular degeneration (scored as trace in severity) was observed in one rabbit given TGEE and one rabbit given TGME. Testicular effects included spermatid giant cells, focal tubular hypospermatogenesis, and increased cytoplasmic vacuolisation . Due to a high incidence of similar spontaneous changes
in normal New Zealand White rabbits , the testicular effects were considered not to be related to treatment . Thus, the NOAELs for TGME, TGEE and TGBE were established at 1000 mg/kg/day. Findings from this report were considered unremarkable.
A 2-week dermal study was conducted in rats administered TGME at doses of 1,000, 2,500, and 4,000 mg/kg/day . In this study, significantly-increased red blood cells at 4,000 mg/kg/day and significantly-increased urea concentrations in the urine at 2,500 mg/kg/day were observed. A few of the rats given 2,500 or 4,000 mg/kg/day had watery caecal contents and/or haemolysed blood in the stomach These gross pathologic observations were not associated with any histologic abnormalities in these tissues or alterations in haematologic and clinical chemistry parameters. A few males and females treated with either 1,000 or 2,500 mg/kg/day had a few small scabs or crusts at the test site. These alterations were slight in degree and did not adversely affect the rats
In a 13-week drinking water study, TGME was administered to rats at doses of 400, 1,200, and 4,000 mg/kg/day. Statistically-significant changes in relative liver weight were observed at 1,200 mg/kg/day and higher. Histopathological effects included hepatocellular cytoplasmic vacuolisation (minimal to mild in most animals) and hypertrophy (minimal to mild) in males at all doses and hepatocellular hypertrophy (minimal to mild) in high dose females. These effects were statistically significant at 4,000 mg/kg/day. Cholangiofibrosis was observed in 7/15 high-dose males; this effect was observed in a small number of bile ducts and was of mild severity. Significant, small decreases in total test session motor activity were observed in the high-dose animals, but no other neurological effects were observed. The changes in motor activity were secondary to systemic toxicity
**Mutagenicity:** Mutagenicity studies have been conducted for several category members. All in vitro and in vivo studies were negative at concentrations up to 5,000 micrograms/plate and 5,000 mg/kg, respectively, indicating that the category members are not genotoxic at the concentrations used in these studies. The uniformly negative outcomes of various mutagenicity studies performed on category members lessen the concern for carcinogenicity.
**Reproductive toxicity:** Although mating studies with either the category members or surrogates have not been performed, several of the repeated dose toxicity tests with the surrogates have included examination of reproductive organs. A lower molecular weight glycol ether, ethylene glycol methyl ether (EGME), has been shown to be a testicular toxicant. In addition, results of repeated dose toxicity tests with TGME clearly show testicular toxicity at an oral dose of 4,000 mg/kg/day four times greater that the limit dose of 1,000 mg/kg/day recommended for repeat dose studies. It should be noted that TGME is 350 times less potent for testicular effects than EGME. TGBE is not associated with testicular toxicity, TetraME is not likely to be metabolised by any large extent to 2-MAA (the toxic metabolite of EGME), and a mixture containing predominantly methylated glycol ethers in the C5-C11 range does not produce testicular toxicity (even when administered intravenously at 1,000 mg/kg/day).
**Developmental toxicity**: The bulk of the evidence shows that effects on the foetus are not noted in treatments with . 1,000 mg/kg/day during gestation. At 1,250 to 1,650 mg/kg/day TGME (in the rat) and 1,500 mg/kg/day (in the rabbit), the developmental effects observed included skeletal variants and decreased body weight gain.
for nonylphenol:
Nonylphenol was studied for oral toxicity in rats in a 28-day repeat dose toxicity test at doses of 0, 4, 15, 60 and 250 mg/kg/day. Changes suggesting renal dysfunction were mainly noted in both sexes given 250 mg/kg. Liver weights were increased in males given 60 mg/kg and in both sexes given 250 mg/kg group. Histopathologically, hypertrophy of the centrilobular hepatocytes was noted in both sexes given 250 mg/kg. Kidney weights were increased in males given 250 mg/kg and macroscopically, disseminated white spots, enlargement and pelvic dilatation were noted in females given 250 mg/kg. Histopathologically, the following lesions were noted in the 250 mg/kg group: basophilic change of the proximal tubules in both sexes, single cell necrosis of the proximal tubules, inflammatory cell infiltration in the interstitium and casts in females, basophilic change and dilatation of the collecting tubules in both sexes, simple hyperplasia of the pelvic mucosa and pelvic dilatation in females. In the urinary bladder, simple hyperplasia was noted in both sexes given 250 mg/kg. In the caecum, macroscopic dilatation was noted in both sexes given 250 mg/kg. Almost all changes except those in the kidney disappeared after a 14-day recovery period. The NOELs

| | |
|---|---|
| | for males and females are considered to be 15 mg/kg/day and 60 mg/kg/day, respectively, under the conditions of the present study.<br>Nonylphenol was not mutagenic to Salmonella typhimurium, TA100, TA1535, TA98, TA1537 and Escherichia coli WP2 uvrA, with or without an exogeneous metabolic activation system.<br>Nonylphenol induced neither structural chromosomal aberrations nor polyploidy in CHL/IU cells, in the absence or presence of an exogenous metabolic activation system.<br><br>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.<br>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.<br>for linear material: Maternal effects, effects on fertility recorded. |
| **PROPETAMPHOS** | For propetamphos:<br>**Acute toxicity:** Effects due to acute exposure to propetamphos include those which occur with exposure to other orghanophosphate pesticides, including neurological and neuromuscular effects due to cholinesterase inhibition. Very high doses may result in unconsciousness, incontinence, and convulsions or fatality.<br>**Chronic toxicity:** Rats fed propetamphos for 13 weeks exhibited no effects at a low dose of 0.2 mg/kg/day. Over a 77-week study the rats exhibited no adverse effects at or below the very low dose of 0.05 mg/kg/day. In a 2-year feeding studies with rats, there were no effects noted at or below a dose of 6 mg/kg in their diets. Dogs fed the compound for 6 months showed no adverse effects at the dose of 0.05 mg/kg/day.<br>**Reproductive effects:** A three-generation rat study showed no significant effects in litters at 1 mg/kg/day. Available data suggest that propetamphos does not cause reproductive toxicity.<br>**Teratogenic effects:** A teratology study in rabbits was negative. Available data indicate that propetamphos is not teratogenic.<br>**Mutagenic effects:** In studies with the fruit fly Drosophila, propetamphos did not cause chromosome damage. However, in mouse tissue, high levels of the compound caused some mild chromosome damage. These data suggest that the compound is nonmutagenic or weakly mutagenic.<br>**Carcinogenic effects:** A two-year carcinogenicity test on rats and a lifetime carcinogenesis study on mice were both negative. The highest dose administered to the rats was 6 mg/kg/day, and the maximum dose administered to the mice was 21 mg/kg/day. This evidence suggests that propetamphos does not cause cancer.<br>**Organ toxicity:** The primary target organ affected by propetamphos is the nervous system.<br>**Fate in humans and animals:** Cultured preparations of house fly, cockroach and mouse liver cells all shown the ability to breakdown the compound<br>propetamphos technical |

| | | | |
|---|---|---|---|
| **Acute Toxicity** | | **Carcinogenicity** | |
| **Skin Irritation/Corrosion** | | **Reproductivity** | |
| **Serious Eye Damage/Irritation** | | **STOT - Single Exposure** | |
| **Respiratory or Skin sensitisation** | | **STOT - Repeated Exposure** | |
| **Mutagenicity** | | **Aspiration Hazard** | |

***Legend:*** *– Data either not available or does not fill the criteria for classification*
*– Data available to make classification*

## SECTION 12 Ecological information

### Toxicity

**Maggo**

| Endpoint | Test Duration (hr) | Species | Value | Source |
|---|---|---|---|---|
| Not Available | Not Available | Not Available | Not Available | Not Available |

**solvent naphtha petroleum, heavy aromatic**

| Endpoint | Test Duration (hr) | Species | Value | Source |
|---|---|---|---|---|
| EC50(ECx) | 48 | Crustacea | 0.95mg/l | 1 |
| LC50 | 96 | Fish | 0.58mg/l | 2 |
| EC50 | 48 | Crustacea | 0.95mg/l | 1 |
| EC50 | 72 | Algae or other aquatic plants | <1mg/l | 1 |
| EC50 | 96 | Algae or other aquatic plants | 1mg/l | 2 |

**1,4-dichlorobenzene**

| Endpoint | Test Duration (hr) | Species | Value | Source |
|---|---|---|---|---|
| EC50 | 48 | Crustacea | 0.7mg/l | 2 |
| | | | | |

| | | | | | |
|---|---|---|---|---|---|
| | LC50 | 96 | Fish | 0.008-0.011mg/L | 4 |
| | BCF | 840 | Fish | 33-72 | 7 |
| | EC50(ECx) | 24 | Algae or other aquatic plants | <0.001mg/L | 4 |
| | EC50 | 72 | Algae or other aquatic plants | 0.228-0.443mg/L | 4 |
| | EC50 | 96 | Algae or other aquatic plants | 0.198-0.515mg/L | 4 |

| 4-nonylphenol, branched, ethoxylated | Endpoint | Test Duration (hr) | Species | Value | Source |
|---|---|---|---|---|---|
| | LC50 | 96 | Fish | >10mg/l | 2 |
| | EC50 | 48 | Crustacea | 14mg/l | 2 |
| | EC50 | 72 | Algae or other aquatic plants | 19.485mg/l | 2 |
| | NOEC(ECx) | 96 | Algae or other aquatic plants | 8mg/l | 2 |
| | EC50 | 96 | Algae or other aquatic plants | 12mg/l | 2 |

| propetamphos | Endpoint | Test Duration (hr) | Species | Value | Source |
|---|---|---|---|---|---|
| | LC50 | 96 | Fish | 0.002-0.003mg/L | 4 |
| | EC50 | 72 | Algae or other aquatic plants | 9.5mg/l | 4 |
| | EC50(ECx) | 48 | Crustacea | <0.001mg/L | 4 |
| | EC50 | 48 | Crustacea | <0.001mg/L | 4 |

| ***Legend:*** | *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data* |
|---|---|

Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
**DO NOT** discharge into sewer or waterways.

### Persistence and degradability

| Ingredient | Persistence: Water/Soil | Persistence: Air |
|---|---|---|
| 1,4-dichlorobenzene | HIGH (Half-life = 360 days) | MEDIUM (Half-life = 83.58 days) |
| propetamphos | HIGH | HIGH |

### Bioaccumulative potential

| Ingredient | Bioaccumulation |
|---|---|
| solvent naphtha petroleum, heavy aromatic | LOW (BCF = 159) |
| 1,4-dichlorobenzene | LOW (BCF = 190) |
| propetamphos | MEDIUM (LogKOW = 3.82) |

### Mobility in soil

| Ingredient | Mobility |
|---|---|
| 1,4-dichlorobenzene | LOW (KOC = 434) |
| propetamphos | LOW (KOC = 122.4) |

## SECTION 13 Disposal considerations

### Waste treatment methods

| Product / Packaging disposal | Containers may still present a chemical hazard/ danger when empty.<br>Return to supplier for reuse/ recycling if possible.<br>Otherwise:<br>If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. |
|---|---|

| | |
|---|---|
| | Where possible retain label warnings and SDS and observe all notices pertaining to the product.<br>**DO NOT** allow wash water from cleaning or process equipment to enter drains.<br>It may be necessary to collect all wash water for treatment before disposal.<br>In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.<br>Where in doubt contact the responsible authority.<br>Recycle wherever possible or consult manufacturer for recycling options.<br>Consult State Land Waste Authority for disposal.<br>Bury or incinerate residue at an approved site.<br>Recycle containers if possible, or dispose of in an authorised landfill. |

## SECTION 14 Transport information

### Labels Required

| | |
|---|---|
| |  |
| **Marine Pollutant** |  |
| **HAZCHEM** | •3Z |

### Land transport (ADG)

| | |
|---|---|
| **UN number** | 3082 |
| **UN proper shipping name** | ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains 1,4-dichlorobenzene) |
| **Transport hazard class(es)** | Class: 9<br>Subrisk: Not Applicable |
| **Packing group** | III |
| **Environmental hazard** | Environmentally hazardous |
| **Special precautions for user** | Special provisions: 274 331 335 375 AU01<br>Limited quantity: 5 L |

Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082
are not subject to this Code when transported by road or rail in;
(a) packagings;
(b) IBCs; or
(c) any other receptacle not exceeding 500 kg(L).
- Australian Special Provisions (SP AU01) - ADG Code 7th Ed.

### Air transport (ICAO-IATA / DGR)

| | |
|---|---|
| **UN number** | 3082 |
| **UN proper shipping name** | Environmentally hazardous substance, liquid, n.o.s. * (contains 1,4-dichlorobenzene) |
| **Transport hazard class(es)** | ICAO/IATA Class: 9<br>ICAO / IATA Subrisk: Not Applicable<br>ERG Code: 9L |
| **Packing group** | III |
| **Environmental hazard** | Environmentally hazardous |
| **Special precautions for user** | Special provisions: A97 A158 A197 A215<br>Cargo Only Packing Instructions: 964<br>Cargo Only Maximum Qty / Pack: 450 L<br>Passenger and Cargo Packing Instructions: 964<br>Passenger and Cargo Maximum Qty / Pack: 450 L<br>Passenger and Cargo Limited Quantity Packing Instructions: Y964 |

| | | |
|---|---|---|
| | Passenger and Cargo Limited Maximum Qty / Pack | 30 kg G |

### Sea transport (IMDG-Code / GGVSee)

| | |
|---|---|
| **UN number** | 3082 |
| **UN proper shipping name** | ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains 1,4-dichlorobenzene) |
| **Transport hazard class(es)** | IMDG Class: 9<br>IMDG Subrisk: Not Applicable |
| **Packing group** | III |
| **Environmental hazard** | Marine Pollutant |
| **Special precautions for user** | EMS Number: F-A , S-F<br>Special provisions: 274 335 969<br>Limited Quantities: 5 L |

### Transport in bulk according to Annex II of MARPOL and the IBC code

Not Applicable

### Transport in bulk in accordance with MARPOL Annex V and the IMSBC Code

| Product name | Group |
|---|---|
| solvent naphtha petroleum, heavy aromatic | Not Available |
| 1,4-dichlorobenzene | Not Available |
| 4-nonylphenol, branched, ethoxylated | Not Available |
| propetamphos | Not Available |

### Transport in bulk in accordance with the ICG Code

| Product name | Ship Type |
|---|---|
| solvent naphtha petroleum, heavy aromatic | Not Available |
| 1,4-dichlorobenzene | Not Available |
| 4-nonylphenol, branched, ethoxylated | Not Available |
| propetamphos | Not Available |

## SECTION 15 Regulatory information

### Safety, health and environmental regulations / legislation specific for the substance or mixture

**solvent naphtha petroleum, heavy aromatic is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

**1,4-dichlorobenzene is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans

**4-nonylphenol, branched, ethoxylated is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List

**propetamphos is found on the following regulatory lists**

| | |
|---|---|
| Australia Chemicals with non-industrial uses removed from the Australian Inventory of Chemical Substances (old Inventory) | Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6 |
| Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals | |

### National Inventory Status

| National Inventory | Status |
|---|---|
| Australia - AIIC / Australia Non-Industrial Use | Yes |
| Canada - DSL | No (propetamphos) |
| Canada - NDSL | No (solvent naphtha petroleum, heavy aromatic; 1,4-dichlorobenzene; 4-nonylphenol, branched, ethoxylated; propetamphos) |
| China - IECSC | No (propetamphos) |
| Europe - EINEC / ELINCS / NLP | Yes |
| Japan - ENCS | No (solvent naphtha petroleum, heavy aromatic; 4-nonylphenol, branched, ethoxylated) |
| Korea - KECI | Yes |
| New Zealand - NZIoC | Yes |
| Philippines - PICCS | No (propetamphos) |
| USA - TSCA | No (propetamphos) |
| Taiwan - TCSI | Yes |
| Mexico - INSQ | No (propetamphos) |
| Vietnam - NCI | Yes |
| Russia - FBEPH | No (propetamphos) |
| ***Legend:*** | *Yes = All CAS declared ingredients are on the inventory*<br>*No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)* |

## SECTION 16 Other information

| | |
|---|---|
| **Revision Date** | 19/12/2020 |
| **Initial Date** | 19/12/2020 |

### Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.

The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

### Definitions and abbreviations

PC - TWA: Permissible Concentration-Time Weighted Average
PC - STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists
STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit。
IDLH: Immediately Dangerous to Life or Health Concentrations
ES: Exposure Standard
OSF: Odour Safety Factor
NOAEL :No Observed Adverse Effect Level
LOAEL: Lowest Observed Adverse Effect Level
TLV: Threshold Limit Value
LOD: Limit Of Detection
OTV: Odour Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index

AIIC: Australian Inventory of Industrial Chemicals
DSL: Domestic Substances List
NDSL: Non-Domestic Substances List
IECSC: Inventory of Existing Chemical Substance in China
EINECS: European INventory of Existing Commercial chemical Substances
ELINCS: European List of Notified Chemical Substances
NLP: No-Longer Polymers
ENCS: Existing and New Chemical Substances Inventory
KECI: Korea Existing Chemicals Inventory
NZIoC: New Zealand Inventory of Chemicals
PICCS: Philippine Inventory of Chemicals and Chemical Substances
TSCA: Toxic Substances Control Act
TCSI: Taiwan Chemical Substance Inventory
INSQ: Inventario Nacional de Sustancias Químicas
NCI: National Chemical Inventory
FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances