# CRC (NZ) 3045 Power Lube w/Teflon (Aerosol)

**CRC Industries (CRC Industries New Zealand)**

Chemwatch Hazard Alert Code: **3**

Chemwatch: **4546-63**
Version No: **8.1.1.1**
Safety Data Sheet according to WHS and ADG requirements

Issue Date: **01/11/2019**
Print Date: **03/04/2020**
L.GHS.AUS.EN

## SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

### Product Identifier

| | |
|---|---|
| **Product name** | CRC (NZ) 3045 Power Lube w/Teflon (Aerosol) |
| **Synonyms** | Not Available |
| **Proper shipping name** | AEROSOLS |
| **Other means of identification** | Not Available |

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

| | |
|---|---|
| **Relevant identified uses** | Multipurpose lubricant.<br>The use of a quantity of material in an unventilated or confined space may result in increased exposure and an irritating atmosphere developing.<br>Before starting consider control of exposure by mechanical ventilation.<br>Application is by spray atomisation from a hand held aerosol pack |

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

| | |
|---|---|
| **Registered company name** | **CRC Industries (CRC Industries New Zealand)** |
| **Address** | 10 Highbrook Drive East Tamaki Auckland New Zealand |
| **Telephone** | +64 9 272 2700 |
| **Fax** | +64 9 274 9696 |
| **Website** | www.crc.co.nz |
| **Email** | customerservices@crc.co.nz |

### Emergency telephone number

| | |
|---|---|
| **Association / Organisation** | **CRC Industries (CRC Industries New Zealand)** |
| **Emergency telephone numbers** | NZ Poisons Centre 0800 POISON (0800 764 766) |
| **Other emergency telephone numbers** | 111 (NZ Emergency Services) |

## SECTION 2 HAZARDS IDENTIFICATION

### Classification of the substance or mixture

| | |
|---|---|
| **Poisons Schedule** | Not Applicable |
| **Classification [1]** | Flammable Aerosols Category 1, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Skin Sensitizer Category 1, Reproductive Toxicity Category 2, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Specific target organ toxicity - repeated exposure Category 2, Aspiration Hazard Category 1, Acute Aquatic Hazard Category 2, Chronic Aquatic Hazard Category 2 |
| ***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)

| | |
|---|---|
| **H222** | Extremely flammable aerosol. |
| **H315** | Causes skin irritation. |
| **H319** | Causes serious eye irritation. |
| **H317** | May cause an allergic skin reaction. |
| **H361** | Suspected of damaging fertility or the unborn child. |
| **H336** | May cause drowsiness or dizziness. |
| **H373** | May cause damage to organs through prolonged or repeated exposure. |
| **H304** | May be fatal if swallowed and enters airways. |
| **H411** | Toxic to aquatic life with long lasting effects. |

**Continued...**

| | |
|---|---|
| **AUH044** | Risk of explosion if heated under confinement. |

**Precautionary statement(s) Prevention**

| | |
|---|---|
| **P201** | Obtain special instructions before use. |
| **P210** | Keep away from heat/sparks/open flames/hot surfaces. - No smoking. |
| **P211** | Do not spray on an open flame or other ignition source. |
| **P251** | Pressurized container: Do not pierce or burn, even after use. |
| **P260** | Do not breathe mist/vapours/spray. |
| **P271** | Use only outdoors or in a well-ventilated area. |
| **P280** | Wear protective gloves/protective clothing/eye protection/face protection. |
| **P281** | Use personal protective equipment as required. |
| **P273** | Avoid release to the environment. |
| **P272** | Contaminated work clothing should not be allowed out of the workplace. |

**Precautionary statement(s) Response**

| | |
|---|---|
| **P301+P310** | IF SWALLOWED: Immediately call a POISON CENTER or doctor/physician. |
| **P308+P313** | IF exposed or concerned: Get medical advice/attention. |
| **P321** | Specific treatment (see advice on this label). |
| **P331** | Do NOT induce vomiting. |
| **P362** | Take off contaminated clothing and wash before reuse. |
| **P302+P352** | IF ON SKIN: Wash with plenty of water and soap. |
| **P305+P351+P338** | IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing. |
| **P312** | Call a POISON CENTER or doctor/physician if you feel unwell. |
| **P333+P313** | If skin irritation or rash occurs: Get medical advice/attention. |
| **P337+P313** | If eye irritation persists: Get medical advice/attention. |
| **P391** | Collect spillage. |
| **P304+P340** | IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing. |

**Precautionary statement(s) Storage**

| | |
|---|---|
| **P405** | Store locked up. |
| **P410+P412** | Protect from sunlight. Do not expose to temperatures exceeding 50 °C/122 °F. |
| **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 |
|---|---|---|
| 64742-54-7. | 23-33 | paraffinic distillate, heavy, hydrotreated (severe) |
| 107-83-5 | 30-40 | 2-methylpentane |
| 67-64-1 | 20-30 | acetone |
| 110-54-3 | 0-10 | n-hexane |
| 34590-94-8 | 0-10 | dipropylene glycol monomethyl ether |
| 19210-06-1 | 0-10 | zinc dithiophosphate |
| 119-36-8 | 0-5 | methyl salicylate |
| 9002-84-0 | NotSpec | polytetrafluoroethylene |
| 68476-85-7. | 10-30 | hydrocarbon propellant |

## SECTION 4 FIRST AID MEASURES

**Description of first aid measures**

| | |
|---|---|
| **Eye Contact** | If aerosols come in contact with the eyes:<br>▸ Immediately hold the eyelids apart and flush the eye continuously for at least 15 minutes with fresh 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>▸ Transport to hospital or doctor without delay.<br>▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. |

**Continued...**

| | |
|---|---|
| **Skin Contact** | If solids or aerosol mists are deposited upon the skin:<br>▸ Flush skin and hair with running water (and soap if available).<br>▸ Remove any adhering solids with industrial skin cleansing cream.<br>▸ **DO NOT** use solvents.<br>▸ Seek medical attention in the event of irritation. |
| **Inhalation** | If aerosols, fumes or combustion products are inhaled:<br>▸ Remove to fresh air.<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>▸ If breathing is shallow or has stopped, ensure clear airway and apply resuscitation, 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** | ▸ Not considered a normal route of entry.<br>▸ 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. |

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

Treat symptomatically.
- Heavy and persistent skin contamination over many years may lead to dysplastic changes. Pre-existing skin disorders may be aggravated by exposure to this product.
- In general, emesis induction is unnecessary with high viscosity, low volatility products, i.e. most oils and greases.
- High pressure accidental injection through the skin should be assessed for possible incision, irrigation and/or debridement.

**NOTE:** Injuries may not seem serious at first, but within a few hours tissue may become swollen, discoloured and extremely painful with extensive subcutaneous necrosis. Product may be forced through considerable distances along tissue planes.
For acute or short term repeated exposures to acetone:
- Symptoms of acetone exposure approximate ethanol intoxication.
- About 20% is expired by the lungs and the rest is metabolised. Alveolar air half-life is about 4 hours following two hour inhalation at levels near the Exposure Standard; in overdose, saturable metabolism and limited clearance, prolong the elimination half-life to 25-30 hours.
- There are no known antidotes and treatment should involve the usual methods of decontamination followed by supportive care.

[Ellenhorn and Barceloux: Medical Toxicology]
Management:
Measurement of serum and urine acetone concentrations may be useful to monitor the severity of ingestion or inhalation.
Inhalation Management:
- Maintain a clear airway, give humidified oxygen and ventilate if necessary.
- If respiratory irritation occurs, assess respiratory function and, if necessary, perform chest X-rays to check for chemical pneumonitis.
- Consider the use of steroids to reduce the inflammatory response.
- Treat pulmonary oedema with PEEP or CPAP ventilation.

Dermal Management:
- Remove any remaining contaminated clothing, place in double sealed, clear bags, label and store in secure area away from patients and staff.
- Irrigate with copious amounts of water.
- An emollient may be required.

Eye Management:
- Irrigate thoroughly with running water or saline for 15 minutes.
- Stain with fluorescein and refer to an ophthalmologist if there is any uptake of the stain.

Oral Management:
- No **GASTRIC LAVAGE OR EMETIC**
- Encourage oral fluids.

Systemic Management:
- Monitor blood glucose and arterial pH.
- Ventilate if respiratory depression occurs.
- If patient unconscious, monitor renal function.
- Symptomatic and supportive care.

The Chemical Incident Management Handbook:
Guy's and St. Thomas' Hospital Trust, 2000
BIOLOGICAL EXPOSURE INDEX
These represent the determinants observed in specimens collected from a healthy worker exposed at the Exposure Standard (ES or TLV):

| Determinant | Sampling Time | Index | Comments |
|---|---|---|---|
| Acetone in urine | End of shift | 50 mg/L | NS |

NS: Non-specific determinant; also observed after exposure to other material
For acute or short term repeated exposures to petroleum distillates or related hydrocarbons:
- Primary threat to life, from pure petroleum distillate ingestion and/or inhalation, is respiratory failure.
- Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO2 50 mm Hg) should be intubated.
- Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial injury has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
- A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
- Epinephrine (adrenalin) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.
- Lavage is indicated in patients who require decontamination; ensure use of cuffed endotracheal tube in adult patients. [Ellenhorn and Barceloux: Medical Toxicology]

for salicylate intoxication:
- Pending gastric lavage, use emetics such as syrup of Ipecac or delay gastric emptying and absorption by swallowing a slurry of activated charcoal. Do not give ipecac after charcoal.
- Gastric lavage with water or perhaps sodium bicarbonate solution (3%-5%). Mild alkali delays salicylate absorption from the stomach and perhaps slightly from the duodenum.
- Saline catharsis with sodium or magnesium sulfate (15-30 gm in water).
- Take an immediate blood sample for an appraisal of the patient's acid-base status. A pH determination on an anaerobic sample of arterial blood is best. An analysis of the plasma salicylate concentration should be made at the same time. Laboratory controls are almost essential for the proper management of severe salicylism.
- In the presence of an established acidosis, alkali therapy is essential, but at least in an adult, alkali should be withheld until its need is demonstrated by chemical analysis. The intensity of treatment depends on the intensity of acidosis. In the presence of vomiting, intravenous sodium bicarbonate is the most satisfactory of all alkali therapy.
- Correct dehydration and hypoglycaemia (if present) by the intravenous administration of glucose in water or in isotonic saline. The administration of glucose may also serve to remedy ketosis which is often seen in poisoned children.

**Continued...**

- Even in patients without hypoglycaemia, infusions of glucose adequate to produce distinct hyperglycaemia are recommended to prevent glucose depletion in the brain. This recommendation is based on impressive experimental data in animals.
- Renal function should be supported by correcting dehydration and incipient shock. Overhydration is not justified. An alkaline urine should be maintained by the administration of alkali if necessary with care to prevent a severe systemic alkalosis. As long as urine remains alkaline (pH above 7.5), administration of an osmotic diuretic such as mannitol or perhaps THAM is useful, but one must be careful to avoid hypokalaemia. Supplements of potassium chloride should be included in parenteral fluids.
- Small doses of barbiturates, diazepam, paraldehyde, or perhaps other sedatives (but probably not morphine) may be required to suppress extreme restlessness and convulsions.
- For hyperpyrexia, use sponge baths.

The presence of petechiae or other signs of haemorrhagic tendency calls for a large Vitamin K dose and perhaps ascorbic acid. Minor transfusions may be necessary since bleeding in salicylism is not always due to a prothrombin effect.

- Haemodialysis and haemoperfusion have proved useful in salicylate poisoning, as have peritoneal dialysis and exchange transfusions, but alkaline diuretic therapy is probably sufficient except in fulminating cases.

[*GOSSELIN, et.al.: Clinical Toxicology of Commercial Products*]

The mechanism of the toxic effect involves metabolic acidosis, respiratory alkalosis, hypoglycaemia, and potassium depletion. Salicylate poisoning is characterised by extreme acid-base disturbances, electrolyte disturbances and decreased levels of consciousness. There are differences between acute and chronic toxicity and a varying clinical picture which is dependent on the age of the patient and their kidney function. The major feature of poisoning is metabolic acidosis due to "uncoupling of oxidative phosphorylation" which produces an increased metabolic rate, increased oxygen consumption, increased formation of carbon dioxide, increased heat production and increased utilisation of glucose. Direct stimulation of the respiratory centre leads to hyperventilation and respiratory alkalosis. This leads to compensatory increased renal excretion of bicarbonate which contributes to the metabolic acidosis which may coexist or develop subsequently. Hypoglycaemia may occur as a result of increased glucose demand, increased rates of tissue glycolysis, and impaired rate of glucose synthesis. **NOTE:** Tissue glucose levels may be lower than plasma levels. Hyperglycaemia may occur due to increased glycogenolysis. Potassium depletion occurs as a result of increased renal excretion as well as intracellular movement of potassium.

Salicylates competitively inhibit vitamin K dependent synthesis of factors II, VII, IX, X and in addition, may produce a mild dose dependent hepatitis. Salicylates are bound to albumin. The extent of protein binding is concentration dependent (and falls with higher blood levels). This, and the effects of acidosis, decreasing ionisation, means that the volume of distribution increases markedly in overdose as does CNS penetration. The extent of protein binding (50-80%) and the rate of metabolism are concentration dependent. Hepatic clearance has zero order kinetics and thus the therapeutic half-life of 2-4.5 hours but the half-life in overdose is 18-36 hours. Renal excretion is the most important route in overdose. Thus when the salicylate concentrations are in the toxic range there is increased tissue distribution and impaired clearance of the drug.

*HyperTox 3.0 http://www.ozemail.com.au/-ouad/SALI0001.HTA*

## SECTION 5 FIREFIGHTING MEASURES

### Extinguishing media

**SMALL FIRE:**
- Water spray, dry chemical or CO2

**LARGE FIRE:**
- Water spray or fog.

### 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>▸ May be violently or explosively reactive.<br>▸ Wear breathing apparatus plus protective gloves.<br>▸ Prevent, by any means available, spillage from entering drains or water course.<br>▸ If safe, switch off electrical equipment until vapour fire hazard removed.<br>▸ Use water delivered as a fine spray to control fire and cool adjacent area.<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.<br>▸ Equipment should be thoroughly decontaminated after use. |
| **Fire/Explosion Hazard** | ▸ Liquid and vapour are flammable.<br>▸ Moderate fire hazard when exposed to heat or flame.<br>▸ Vapour forms an explosive mixture with air.<br>▸ Moderate explosion hazard when exposed to heat or flame.<br>▸ Vapour may travel a considerable distance to source of ignition.<br>▸ Heating may cause expansion or decomposition leading to violent rupture of containers.<br>▸ Aerosol cans may explode on exposure to naked flame.<br>▸ Rupturing containers may rocket and scatter burning materials.<br>▸ Hazards may not be restricted to pressure effects.<br>▸ May emit acrid, poisonous or corrosive fumes.<br>▸ On combustion, may emit toxic fumes of carbon monoxide (CO).<br>Combustion products include:<br>carbon dioxide (CO2)<br>sulfur oxides (SOx)<br>other pyrolysis products typical of burning organic material.<br>**Contains low boiling substance:** Closed containers may rupture due to pressure buildup under fire conditions. |
| **HAZCHEM** | Not Applicable |

## 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

**Continued...**

| | |
|---|---|
| **Minor Spills** | Slippery when spilt.<br>▸ Clean up all spills immediately.<br>▸ Avoid breathing vapours and contact with skin and eyes.<br>▸ Wear protective clothing, impervious gloves and safety glasses.<br>▸ Shut off all possible sources of ignition and increase ventilation.<br>▸ Wipe up.<br>▸ If safe, damaged cans should be placed in a container outdoors, away from all ignition sources, until pressure has dissipated.<br>▸ Undamaged cans should be gathered and stowed safely. |
| **Major Spills** | Slippery when spilt.<br>▸ Clear area of personnel and move upwind.<br>▸ Alert Fire Brigade and tell them location and nature of hazard.<br>▸ May be violently or explosively reactive.<br>▸ Wear breathing apparatus plus protective gloves.<br>▸ Prevent, by any means available, spillage from entering drains or water courses<br>▸ No smoking, naked lights or ignition sources.<br>▸ Increase ventilation.<br>▸ Stop leak if safe to do so.<br>▸ Water spray or fog may be used to disperse / absorb vapour.<br>▸ Absorb or cover spill with sand, earth, inert materials or vermiculite.<br>▸ If safe, damaged cans should be placed in a container outdoors, away from ignition sources, until pressure has dissipated.<br>▸ Undamaged cans should be gathered and stowed safely.<br>▸ Collect residues and seal in labelled drums for disposal. |

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

## SECTION 7 HANDLING AND STORAGE

### Precautions for safe handling

| | |
|---|---|
| **Safe handling** | ▸ 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>▸ **DO NOT** incinerate or puncture aerosol cans.<br>▸ **DO NOT** spray directly on humans, exposed food or food utensils.<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 are maintained.<br>▸ **DO NOT** allow clothing wet with material to stay in contact with skin |
| **Other information** | ▸ Keep dry to avoid corrosion of cans. Corrosion may result in container perforation and internal pressure may eject contents of can<br>▸ Store in original containers in approved flammable liquid storage area.<br>▸ **DO NOT** store in pits, depressions, basements or areas where vapours may be trapped.<br>▸ No smoking, naked lights, heat or ignition sources.<br>▸ Keep containers securely sealed. Contents under pressure.<br>▸ Store away from incompatible materials.<br>▸ Store in a cool, dry, well ventilated area.<br>▸ Avoid storage at temperatures higher than 40 deg C.<br>▸ Store in an upright position.<br>▸ Protect containers against physical damage.<br>▸ Check regularly for spills and leaks.<br>▸ Observe manufacturer's storage and handling recommendations contained within this SDS. |

### Conditions for safe storage, including any incompatibilities

| | |
|---|---|
| **Suitable container** | ▸ Aerosol dispenser.<br>▸ Check that containers are clearly labelled. |
| **Storage incompatibility** | ▸ Avoid reaction with oxidising agents |

## 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 | paraffinic distillate, heavy, hydrotreated (severe) | Oil mist, refined mineral | 5 mg/m3 | Not Available | Not Available | Not Available |
| Australia Exposure Standards | 2-methylpentane | Hexane, other isomers | 500 ppm / 1760 mg/m3 | 3500 mg/m3 / 1000 ppm | Not Available | Not Available |
| Australia Exposure Standards | acetone | Acetone | 500 ppm / 1185 mg/m3 | 2375 mg/m3 / 1000 ppm | Not Available | Not Available |
| Australia Exposure Standards | n-hexane | Hexane (n-Hexane) | 20 ppm / 72 mg/m3 | Not Available | Not Available | Not Available |

**Continued...**

| | | | | | | |
|---|---|---|---|---|---|---|
| Australia Exposure Standards | dipropylene glycol monomethyl ether | (2-Methoxymethylethoxy) propanol | 50 ppm / 308 mg/m3 | Not Available | Not Available | Not Available |
| Australia Exposure Standards | hydrocarbon propellant | LPG (liquified petroleum gas) | 1000 ppm / 1800 mg/m3 | Not Available | Not Available | Not Available |

**EMERGENCY LIMITS**

| Ingredient | Material name | TEEL-1 | TEEL-2 | TEEL-3 |
|---|---|---|---|---|
| paraffinic distillate, heavy, hydrotreated (severe) | Mineral oil, heavy or light; (paraffin oil; Deobase, deodorized; heavy paraffinic; heavy naphthenic); distillates; includes 64741-53-3, 64741-88-4, 8042-47-5, 8012-95-1; 64742-54-7 | 140 mg/m3 | 1,500 mg/m3 | 8,900 mg/m3 |
| 2-methylpentane | Methylpentane, 2-; (Isohexane) | 1,000 ppm | 11000** ppm | 66000*** ppm |
| acetone | Acetone | Not Available | Not Available | Not Available |
| n-hexane | Hexane | 260 ppm | Not Available | Not Available |
| dipropylene glycol monomethyl ether | Dipropylene glycol methyl ether | 150 ppm | 1700* ppm | 9900** ppm |
| methyl salicylate | Methyl salicylate | 2.3 ppm | 25 ppm | 150 ppm |
| polytetrafluoroethylene | Polytetrafluoroethylene; (Teflon) | 12 mg/m3 | 130 mg/m3 | 790 mg/m3 |
| hydrocarbon propellant | Liquified petroleum gas; (L.P.G.) | 65,000 ppm | 2.30E+05 ppm | 4.00E+05 ppm |

| Ingredient | Original IDLH | Revised IDLH |
|---|---|---|
| paraffinic distillate, heavy, hydrotreated (severe) | 2,500 mg/m3 | Not Available |
| 2-methylpentane | Not Available | Not Available |
| acetone | 2,500 ppm | Not Available |
| n-hexane | 1,100 ppm | Not Available |
| dipropylene glycol monomethyl ether | 600 ppm | Not Available |
| zinc dithiophosphate | Not Available | Not Available |
| methyl salicylate | Not Available | Not Available |
| polytetrafluoroethylene | Not Available | Not Available |
| hydrocarbon propellant | 2,000 ppm | Not Available |

**OCCUPATIONAL EXPOSURE BANDING**

| Ingredient | Occupational Exposure Band Rating | Occupational Exposure Band Limit |
|---|---|---|
| zinc dithiophosphate | E | ≤ 0.1 ppm |
| methyl salicylate | 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**

Odour Safety Factor(OSF) OSF=0.001 (polytetrafluoroethylene)

Exposed individuals are **NOT** reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odour Safety Factor (OSF) is determined to fall into either Class C, D or E.

The Odour Safety Factor (OSF) is defined as:

OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm

Classification into classes follows:

| Class | OSF | Description |
|---|---|---|
| A | 550 | Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by working activities |
| B | 26-550 | As "A" for 50-90% of persons being distracted |
| C | 1-26 | As "A" for less than 50% of persons being distracted |
| D | 0.18-1 | 10-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached |
| E | <0.18 | As "D" for less than 10% of persons aware of being tested |

NOTE H: Special requirements exist in relation to classification and labelling of this substance. This note applies to certain coal- and oil -derived substances and to certain entries for groups of substances in Annex VI. European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP
NOTE L: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 3% DMSO extract as measured by IP 346.
European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP
NOTE K: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.1%w/w 1,3-butadiene (EINECS No 203-450-8). - European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

## 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 |

**Continued...**

"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.
Employers may need to use multiple types of controls to prevent employee overexposure.

General exhaust is adequate under normal conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection.
Provide adequate ventilation in warehouse or closed storage areas.
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: | Speed: |
|---|---|
| aerosols, (released at low velocity into zone of active generation) | 0.5-1 m/s |
| direct spray, spray painting in shallow booths, gas discharge (active generation into zone of rapid air motion) | 1-2.5 m/s (200-500 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**

- No special equipment needed when handling small quantities.
- **OTHERWISE:**
- For potentially moderate exposures:
- Wear general protective gloves, eg. light weight rubber gloves.
- For potentially heavy exposures:
- Wear chemical protective gloves, eg. PVC. and safety footwear.

**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.

**Body protection**

See Other protection below

**Other protection**

No special equipment needed when handling small quantities.
**OTHERWISE:**

- Overalls.
- Skin cleansing cream.
- Eyewash unit.
- Do not spray on hot surfaces.

### 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:
CRC (NZ) 3045 Power Lube w/Teflon (Aerosol)

| Material | CPI |
|---|---|
| PE/EVAL/PE | A |
| SARANEX-23 2-PLY | B |
| TEFLON | B |
| BUTYL | C |
| BUTYL/NEOPRENE | C |
| CPE | C |
| HYPALON | C |
| NATURAL RUBBER | C |

### Respiratory protection

Type AX-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 10 x ES | AX-AUS P2 | - | AX-PAPR-AUS / Class 1 P2 |
| up to 50 x ES | - | AX-AUS / Class 1 P2 | - |
| up to 100 x ES | - | AX-2 P2 | AX-PAPR-2 P2 ^ |

^ - 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

**Continued...**

| | |
|---|---|
| NATURAL+NEOPRENE | C |
| NEOPRENE | C |
| NEOPRENE/NATURAL | C |
| NITRILE | C |
| NITRILE+PVC | C |
| PVA | C |
| PVC | C |
| PVDC/PE/PVDC | C |
| SARANEX-23 | C |
| VITON | C |
| VITON/CHLOROBUTYL | C |
| VITON/NEOPRENE | 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.

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)

## SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

### Information on basic physical and chemical properties

| | |
|---|---|
| **Appearance** | Amber highly flammable liquid aerosol with an oil of wintergreen odour; not miscible with water. |

| | | | |
|---|---|---|---|
| **Physical state** | Liquid | **Relative density (Water = 1)** | 0.826 |
| **Odour** | Not Available | **Partition coefficient n-octanol / water** | Not Available |
| **Odour threshold** | Not Available | **Auto-ignition temperature (°C)** | Not Available |
| **pH (as supplied)** | Not Applicable | **Decomposition temperature** | Not Available |
| **Melting point / freezing point (°C)** | Not Available | **Viscosity (cSt)** | Not Available |
| **Initial boiling point and boiling range (°C)** | 56.11 (initial) | **Molecular weight (g/mol)** | Not Applicable |
| **Flash point (°C)** | -6.67 | **Taste** | Not Available |
| **Evaporation rate** | Fast | **Explosive properties** | Not Available |
| **Flammability** | HIGHLY FLAMMABLE. | **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** | Immiscible | **pH as a solution (1%)** | Not Applicable |
| **Vapour density (Air = 1)** | > 1 | **VOC g/L** | Not Available |

## SECTION 10 STABILITY AND REACTIVITY

| | |
|---|---|
| **Reactivity** | See section 7 |
| **Chemical stability** | ▸ Elevated temperatures.<br>▸ Presence of open flame.<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

| | |
|---|---|
| **Inhaled** | Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.<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 |

**Continued...**

| | |
|---|---|
| | 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>Common, generalised symptoms associated with toxic gas inhalation include:<br>▸ central nervous system effects such as depression, headache, confusion, dizziness, progressive stupor, coma and seizures;<br>▸ respiratory system complications may include acute pulmonary oedema, dyspnoea, stridor, tachypnoea, bronchospasm, wheezing and other reactive airway symptoms, and respiratory arrest;<br>▸ cardiovascular effects may include cardiovascular collapse, arrhythmias and cardiac arrest;<br>▸ gastrointestinal effects may also be present and may include mucous membrane irritation, nausea and vomiting (sometimes bloody), and abdominal pain.<br>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>Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination<br><br>Material is highly volatile and may quickly form a concentrated atmosphere in confined or unventilated areas. The vapour may displace and replace air in breathing zone, acting as a simple asphyxiant. This may happen with little warning of overexposure.<br>**WARNING**:Intentional misuse by concentrating/inhaling contents may be lethal.<br>If exposure to highly concentrated solvent atmosphere is prolonged this may lead to narcosis, unconsciousness, even coma and possible death.<br>Exposure to ketone vapours may produce nose, throat and mucous membrane irritation. High concentrations of vapour may produce central nervous system depression characterised by headache, vertigo, loss of coordination, narcosis and cardiorespiratory failure. Some ketones produce neurological disorders (polyneuropathy) characterised by bilateral symmetrical paresthesia and muscle weakness primarily in the legs and arms. |
| **Ingestion** | Not normally a hazard due to physical form of product.<br>Considered an unlikely route of entry in commercial/industrial environments<br>Accidental ingestion of the material may be damaging to the health of the individual.<br>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). |
| **Skin Contact** | Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.<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>Spray mist may produce discomfort<br>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects.<br>Examine the skin prior to the use of the material and ensure that any external damage is suitably protected. |
| **Eye** | Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur. |
| **Chronic** | Principal route of occupational exposure to the gas is by inhalation.<br>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.<br>On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body 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>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>Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects.<br>Workers exposed to 700 ppm acetone for 3 hours/day for 7-15 years showed inflammation of the respiratory tract, stomach and duodenum, attacks of giddiness and loss of strength. Exposure to acetone may enhance liver toxicity of chlorinated solvents. |

| | | |
|---|---|---|
| **CRC (NZ) 3045 Power Lube w/Teflon (Aerosol)** | **TOXICITY** | **IRRITATION** |
| | Not Available | Not Available |
| **paraffinic distillate, heavy, hydrotreated (severe)** | **TOXICITY** | **IRRITATION** |
| | Dermal (rabbit) LD50: >2000 mg/kg[2] | Eye: no adverse effect observed (not irritating)[1] |
| | Inhalation (rat) LC50: >5.3 mg/l4 h[1] | Skin: no adverse effect observed (not irritating)[1] |
| | Oral (rat) LD50: >2000 mg/kg[2] | |
| **2-methylpentane** | **TOXICITY** | **IRRITATION** |
| | Not Available | Not Available |
| **acetone** | **TOXICITY** | **IRRITATION** |
| | Dermal (rabbit) LD50: =20 mg/kg[2] | Eye (human): 500 ppm - irritant |
| | Inhalation (rat) LC50: 100.2 mg/l/8hr[2] | Eye (rabbit): 20mg/24hr -moderate |
| | Oral (rat) LD50: 1800-7300 mg/kg[2] | Eye (rabbit): 3.95 mg - SEVERE |
| | | Eye: adverse effect observed (irritating)[1] |
| | | Skin (rabbit): 500 mg/24hr - mild |
| | | Skin (rabbit):395mg (open) - mild |
| | | Skin: no adverse effect observed (not irritating)[1] |

**Continued...**

| | | |
|---|---|---|
| **n-hexane** | **TOXICITY** | **IRRITATION** |
| | Dermal (rabbit) LD50: =3000 mg/kg[2] | Eye(rabbit): 10 mg - mild |
| | Inhalation (rat) LC50: 47945.232 mg/l/4H[2] | |
| | Oral (rat) LD50: 15840 mg/kg[2] | |
| **dipropylene glycol monomethyl ether** | **TOXICITY** | **IRRITATION** |
| | Dermal (rabbit) LD50: 9500 mg/kg[2] | Eye (human): 8 mg - mild |
| | Oral (rat) LD50: 5130 mg/kg[2] | Eye (rabbit): 500 mg/24hr - mild |
| | | Skin (rabbit): 238 mg - mild |
| | | Skin (rabbit): 500 mg (open)-mild |
| **zinc dithiophosphate** | **TOXICITY** | **IRRITATION** |
| | Dermal (rabbit) LD50: >5010 mg/kg[2] | Eye (rabbit): 100 mg - SEVERE |
| | Oral (rat) LD50: 4170 mg/kg[2] | |
| **methyl salicylate** | **TOXICITY** | **IRRITATION** |
| | dermal (rat) LD50: >=2500 mg/kg[2] | Eye (rabbit): 500 mg/24 h - mild |
| | Oral (rat) LD50: 887 mg/kg[2] | Eye: no adverse effect observed (not irritating)[1] |
| | | Skin (rabbit): 500 mg/24 h - moderate |
| | | Skin: no adverse effect observed (not irritating)[1] |
| **polytetrafluoroethylene** | **TOXICITY** | **IRRITATION** |
| | Oral (rat) LD50: 1250 mg/kg[2] | Not Available |
| **hydrocarbon propellant** | **TOXICITY** | **IRRITATION** |
| | Not Available | Not Available |
| ***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* | |

**PARAFFINIC DISTILLATE, HEAVY, HYDROTREATED (SEVERE)**

The materials included in the Lubricating Base Oils category are related from both process and physical-chemical perspectives;
The potential toxicity of a specific distillate base oil is inversely related to the severity or extent of processing the oil has undergone, since:
· The adverse effects of these materials are associated with undesirable components, and
· The levels of the undesirable components are inversely related to the degree of processing;
· Distillate base oils receiving the same degree or extent of processing will have similar toxicities;
· The potential toxicity of *residual base oils* is independent of the degree of processing the oil receives.
· The reproductive and developmental toxicity of the distillate base oils is inversely related to the degree of processing.
The degree of refining influences the carcinogenic potential of the oils. Whereas mild acid / earth refining processes are inadequate to substantially reduce the carcinogenic potential of lubricant base oils, hydrotreatment and / or solvent extraction methods can yield oils with no carcinogenic potential.
Unrefined and mildly refined distillate base oils contain the highest levels of undesirable components, have the largest variation of hydrocarbon molecules and have shown the highest potential carcinogenic and mutagenic activities. Highly and severely refined distillate base oils are produced from unrefined and mildly refined oils by removing or transforming undesirable components. In comparison to unrefined and mildly refined base oils, the highly and severely refined distillate base oils have a smaller range of hydrocarbon molecules and have demonstrated very low mammalian toxicity. Mutagenicity and carcinogenicity testing of residual oils has been negative, supporting the belief that these materials lack biologically active components or the components are largely non-bioavailable due to their molecular size.
Toxicity testing has consistently shown that lubricating base oils have low acute toxicities. Numerous tests have shown that a lubricating base oil's mutagenic and carcinogenic potential correlates with its 3-7 ring polycyclic aromatic compound (PAC) content, and the level of DMSO extractables (e.g. IP346 assay), both characteristics that are directly related to the degree/conditions of processing
Skin irritating is not significant (CONCAWE) based on 14 tests on 10 CASs from the OLBO class (Other Lubricant Base Oils). Each study lasted for 24 hours, a period of time 6 times longer than the duration recommended by the OECD method).
Eye irritation is not significant according to experimental data (CONCAWE studies) based on 9 "in vivo" tests on 7 CASs from the OLBO class(Other Lubricant Base Oils).
Sensitisation: The substance does not cause the sensitization of the respiratory tract or of the skin. (CONCAWE studies based on 14 tests on 11 CASs from the OLBO class(Other Lubricant Base Oils))
Germ cell mutagenicity: The tests performed within the 'in vivo" studies regarding gene mutation at mice micronuclei indicated negative results (CONCAWE studies. AMES tests had negative results in 7 studies performed on 4 CASs from the OLBO class(Other Lubricant Base Oils)).
Reproduction toxicity: Reproduction / development toxicity monitoring according to OECD 421 or 422 methods. CONCAWE tests gave negative results in oral gavage studies. Pre-birth studies regarding toxicity in the unborn foetus development process showed a maternal LOAEL (Lowest Observed Adverse Effect Level) of 125 mg/kg body/day, based on dermal irritation and a NOAEL (No Observable Adverse Effect Level) of 2000 mg/kg body/day, which shows that the substance
is not toxic for reproduction.
STOT (toxicity on specific target organs) – repeated exposure: Studies with short term repeated doses (28-day test) on rabbit skin indicated the NOAEL value of 1000 mg/kg. NOAEL for inhalation, local effects > 280 mg/m3 and for systemic effects NOAEL > 980 mg/m3.
Sub-chronic toxicity
90-day study Dermal: NOAEL > 2000 mg/kg (CONCAWE studies).
Repeat dose toxicity:
Oral
NOAEL for heavy paraffinic distillate aromatic extract could not be identified and is less than 125 mg/kg/day when administered orally.
Inhalation
The NOAEL for lung changes associated with oil deposition in the lungs was 220 mg/m3. As no systemic toxicity was observed, the overall NOAEL for systemic effects was > 980 mg/m3.
Dermal
In a 90 day subchronic dermal study, the administration of Light paraffinic distillate solvent extract had an adverse effect on survivability, body weights, organ weights (particularly the liver and thymus), and variety of haematology and serum chemistry parameters in exposed animals.

**Continued...**

| | |
|---|---|
| | Histopathological changes which were treatment-related were most prominent in the adrenals, bone marrow, kidneys, liver, lymph nodes, skin, stomach, and thymus. Based on the results of this study, the NOAEL for the test material is less than 30 mg/kg/day.<br>Toxicity to reproduction:<br>Mineral oil (a white mineral oil) caused no reproductive or developmental toxicity with 1 mL/kg/day (i.e., 1000 mg/kg/day) in an OECD 421 guideline study, but did cause mild to moderate skin irritation. Therefore, the reproductive/developmental NOAEL for this study is =1000 mg/kg/day and no LOAEL was determined.<br>Developmental toxicity, teratogenicity:<br>Heavy paraffinic distillate furfural extract produced maternal, reproductive and foetal toxicity. Maternal toxicity was exhibited as vaginal discharge (dose-related), body weight decrease, reduction in thymus weight and increase in liver weight (125 mg/kg/day and higher) and aberrant haematology and serum chemistry (125 and/or 500 mg/kg/day). Evidence of potential reproductive effects was shown by an increased number of dams with resorptions and intrauterine death. Distillate aromatic extract (DAE) was developmentally toxic regardless of exposure duration as indicated by increased resorptions and decreased foetal body weights. Furthermore, when exposures were increased to 1000 mg/kg/day and given only during gestation days 10 through 12, cleft palate and ossification delays were observed. Cleft palate was considered to indicate a potential teratogenic effect of DAE.<br>The following Oil Industry Note (OIN) has been applied: OIN 8 - The classifications as a reproductive toxicant category 2; H361d (Suspected of damaging the unborn child) and specific target organ toxicant category 1; H372 (Causes damage to organs through prolonged or repeated exposure) need not apply if the substance is not classified as carcinogenic<br>Toxicokinetics of lubricant base oils has been examined in rodents. Absorption of other lubricant base oils across the small intestine is related to carbon chain length; hydrocarbons with smaller chain length are more readily absorbed than hydrocarbons with a longer chain length. The majority of an oral dose of mineral hydrocarbon is not absorbed and is excreted unchanged in the faeces. Distribution of mineral hydrocarbons following absorption has been observed in liver, fat, kidney, brain and spleen. Excretion of absorbed mineral hydrocarbons occurs via the faeces and urine. Based on the pharmacokinetic parameters and disposition profiles, the data indicate inherent strain differences in the total systemic exposure (~4 fold greater systemic dose in F344 vs SD rats), rate of metabolism, and hepatic and lymph node retention of C26H52, which may be associated with the different strain sensitivities to the formation of liver granulomas and MLN histiocytosis.<br><br>Highly and Severely Refined Distillate Base Oils<br>**Acute toxicity:** Multiple studies of the acute toxicity of highly & severely refined base oils have been reported. Irrespective of the crude source or the method or extent of processing, the oral LD50s have been observed to be >5 g/kg (bw) and the dermal LD50s have ranged from >2 to >5g/kg (bw). The LC50 for inhalation toxicity ranged from 2.18 mg/l to> 4 mg/l.<br>When tested for skin and eye irritation, the materials have been reported as “non-irritating” to “moderately irritating”<br>Testing in guinea pigs for sensitization has been negative<br>**Repeat dose toxicity: .** Several studies have been conducted with these oils**. T**he weight of evidence from all available data on highly & severely refined base oils support the presumption that a distillate base oil’s toxicity is inversely related to the degree of processing it receives. Adverse effects have been reported with even the most severely refined white oils - these appear to depend on animal species and/ or the peculiarities of the study.<br>▸ The granulomatous lesions induced by the oral administration of white oils are essentially foreign body responses. The lesions occur only in rats, of which the Fischer 344 strain is particularly sensitive,<br>▸ The testicular effects seen in rabbits after dermal administration of a highly to severely refined base oil were unique to a single study and may have been related to stress induced by skin irritation, and<br>▸ The accumulation of foamy macrophages in the alveolar spaces of rats exposed repeatedly via inhalation to high levels of highly to severely refined base oils is not unique to these oils, but would be seen after exposure to many water insoluble materials.<br>**Reproductive and developmental toxicity:** A highly refined base oil was used as the vehicle control in a one-generation reproduction study. The study was conducted according to the OECD Test Guideline 421. There was no effect on fertility and mating indices in either males or females. At necropsy, there were no consistent findings and organ weights and histopathology were considered normal by the study’s authors. A single generation study in which a white mineral oil (a food/ drug grade severely refined base oil) was used as a vehicle control is reported. Two separate groups of pregnant rats were administered 5 ml/kg (bw)/day of the base oil via gavage, on days 6 through 19 of gestation. In one of the two base oil dose groups, three malformed foetuses were found among three litters The study authors considered these malformations to be minor and within the normal ranges for the strain of rat.<br>**Genotoxicity**:<br>*In vitro* (mutagenicity): Several studies have reported the results of testing different base oils for mutagenicity using a modified Ames assay Base oils with no or low concentrations of 3-7 ring PACs had low mutagenicity indices.<br>*In vivo* (chromosomal aberrations): A total of seven base stocks were tested in male and female Sprague-Dawley rats using a bone marrow cytogenetics assay. The test materials were administered via gavage at dose levels ranging from 500 to 5000 mg/kg (bw). Dosing occurred for either a single day or for five consecutive days. None of the base oils produced a significant increase in aberrant cells.<br>**Carcinogenicity:** Highly & severely refined base oils are not carcinogens, when given either orally or dermally. |
| **ACETONE** | for acetone:<br>The acute toxicity of acetone is low. Acetone is not a skin irritant or sensitiser but is a defatting agent to the skin. Acetone is an eye irritant. The subchronic toxicity of acetone has been examined in mice and rats that were administered acetone in the drinking water and again in rats treated by oral gavage. Acetone-induced increases in relative kidney weight changes were observed in male and female rats used in the oral 13-week study. Acetone treatment caused increases in the relative liver weight in male and female rats that were not associated with histopathologic effects and the effects may have been associated with microsomal enzyme induction. Haematologic effects consistent with macrocytic anaemia were also noted in male rats along with hyperpigmentation in the spleen. The most notable findings in the mice were increased liver and decreased spleen weights. Overall, the no-observed-effect-levels in the drinking water study were 1% for male rats (900 mg/kg/d) and male mice (2258 mg/kg/d), 2% for female mice (5945 mg/kg/d), and 5% for female rats (3100 mg/kg/d). For developmental effects, a statistically significant reduction in foetal weight, and a slight, but statistically significant increase in the percent incidence of later resorptions were seen in mice at 15,665 mg/m3 and in rats at 26,100 mg/m3. The no-observable-effect level for developmental toxicity was determined to be 5220 mg/m3 for both rats and mice.<br>Teratogenic effects were not observed in rats and mice tested at 26,110 and 15,665 mg/m3, respectively. Lifetime dermal carcinogenicity studies in mice treated with up to 0.2 mL of acetone did not reveal any increase in organ tumor incidence relative to untreated control animals. The scientific literature contains many different studies that have measured either the neurobehavioural performance or neurophysiological response of humans exposed to acetone. Effect levels ranging from about 600 to greater than 2375 mg/m3 have been reported. Neurobehavioral studies with acetone-exposed employees have recently shown that 8-hr exposures in excess of 2375 mg/m3 were not associated with any dose-related changes in response time, vigilance, or digit span scores. Clinical case studies, controlled human volunteer studies, animal research, and occupational field evaluations all indicate that the NOAEL for this effect is 2375 mg/m3 or greater. |
| **DIPROPYLENE GLYCOL MONOMETHYL ETHER** | for propylene glycol ethers (PGEs):<br>Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM).<br>Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids.<br>Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects). |

**Continued...**

| | |
|---|---|
| | This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product.<br>Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body.<br>As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces.<br>As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m3 for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m3. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m3), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating<br>None are skin sensitisers.<br>In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested).<br>Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m3 (600 ppm) for PnB and 2,010 mg/m3 (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m3 (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m3 (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members.<br>One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m3) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m3). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m3), with decreased body weights occurring at 3000 ppm (11058 mg/m3). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. in a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health.<br>In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity.<br>The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic *in vivo*. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice. |
| **ZINC DITHIOPHOSPHATE** | Somnolence, diarrhoea, haemorrhage recorded. |
| **METHYL SALICYLATE** | The following information refers to contact allergens as a group and may not be specific to this product.<br>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.<br>Adverse reactions to fragrances in perfumes and in fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, photosensitivity, immediate contact reactions (contact urticaria), and pigmented contact dermatitis. Airborne and connubial contact dermatitis occur.<br>Intolerance to perfumes, by inhalation, may occur if the perfume contains a sensitising principal. Symptoms may vary from general illness, coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea, acute respiratory illness, hayfever, and other respiratory diseases (including asthma). Perfumes can induce hyper-reactivity of the respiratory tract without producing an IgE-mediated allergy or demonstrable respiratory obstruction. This was shown by placebo-controlled challenges of nine patients to "perfume mix". The same patients were also subject to perfume provocation, with or without a carbon filter mask, to ascertain whether breathing through a filter with active carbon would prevent symptoms. The patients breathed through the mouth, during the provocations, as a nose clamp was used to prevent nasal inhalation. The patient's earlier symptoms were verified; breathing through the carbon filter had no protective effect. The symptoms were not transmitted via the olfactory nerve but they may have been induced by trigeminal reflex via the respiratory tract or by the eyes.<br>Cases of occupational asthma induced by perfume substances such as isoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to give persistent symptoms even though the exposure is below occupational exposure limits.<br>Inhalation intolerance has also been produced in animals. The emissions of five fragrance products, for one hour, produced various combinations of sensory irritation, pulmonary irritation, decreases in expiratory airflow velocity as well as alterations of the functional observational battery indicative of neurotoxicity in mice. Neurotoxicity was found to be more severe after mice were repeatedly exposed to the fragrance products, being four brands of cologne and one brand of toilet water.<br>Contact allergy to fragrances is relatively common, affecting 1 to 3% of the general population, based on limited testing with eight common fragrance allergens and about 16 % of patients patch tested for suspected allergic contact dermatitis.<br>Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a suffcient degree of fragrance contact allergens. Contact allergy is a life-long, specifically altered reactivity in the immune system. This means that once contact allergy is developed, cells in the immune system will be present which can recognise and react towards the allergen. As a consequence, symptoms, i.e. allergic contact dermatitis, may occur upon re-exposure to the fragrance allergen(s) in question. Allergic contact dermatitis is an inflammatory skin disease characterised by erythema, swelling and vesicles in the acute phase. If exposure continues it may develop into a chronic condition with scaling and painful fissures of the skin. Allergic contact dermatitis to fragrance ingredients is most often caused by cosmetic products and usually involves the face and/or hands. It may affect fitness for work and the quality of life of the individual. Fragrance contact allergy has long been recognised as a frequent and potentially disabling problem. Prevention is possible as it is an environmental disease and if the environment is modified (e.g. by reduced use concentrations of allergens), the disease frequency and severity will decrease Fragrance contact allergy is mostly non-occupational and related to the personal use of cosmetic products. Allergic contact dermatitis can be severe and widespread, with a significant impairment of quality of life and potential consequences for fitness for work. Thus, prevention of contact sensitisation to fragrances, both in terms of primary prevention (avoiding sensitisation) and secondary prevention (avoiding relapses of allergic contact dermatitis in those already sensitised), is an important objective of public health risk management measure.<br>**Hands:** Contact sensitisation may be the primary cause of hand eczema, or may be a complication of irritant or atopic hand eczema. The number of positive patch tests has been reported to correlate with the duration of hand eczema, indicating that long-standing hand eczema |

**Continued...**

may often be complicated by sensitisation .Fragrance allergy may be a relevant problem in patients with hand eczema; perfumes are present in consumer products to which their hands are exposed. A significant relationship between hand eczema and fragrance contact allergy has been found in some studies based on patients investigated for contact allergy. However, hand eczema is a multi-factorial disease and the clinical significance of fragrance contact allergy in (severe) chronic hand eczema may not be clear.
**Axillae Bilateral axillary** (underarm) dermatitis may be caused by perfume in deodorants and, if the reaction is severe, it may spread down the arms and to other areas of the body. In individuals who consulted a dermatologist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy.
**Face** Facial eczema is an important manifestation of fragrance allergy from the use of cosmetic products (16). In men, after-shave products can cause an eczematous eruption of the beard area and the adjacent part of the neck and men using wet shaving as opposed to dry have been shown to have an increased risk of of being fragrance allergic.
**Irritant reactions (including contact urticaria):** Irritant effects of some individual fragrance ingredients, e.g. citral are known. Irritant contact dermatitis from perfumes is believed to be common, but there are no existing investigations to substantiate this, Many more people complain about intolerance or rashes to perfumes/perfumed products than are shown to be allergic by testing. This may be due to irritant effects or inadequate diagnostic procedures. Fragrances may cause a dose-related contact urticaria of the non-immunological type (irritant contact urticaria). Cinnamal, cinnamic alcohol, and Myroxylon pereirae are well recognised causes of contact urticaria, but others, including menthol, vanillin and benzaldehyde have also been reported . The reactions to Myroxylon pereirae may be due to cinnamates. A relationship to delayed contact hypersensitivity was suggested , but no significant difference was found between a fragrance-allergic group and a control group in the frequency of immediate reactions to fragrance ingredients in keeping with a nonimmunological basis for the reactions seen.
**Pigmentary anomalies:** The term “pigmented cosmetic dermatitis” was introduced in 1973 for what had previously been known as melanosis faciei feminae when the mechanism (type IV allergy) and causative allergens were clarified.. It refers to increased pigmentation, usually on the face/neck, often following sub-clinical contact dermatitis. Many cosmetic ingredients were patch tested at non-irritant concentrations and statistical evaluation showed that a number of fragrance ingredients were associated: jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol, geranium oil.
**Photo-reactions** Musk ambrette produced a considerable number of allergic photocontact reactions (in which UV-light is required) in the 1970s and was later banned from use in the EU. Nowadays, photoallergic contact dermatitis is uncommon . Furocoumarins (psoralens) in some plant-derived fragrance ingredients caused phototoxic reactions with erythema followed by hyperpigmentation resulting in Berloque dermatitis. There are now limits for the amount of furocoumarins in fragrance products. Phototoxic reactions still occur but are rare.
**General/respiratory:** Fragrances are volatile and therefore, in addition to skin exposure, a perfume also exposes the eyes and naso-respiratory tract. It is estimated that 2-4% of the adult population is affected by respiratory or eye symptoms by such an exposure. It is known that exposure to fragrances may exacerbate pre-existing asthma . Asthma-like symptoms can be provoked by sensory mechanisms. In an epidemiological investigation, a significant association was found between respiratory complaints related to fragrances and contact allergy to fragrance ingredients, in addition to hand eczema, which were independent risk factors in a multivariate analysis.
Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A prehapten is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems. A prohapten is a chemical that itself is non- or low-sensitising but that is transformed into a hapten in the skin (bioactivation) usually via enzyme catalysis. It is not always possible to know whether a particular allergen that is not directly reactive acts as a prehapten or as a prohapten, or both, because air oxidation and bioactivation can often give the same product (geraniol is an example). Some chemicals might act by all three pathways.
**Prohaptens**
Compounds that are bioactivated in the skin and thereby form haptens are referred to as prohaptens.
In the case of prohaptens, the possibility to become activated is inherent to the molecule and activation cannot be avoided by extrinsic measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Crossreactivity has been shown for certain alcohols and their corresponding aldehydes, i.e. between geraniol and geranial (citral) and between cinnamyl alcohol and cinnamal.
The human skin expresses enzyme systems that are able to metabolise xenobiotics, modifying their chemical structure to increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases: phase I and phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask hydrophilic functional groups. If the metabolites are sufficiently polar at this point they will be eliminated. However, many phase I products have to undergo subsequent phase II transformations, i.e. conjugation to make them sufficiently water soluble to be eliminated. Although the purpose of xenobiotic metabolism is detoxification, it can also convert relatively harmless compounds into reactive species. Cutaneous enzymes that catalyse phase I transformations include the cytochrome P450 mixed-function oxidase system, alcohol and aldehyde dehydrogenases, monoamine oxidases, flavin-containing monooxygenases and hydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferases and sulfotransferases are examples of phase II enzymes that have been shown to be present in human skin . These enzymes are known to catalyse both activating and deactivating biotransformations, but the influence of the reactions on the allergenic activity of skin sensitisers has not been studied in detail. Skin sensitising prohaptens can be recognised and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or in vivo and in vitro studies of sensitisation potential and chemical reactivity.
**QSAR prediction:** The relationships between molecular structure and reactivity that form the basis for structural alerts are based on well established principles of mechanistic organic chemistry. Examples of structural alerts are aliphatic aldehydes (alerting to the possibility of sensitisation via a Schiff base reaction with protein amino groups), and alpha,beta-unsaturated carbonyl groups, C=C-CO- (alerting to the possibility of sensitisation via Michael addition of protein thiol groups). Prediction of the sensitisation potential of compounds that can act via abiotic or metabolic activation (pre- or prohaptens) is more complex compared to that of compounds that act as direct haptens without any activation. The autoxidation patterns can differ due to differences in the stability of the intermediates formed, e.g. it has been shown that autoxidation of the structural isomers linalool and geraniol results in different major haptens/allergens. Moreover, the complexity of the prediction increases further for those compounds that can act both as pre- and prohaptens. In such cases, the impact on the sensitisation potency depends on the degree of abiotic activation (e.g. autoxidation) in relation to the metabolic activation
For certain benzyl derivatives:
All members of this group (benzyl, benzoate and 2-hydroxybenzoate (salicylate) esters) contain a benzene ring bonded directly to an oxygenated functional group (aldehyde or ester) that is hydrolysed and/or oxidised to a benzoic acid derivative. As a stable animal metabolite, benzoic acid derivatives are efficiently excreted primarily in the urine. These reaction pathways have been reported in both aquatic and terrestrial species. The similarity of their toxicologic properties is a reflection their participation in these common metabolic pathways.
In general, members of this group are rapidly absorbed through the gastrointestinal tract, metabolised primarily in the liver, and excreted in the urine either unchanged or as conjugates of benzoic acid derivatives At high doses, conjugation pathways (*e.g.,* glycine) may be saturated; in which case, free benzoic acid is excreted unchanged. Absorption, distribution and excretion studies have been conducted several members of this group and structural relatives. These substances exhibit remarkably similar patterns of pharmacokinetics and metabolism. The benzyl, benzoate, and 2-hydroxybenzoate (salicylate) esters which comprise this category are hydrolysed to the corresponding alcohols and carboxylic acids. The benzyl alcohol and benzaldehyde derivatives are oxidised to the corresponding benzoic acid derivatives that are subsequently excreted unchanged or as glycine or glucuronic acid conjugates. If methoxy or phenolic functional groups are present on the benzene ring, additional minor metabolic options become available. *O*-demethylation yields the corresponding phenol that is subsequently excreted as the glucuronic acid or sulfate conjugates. At high dose levels, gut microflora may act to produce minor amounts of reduction metabolites.
**Acute toxicity**: Oral LD50 values ranged from 887 to greater than 5,000 mg/kg bw demonstrating the low to moderate toxicity of these compounds.
**Repeat dose toxicity:** Overall, numerous repeat-dose studies using various routes of exposure have been conducted in different animal species with members of this chemical category or their close structural relatives. It is important to note that all the benzyl derivatives in this category are eventually metabolised to a common metabolite, benzoic acid, and are rapidly excreted in the urine as benzoic acid or as its glycine, sulfate, or glucuronic acid conjugate. For this reason, the repeat-dose studies currently available provide adequate support for the safety of the benzyl derivatives. Moreover, the levels at which no adverse effects were reported were sufficiently high to accommodate any potential differences among the members of the category.
**Reproductive toxicity:** Several reproductive toxicity studies have been conducted with representatives of this group and produced no

**Continued...**

evidence of reproductive toxicity As with the repeat-dose studies, the benzyl derivatives generally follow the similar metabolic pathways and the studies conducted provide an adequate database for this endpoint. In addition, the dose levels tested provide margins of safety large enough to accommodate any differences among the group.
**Developmental toxicity:** Representative substances from this group were tested for developmental toxicity with uniform results, and indicated no teratogenic potential in the absence of maternal toxicity. Again, the representative substances undergo similar metabolism to the entire benzyl derivative group and therefore, provide an adequate representation for this endpoint.
**Genetic toxicity:** Overall, *in vitro* and *in vivo* genotoxicity studies have been conducted with substances representing the structural characteristics of the benzyl category. The results of these studies were predominantly negative demonstrating a low order of genotoxic potential.. Limited positive and/or equivocal findings have been reported for 3 aldehydes and benzyl acetate, but, in most cases, other studies of the same endpoint with same test substance show no activity. Most importantly, *in vivo* studies on benzaldehyde derivatives and closely related benzyl esters have all yielded negative results. These negative *in vivo* genotoxicity assays are supported by the lack of tumorigenicity in chronic animal studies with representatives of this group.
Data available for more than 100 *in vitro* genotoxicity assays for 9 members of the category and five metabolic precursors or metabolites of benzyl derivatives indicate a low genotoxic potential for members of this chemical category
Equivocal results have been reported mainly for aromatic aldehydes in the MLA and ABS assays.

A member or analogue of a group of hydroxy and alkoxy-substituted benzyl derivatives generally regarded as safe (GRAS) based in part on their self-limiting properties as flavouring substances in food; their rapid absorption. metabolic detoxification, and excretion in humans and other animals, their low level of flavour use, the wide margin of safety between the conservative estimates of intake and the no-observed-adverse effect levels determined from chronic and subchronic studies and the lack of significant genotoxic and mutagenic potential. This evidence of safety is supported by the fact that the intake of benzyl derivatives as natural components of traditional foods is greater than the intake as intentionally added flavouring substances.
All members of this group are aromatic primary alcohols, aldehydes, carboxylic acids or their corresponding esters or acetals. The structural features common to all members of the group is a primary oxygenated functional group bonded directly to a benzene ring. The ring also contains hydroxy or alkoxy substituents.
The hydroxy- and alkoxy- substituted benzyl derivatives are raidly absorbed by the gastrointestinal tract, metabolised in the liver to yield benzoic acid derivatives and excreted primarily in the urine either unchanged or conjugated.
It is expected than aromatic esters and acetals will be hydrolysed in vivo through the catalytic activity of carboxylesterases, (A-esterases), Acetals hydrolyse uncatalysed in gastric juices and intestinal fluids to yield acetaldehydes. Substituted benzyl esters and benzaldehyde acetals are hydrolysed to the corresponding alcoholic alcohols and carboxylic acid.
In general hydroxy- and alkoxy- derivatives of benzaldehyde and benzyl alcohol are oxidised to the corresponding benzoic aid derivatives and, to a lesser extent reduced to corresponding benzyl alcohol derivatives. Following conjugation these are excreted in the urine. Benzyl alcohol derivatives may also be reduced in gut microflora to toluene derivatives.
Flavor and Extract Manufacturers Association (FEMA)
The Research Institute for Fragrance Materials (RIFM) Expert Panel study of fragrance salicylates concluded.

The salicylates are well absorbed by the oral route, and oral bioavailability is assumed to be 100%. Absorption by the dermal route in humans is more limited with bioavailability in the range of 11.8-30.7%.
The salicylates are expected to undergo extensive hydrolysis, primarily in the liver, to salicylic acid which is conjugated with either glycine or glucuronide and is excreted in the urine as salicyluric acid and acyl and phenolic glucuronides. The hydrolyzed side chains are metabolized by common and well-characterized metabolic pathways leading to the formation of innocuous end products. The expected metabolism of the salicylates does not present toxicological concerns.
The acute dermal toxicity of the salicylates is very low, with LD50 values in rabbits reported to be greater than 5000 mg/kg body weight. The acute oral toxicity of the salicylates is moderate, with toxicity generally decreasing with increasing size of the ester R-group and with LD50's between 1000 and >5000 g/kg. In dermal subchronic toxicity studies, extreme doses of methyl salicylate (5 g/kg body weight/day) possibly were nephrotoxic but the data were minimal. The subchronic oral NOAEL is concluded to be 50 mg/kg body weight/day.
Genetic toxicity data, for methyl salicylate, a few other salicylates and for structurally related alkyl- and alkoxy-benzyl derivatives are negative for genotoxicity.
Given the metabolism of salicylate and the evidence that they are non-genotoxic, it can be concluded that the salicylates are without carcinogenic potential.
The reproductive and developmental toxicity data on methyl salicylate demonstrate that high, maternally toxic doses result in a pattern of embryotoxicity and teratogenesis similar to that characterized for salicylic acid.
At concentrations likely to be encountered by humans through the use of the salicylates as fragrance ingredients, these chemicals are considered to be non-irritating to the skin.
The salicylates (with the exception of benzyl salicylate) in general have no or very limited skin sensitization potential.
The salicylates are non-phototoxic and have no photoirritant or photoallergenic activity
The use of the salicylates in fragrances produces low levels of exposure relative to doses that elicit adverse systemic effects in laboratory animals exposed by the dermal or oral route. Based on NOAEL values of 50 mg/kg body weight/day identified in the subchronic and the chronic toxicity studies , a margin of safety for systemic exposure of humans to the individual salicylates in cosmetic products, may be calculated to range from 125 to 2,500,000 (depending upon the assumption of either 12–30% or 100% bioavailability following dermal application) times the maximum daily exposure.

The acute dermal toxicity of the salicylates is very low. Rabbit dermal LD50 values have been reported to be >5000 mg/kg body weight for 15 of the 16 salicylates tested, findings likely related to the limited degree of dermal absorption, the retention of salicylate in the skin, and the relatively moderate toxicity of salicylic acid itself upon systemic exposure (i.e., oral LD50 value of 891 mg/kg body weight in rats) .
Overall, the acute oral toxicity of the salicylates is moderate, with toxicity generally decreasing with increasing size of the ester R-group. For the longer carbon chain salicylates, acute oral LD50's range from 1320 to >5000 mg/kg body weight. The acute oral toxicity of the unsaturated salicylates is likewise low to moderate with rat oral LD50's in the 3200 to >5000 mg/kg body weight range as are the acute oral toxicities of the aromatic salicylates (1300 to >5000 mg/kg body weight)

The 17 compounds assessed in this report include the core salicylate moiety that upon hydrolysis yield salicylic acid and the alcohol of the corresponding alkyl, alkenyl, benzyl, phenyl, phenethyl, etc. side chain. This is consistent with information on other alkyl- and alkoxy- benzyl derivatives whereby aromatic esters are hydrolyzed in vivo by carboxylesterases, or esterases, especially the A-esterases. Potential differences in the metabolism of the individual salicylates would be related to the manner in which the hydrolyzed side chain undergoes further oxidation/reduction and/or conjugation reactions.
Salicylic acid undergoes metabolism primarily in the liver. At low, non-toxic doses, approximately 80% of salicylic acid is further metabolized in the liver via conjugation with glycine and subsequent formation of salicyluric acid.
For each of the salicylates, following hydrolysis to salicylic acid, the resulting side chains, hydroxylated alkyl, alkenyl, and phenyl moieties, could be expected to be further metabolized. In the case of the alcohols formed following hydrolysis. Further metabolism would result in the formation of the corresponding aldehydes and acids, with eventual degradation to $CO_2$ by the fatty acid pathway and the tricarboxylic acid cycle. The secondary alcohols formed by hydrolysis of isobutyl and isoamyl salicylate, would primarily be conjugated with glucuronic acid and excreted. They could also interconvert to the corresponding ketones.
Salicylates bearing alkenyl side chains, may undergo epoxidation and subsequent hydroxylation at points of unsaturation.
However, since both the alkyl and alkenyl side chains would be hydroxylated at one terminus following hydrolysis of the corresponding salicylate, a significant proportion of these hydrolysis products would be excreted in the urine precluding further metabolism and epoxidation.
In the case of hydrolysis of the salicylates containing aromatic side chains, phenyl salicylate and benzyl salicylate, phenol and benzyl alcohol, respectively, would be formed.

**Continued...**

| | |
|---|---|
| | Salicylates were potent and selective inhibitors for AKR1C1 enzymes , a family of aldo-keto reductases implicated in biosynthesis, intermediary metabolism and detoxification.<br>The material may produce severe 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) thickening of the epidermis.<br>Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.<br>Not irritating to human skin at concentrations of 8% in mineral oil* Not sensitising to human skin at concentrations of 8% in mineral oil* Not sensitising to guinea pig (Magnusson and Kligman method) * Not irritating to rabbits on ocular application * Ames test: negative* * Rhodia MSDS |
| **POLYTETRAFLUOROETHYLENE** | For perfluorinated carbons (PFCs):<br>PFCs are inert fluids composed of a complex combination of organic compounds resulting from the distillation of electrochemically fluorinated (ECF) compounds. This class consists of branched, linear and cyclic perfluorinated hydrocarbons having carbon numbers predominantly in the range of C5-Cl8 and boiling in the range of approximately 25 C-255 C (77 F-491 F). Perfluorinated amine and ether compounds may also be present<br><br>Acute oral and inhalation toxicity tests with perfluoroalkanes show no toxicity at any dose tested, and even extremely high-dose intraperitoneal injection resulted in no lethality. In contrast, perfluoroalkenes (such as octafluorocyclopentene, perfluoroisobutylene, hexafluoropropene) have shown evidence of inhalation toxicity, in some cases, extreme.<br><br>PFCs are among the least toxic of all known organic chemicals. PFCs don't oxidise or hydrolyse. They have no functional reactive groups.<br>PFCs owe their low toxicity to the combination of the following properties:<br>▸ Chemical inertness<br>▸ Low solubility in biological media (blood, cell membranes, etc.)<br>▸ High volatility<br>▸ Resistance to biological activation (reductive and oxidative metabolism)<br>Because PFCs are chemically inert, if inhaled and absorbed they do not react chemically with any biological molecules; they simply partition between blood and various organs and tissues.<br>As PFCs have limited ability to dissolve in biological media, they do not reach appreciable concentrations in the tissues of air-exposed animals.<br>As PFCs are highly volatile chemicals and have high air-blood partition coefficients, any fluorochemical remaining after exposure will be rapidly eliminated in the breath. Consequently, all such PFCs have:<br>▸ Very high rodent LC50s (very low acute toxicity)<br>▸ Very high cardiac sensitisation EC50s (very low toxicity)<br>In fact, most PFCs do not induce narcosis (sleep) or cardiac sensitisation at maximum achievable concentration (saturation). Inhalation exposure at levels up to 50,000 ppm for thirteen weeks produced no effects in rats, nor did oral exposure for thirty days at 2,000 mg/kg/day.<br>All PFCs that have undergone evaluation by the ACGIH or WEEL committees in the US have been granted an exposure guideline of 1000 ppm (8-hr TWA). NASA has evaluated the toxicity information associated with PFCs including those that can be used as heat transfer agents and fire extinguishing agents in spacecraft and has established a Space Maximum Allowable Concentration (SMAC) of 11,000 ppm for up to 180 days (24 hours/day)<br><br>PFCs are neutral molecules and because they are maximally fluorinated, they cannot undergo biological oxidation-reduction reactions to form reactive aldehydes, acid fluorides, radicals or acids that have been associated with several types of toxicity.<br>**Genetic toxicity:** As PFCs are not reactive directly with biological tissue and PFCs cannot form reactive metabolites, these fluorochemicals have tested negative in bacterial mutagenicity assays. Ames testing showed no genotoxicity.<br><br>Hydrofluoroethers and hydrofluoropolyethers are highly fluorinated ethers having properties intermediate between the perfluoroethers and hydrocarbon ethers. They are low in toxicity, nonflammable, with densities of 1.4-1.7 g/cm3, surface tensions of 13-16 dyn/cm and low kinematic viscosity. The hydrofluoropolyethers are used as heat-transfer fluids. The hydrofluoroethers are used as heat-transfer fluids as well as precision cleaning solvents and solvents for specialty applications such as coating deposition.<br><br>Perfluorinated compounds are potent peroxisome proliferators and were found to induce 8-hydroxydeoxyguanosine in the liver of treated rats. The material may produce peroxisome proliferation. Peroxisomes are single, membrane limited, cytoplasmic organelles that are found in the cells of animals, plants, fungi and protozoa. Peroxisome proliferators include certain hypolipidaemic drugs, phthalate ester plasticisers, industrial solvents, herbicides, food flavours, leukotriene D4 antagonists and hormones. Numerous studies in rats and mice have demonstrated the hepatocarcinogenic effects of peroxisome proliferators, and these compounds have been unequivocally established as carcinogens. However it is generally conceded that compounds inducing proliferation in rats and mice have little, if any, effect on human liver except at very high doses or extreme conditions of exposure. |
| **HYDROCARBON PROPELLANT** | for Petroleum Hydrocarbon Gases:<br>In many cases, there is more than one potentially toxic constituent in a refinery gas. In those cases, the constituent that is most toxic for a particular endpoint in an individual refinery stream is used to characterize the endpoint hazard for that stream. The hazard potential for each mammalian endpoint for each of the petroleum hydrocarbon gases is dependent upon each petroleum hydrocarbon gas constituent endpoint toxicity values (LC50, LOAEL, etc.) and the relative concentration of the constituent present in that gas. It should also be noted that for an individual petroleum hydrocarbon gas, the constituent characterizing toxicity may be different for different mammalian endpoints, again, being dependent upon the concentration of the different constituents in each, distinct petroleum hydrocarbon gas.<br>All Hydrocarbon Gases Category members contain primarily hydrocarbons (i.e., alkanes and alkenes) and occasionally asphyxiant gases like hydrogen. The inorganic components of the petroleum hydrocarbon gases are less toxic than the C1 - C4 and C5 - C6 hydrocarbon components to both mammalian and aquatic organisms. Unlike other petroleum product categories (*e.g.* gasoline, diesel fuel, lubricating oils, etc.), the inorganic and hydrocarbon constituents of hydrocarbon gases can be evaluated for hazard individually to then predict the screening level hazard of the Category members<br>**Acute toxicity:** No acute toxicity LC50 values have been derived for the C1 -C4 and C5- C6 hydrocarbon (HC) fractions because no mortality was observed at the highest exposure levels tested (~ 5 mg/l) for these petroleum hydrocarbon gas constituents. The order of acute toxicity of petroleum hydrocarbon gas constituents from most to least toxic is:<br>C5-C6 HCs (LC50 > 1063 ppm) > C1-C4 HCs (LC50 > 10,000 ppm) > benzene (LC50 = 13,700 ppm) > butadiene (LC50 = 129,000 ppm) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen).<br>**Repeat dose toxicity:** With the exception of the asphyxiant gases, repeated dose toxicity has been observed in individual selected petroleum hydrocarbon gas constituents. Based upon LOAEL values, the order of order of repeated-dose toxicity of these constituents from most toxic to the least toxic is:<br>Benzene (LOAEL .>=10 ppm) >C1-C4 HCs (LOAEL = 5,000 ppm; assumed to be 100% 2-butene) > C5-C6 HCs (LOAEL = 6,625 ppm) > butadiene (LOAEL = 8,000 ppm) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen).<br>**Genotoxicity:**<br>***In vitro:*** The majority of the Petroleum Hydrocarbon Gases Category components are negative for *in vitro* genotoxicity. The exceptions are: benzene and 1,3-butadiene, which are genotoxic in bacterial and mammalian *in vitro* test systems.<br>***In vivo:*** The majority of the Petroleum Hydrocarbon Gases Category components are negative for *in vivo* genotoxicity**.** The exceptions are benzene and 1,3-butadiene, which are genotoxic in *in vivo* test systems<br>**Developmental toxicity:** Developmental effects were induced by two of the petroleum hydrocarbon gas constituents, benzene and the C5 -C6 hydrocarbon fraction. No developmental toxicity was observed at the highest exposure levels tested for the other petroleum hydrocarbon gas constituents tested for this effect. The asphyxiant gases have not been tested for developmental toxicity. Based on LOAEL and NOAEL values, |

**Continued...**

| | |
|---|---|
| | the order of acute toxicity of these constituents from most to least toxic is:<br>Benzene (LOAEL = 20 ppm) > butadiene (NOAEL .>=1,000 ppm) > C5-C6 HCs (LOAEL = 3,463 ppm) > C1-C4 HCs (NOAEL >=5,000 ppm; assumed to be 100% 2-butene) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen).<br>**Reproductive toxicity:** Reproductive effects were induced by only two petroleum hydrocarbon gas constituents, benzene and isobutane (a constituent of the the C1-C4 hydrocarbon fraction). No reproductive toxicity was observed at the highest exposure levels tested for the other petroleum hydrocarbon gas constituents tested for this effect. The asphyxiant gases have not been tested for reproductive toxicity. Based on LOAEL and NOAEL values, the order of reproductive toxicity of these constituents from most to least toxic is:<br>Benzene (LOAEL = 300 ppm) > butadiene (NOAEL .>=6,000 ppm) > C5-C6 HCs (NOAEL .>=6,521 ppm) > C1-C4 HCs (LOAEL = 9,000 ppm; assumed to be 100% isobutane) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen) |
| **PARAFFINIC DISTILLATE, HEAVY, HYDROTREATED (SEVERE) & POLYTETRAFLUOROETHYLENE** | The substance is classified by IARC as Group 3:<br>**NOT** classifiable as to its carcinogenicity to humans.<br>Evidence of carcinogenicity may be inadequate or limited in animal testing. |
| **2-METHYLPENTANE & HYDROCARBON PROPELLANT** | No significant acute toxicological data identified in literature search. |
| **ACETONE & DIPROPYLENE GLYCOL MONOMETHYL ETHER** | 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. |
| **N-HEXANE & DIPROPYLENE GLYCOL MONOMETHYL ETHER** | The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. |
| **DIPROPYLENE GLYCOL MONOMETHYL ETHER & METHYL SALICYLATE** | Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production. |
| **ZINC DITHIOPHOSPHATE & METHYL SALICYLATE** | The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. |

| | | | |
|---|---|---|---|
| **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

**CRC (NZ) 3045 Power Lube w/Teflon (Aerosol)**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| Not Available | Not Available | Not Available | Not Available | Not Available |

**paraffinic distillate, heavy, hydrotreated (severe)**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| LC50 | 96 | Fish | >100mg/L | 2 |
| EC50 | 48 | Crustacea | >10-mg/L | 2 |
| EC50 | 96 | Algae or other aquatic plants | >1000mg/L | 1 |
| NOEC | 504 | Crustacea | >1mg/L | 1 |

**2-methylpentane**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| LC50 | 96 | Fish | 1.915mg/L | 3 |
| EC50 | 96 | Algae or other aquatic plants | 3.635mg/L | 3 |

**acetone**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| LC50 | 96 | Fish | 5-540mg/L | 2 |
| EC50 | 48 | Crustacea | >100mg/L | 4 |
| EC50 | 96 | Algae or other aquatic plants | 20.565mg/L | 4 |
| NOEC | 240 | Crustacea | 1-866mg/L | 2 |

**n-hexane**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| LC50 | 96 | Fish | 1.674mg/L | 3 |
| EC50 | 48 | Crustacea | 21.85mg/L | 2 |
| EC50 | 96 | Algae or other aquatic plants | 3.089mg/L | 3 |

**Continued...**

| | | | | | |
|---|---|---|---|---|---|
| **dipropylene glycol monomethyl ether** | **ENDPOINT** | **TEST DURATION (HR)** | **SPECIES** | **VALUE** | **SOURCE** |
| | LC50 | 96 | Fish | >1-930mg/L | 2 |
| | EC50 | 48 | Crustacea | 1-930mg/L | 2 |
| | EC50 | 72 | Algae or other aquatic plants | 6-999mg/L | 2 |
| | NOEC | 528 | Crustacea | >=0.5mg/L | 2 |
| **zinc dithiophosphate** | **ENDPOINT** | **TEST DURATION (HR)** | **SPECIES** | **VALUE** | **SOURCE** |
| | Not Available | Not Available | Not Available | Not Available | Not Available |
| **methyl salicylate** | **ENDPOINT** | **TEST DURATION (HR)** | **SPECIES** | **VALUE** | **SOURCE** |
| | LC50 | 96 | Fish | 1-370mg/L | 2 |
| | EC50 | 48 | Crustacea | 28mg/L | 2 |
| | EC50 | 96 | Algae or other aquatic plants | 0.895mg/L | 3 |
| | NOEC | 72 | Algae or other aquatic plants | 0.79mg/L | 2 |
| **polytetrafluoroethylene** | **ENDPOINT** | **TEST DURATION (HR)** | **SPECIES** | **VALUE** | **SOURCE** |
| | LC50 | 96 | Fish | 64.087mg/L | 3 |
| | EC50 | 96 | Algae or other aquatic plants | 248.438mg/L | 3 |
| **hydrocarbon propellant** | **ENDPOINT** | **TEST DURATION (HR)** | **SPECIES** | **VALUE** | **SOURCE** |
| | LC50 | 96 | Fish | 24.11mg/L | 2 |
| | EC50 | 96 | Algae or other aquatic plants | 7.71mg/L | 2 |
| | LC50 | 96 | Fish | 24.11mg/L | 2 |
| | EC50 | 96 | Algae or other aquatic plants | 7.71mg/L | 2 |
| ***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* | | | | |

Drinking Water Standards: hydrocarbon total: 10 ug/l (UK max.).
**DO NOT** discharge into sewer or waterways.
Harmful to aquatic organisms.
May cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

### Persistence and degradability

| Ingredient | Persistence: Water/Soil | Persistence: Air |
|---|---|---|
| 2-methylpentane | LOW | LOW |
| acetone | LOW (Half-life = 14 days) | MEDIUM (Half-life = 116.25 days) |
| n-hexane | LOW | LOW |
| dipropylene glycol monomethyl ether | HIGH | HIGH |
| methyl salicylate | LOW | LOW |
| polytetrafluoroethylene | HIGH | HIGH |

### Bioaccumulative potential

| Ingredient | Bioaccumulation |
|---|---|
| 2-methylpentane | LOW (LogKOW = 3.2145) |
| acetone | LOW (BCF = 0.69) |
| n-hexane | MEDIUM (LogKOW = 3.9) |
| dipropylene glycol monomethyl ether | LOW (BCF = 100) |
| methyl salicylate | LOW (LogKOW = 2.55) |
| polytetrafluoroethylene | LOW (LogKOW = 1.2142) |

### Mobility in soil

| Ingredient | Mobility |
|---|---|
| 2-methylpentane | LOW (KOC = 124.9) |
| acetone | HIGH (KOC = 1.981) |
| n-hexane | LOW (KOC = 149) |
| dipropylene glycol monomethyl ether | LOW (KOC = 10) |
| methyl salicylate | LOW (KOC = 128.2) |

**Continued...**

| | |
|---|---|
| polytetrafluoroethylene | LOW (KOC = 106.8) |

## SECTION 13 DISPOSAL CONSIDERATIONS

### Waste treatment methods

| | |
|---|---|
| **Product / Packaging disposal** | ▸ Consult State Land Waste Management Authority for disposal.<br>▸ Discharge contents of damaged aerosol cans at an approved site.<br>▸ Allow small quantities to evaporate.<br>▸ **DO NOT** incinerate or puncture aerosol cans.<br>▸ Bury residues and emptied aerosol cans at an approved site. |

## SECTION 14 TRANSPORT INFORMATION

### Labels Required

| | |
|---|---|
| |  |
| **Marine Pollutant** |  |
| **HAZCHEM** | Not Applicable |

### Land transport (ADG)

| | |
|---|---|
| **UN number** | 1950 |
| **UN proper shipping name** | AEROSOLS |
| **Transport hazard class(es)** | Class: 2.1<br>Subrisk: Not Applicable |
| **Packing group** | Not Applicable |
| **Environmental hazard** | Environmentally hazardous |
| **Special precautions for user** | Special provisions: 63 190 277 327 344 381<br>Limited quantity: 1000ml |

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

| | |
|---|---|
| **UN number** | 1950 |
| **UN proper shipping name** | Aerosols, flammable |
| **Transport hazard class(es)** | ICAO/IATA Class: 2.1<br>ICAO / IATA Subrisk: Not Applicable<br>ERG Code: 10L |
| **Packing group** | Not Applicable |
| **Environmental hazard** | Environmentally hazardous |
| **Special precautions for user** | Special provisions: A145 A167 A802<br>Cargo Only Packing Instructions: 203<br>Cargo Only Maximum Qty / Pack: 150 kg<br>Passenger and Cargo Packing Instructions: 203<br>Passenger and Cargo Maximum Qty / Pack: 75 kg<br>Passenger and Cargo Limited Quantity Packing Instructions: Y203<br>Passenger and Cargo Limited Maximum Qty / Pack: 30 kg G |

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

| | |
|---|---|
| **UN number** | 1950 |
| **UN proper shipping name** | AEROSOLS |
| **Transport hazard class(es)** | IMDG Class: 2.1<br>IMDG Subrisk: Not Applicable |
| **Packing group** | Not Applicable |

**Continued...**

| | |
|---|---|
| **Environmental hazard** | Marine Pollutant |
| **Special precautions for user** | EMS Number: F-D , S-U<br>Special provisions: 63 190 277 327 344 381 959<br>Limited Quantities: 1000 ml |

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

Not Applicable

## SECTION 15 REGULATORY INFORMATION

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

**PARAFFINIC DISTILLATE, HEAVY, HYDROTREATED (SEVERE) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

**2-METHYLPENTANE IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

**ACETONE IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

**N-HEXANE IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Chemical Footprint Project - Chemicals of High Concern List

**DIPROPYLENE GLYCOL MONOMETHYL ETHER IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Inventory of Chemical Substances (AICS)

**ZINC DITHIOPHOSPHATE IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

**METHYL SALICYLATE IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 3
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

**POLYTETRAFLUOROETHYLENE IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Inventory of Chemical Substances (AICS)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

**HYDROCARBON PROPELLANT IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Chemical Footprint Project - Chemicals of High Concern List

### National Inventory Status

| National Inventory | Status |
|---|---|
| Australia - AICS | No (zinc dithiophosphate) |
| Canada - DSL | Yes |
| Canada - NDSL | No (paraffinic distillate, heavy, hydrotreated (severe); polytetrafluoroethylene; zinc dithiophosphate; acetone; methyl salicylate; hydrocarbon propellant; n-hexane; dipropylene glycol monomethyl ether; 2-methylpentane) |
| China - IECSC | Yes |
| Europe - EINEC / ELINCS / NLP | No (polytetrafluoroethylene; zinc dithiophosphate) |
| Japan - ENCS | No (zinc dithiophosphate) |
| Korea - KECI | Yes |
| New Zealand - NZIoC | Yes |
| Philippines - PICCS | Yes |
| USA - TSCA | Yes |
| Taiwan - TCSI | Yes |
| Mexico - INSQ | No (zinc dithiophosphate) |
| Vietnam - NCI | No (zinc dithiophosphate) |
| Russia - ARIPS | No (zinc dithiophosphate) |

**Continued...**

| | |
|---|---|
| ***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** | 01/11/2019 |
| **Initial Date** | 01/11/2009 |

### SDS Version Summary

| Version | Issue Date | Sections Updated |
|---|---|---|
| 7.1.1.1 | 21/06/2018 | Classification |
| 8.1.1.1 | 01/11/2019 | One-off system update. NOTE: This may or may not change the GHS classification |

### 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
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



**end of SDS**