Otis Bio-CLP

TSA OUTDOORS

Chemwatch Hazard Alert Code: **2**

Chemwatch: **5519-53**
Version No: **2.1**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **17/01/2022**
Print Date: **21/01/2022**
L.GHS.AUS.EN.E

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

Product Identifier

Product name	Otis Bio-CLP
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains antimony diamyldithiocarbamate)
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Use according to manufacturer's directions.

Details of the supplier of the safety data sheet

Registered company name	**TSA OUTDOORS**
Address	Unit 6/ 9 - 13 Winbourne Road Brookvale NSW 2100 Australia
Telephone	+61 2 9938 3244
Fax	+61 2 9939 2972
Website	Tsaoutdoors.com.au
Email	sales@tasco.com.au

Emergency telephone number

Association / Organisation	**Aaron Millard**
Emergency telephone numbers	+61 450 086 593 (Mon-Fri, 9 am-6pm)
Other emergency telephone numbers	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Hazardous to the Aquatic Environment Long-Term 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	**Not Applicable**

Hazard statement(s)

H411	Toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P273	Avoid release to the environment.

Precautionary statement(s) Response

P391	Collect spillage.

Page 1 continued...

Precautionary statement(s) Storage

Not Applicable

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
15890-25-2	2.5-<10	antimony diamyldithiocarbamate
68919-53-9	NotSpec	fatty acids, soya, methyl esters
64742-52-5.	NotSpec	naphthenic distillate, heavy, hydrotreated (severe)
57675-44-2	NotSpec	trimethylolpropane trioleate
Not Available	balance	Ingredients determined not to be hazardous
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available*	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with eyes: ▸ Wash out immediately with water. ▸ If irritation continues, seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▸ Other measures are usually unnecessary.
Ingestion	▸ Immediately give a glass of water. ▸ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

Indication of any immediate medical attention and special treatment needed

Medical literature on human exposure to thiocarbamate derivatives is scarce.
- Animal studies suggest that contact dermatitis and thyroid hyperplasia may occur following exposure.
- These compounds do not have the cholinergic properties of structurally related carbamate insecticides.
- The usual measures for gut and skin contamination are recommended for large doses.
- Some thiocarbamates are structurally similar to disulfiram and may cause the characteristically unpleasant alcohol type reactions lasting for several hours; they may respond to fluids, oxygen and analgesics. Dysrhythmias may occur and patients with serious reactions should have cardiac monitoring.
- Precautions should be taken to prohibit intake of alcohol for 10 days.
- Fats, oils and lipid solvents must not be consumed as they may enhance absorption.

As a general rule thiocarbamates can be absorbed by the skin, mucous membranes and respiratory and gastrointestinal tract. They are eliminated quickly via expired air and urine. Two major pathways exist for the metabolism of thiocarbamates in mammals. One is via sulfoxidation and conjugation with glutathione. The conjugation product is cleaved to the cysteine derivative which is further metabolised to a mercapturic acid compound. The second route involves oxidation of the sulfur to a sulfoxide which is oxidised to a sulfone, or hydroxylation to compounds which enter the carbon metabolic pool.
- Chelation with British Anti-Lewisite (BAL) for serious antimony exposures should be employed.
- Dialyse as needed. The role of exchange diffusion is not clear.
- Be sure to monitor for dysrhythmias.

[Ellenhorn and Barceloux: Medical Toxicology]

SECTION 5 Firefighting measures

Extinguishing media

- There is no restriction on the type of extinguisher which may be used.
- Use extinguishing media suitable for surrounding area.

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. ▸ Wear breathing apparatus plus protective gloves in the event of a fire. ▸ Prevent, by any means available, spillage from entering drains or water courses. ▸ Use fire fighting procedures suitable for surrounding area. ▸ **DO NOT** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire.

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	▸ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	▸ Non combustible. ▸ Not considered to be a significant fire risk. ▸ Expansion or decomposition on heating may lead to violent rupture of containers. ▸ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO). ▸ May emit acrid smoke. Decomposition may produce toxic fumes of: carbon dioxide (CO_2) nitrogen oxides (NOx) sulfur oxides (SOx) metal oxides other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.
HAZCHEM	•3Z

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	▸ Clean up all spills immediately. ▸ Avoid breathing vapours and contact with skin and eyes. ▸ Control personal contact with the substance, by using protective equipment. ▸ Contain and absorb spill with sand, earth, inert material or vermiculite. ▸ Wipe up. ▸ Place in a suitable, labelled container for waste disposal.
Major Spills	Moderate hazard. ▸ Clear area of personnel and move upwind. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear breathing apparatus plus protective gloves. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Stop leak if safe to do so. ▸ Contain spill with sand, earth or vermiculite. ▸ Collect recoverable product into labelled containers for recycling. ▸ Neutralise/decontaminate residue (see Section 13 for specific agent). ▸ Collect solid residues and seal in labelled drums for disposal. ▸ Wash area and prevent runoff into drains. ▸ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▸ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▸ **DO NOT** allow clothing wet with material to stay in contact with skin ▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Avoid contact with moisture. ▸ Avoid contact with incompatible materials. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Keep containers securely sealed when not in use. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	Consider storage under inert gas.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Polyethylene or polypropylene container. ▸ Packing as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▸ Avoid strong acids, bases. ▸ Avoid reaction with oxidising agents ▸ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

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INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	antimony diamyldithiocarbamate	Antimony & compounds (as Sb)	0.5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	naphthenic distillate, heavy, hydrotreated (severe)	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
naphthenic distillate, heavy, hydrotreated (severe)	140 mg/m3	1,500 mg/m3	8,900 mg/m3

Ingredient	Original IDLH	Revised IDLH
antimony diamyldithiocarbamate	50 mg/m3	Not Available
fatty acids, soya, methyl esters	Not Available	Not Available
naphthenic distillate, heavy, hydrotreated (severe)	2,500 mg/m3	Not Available
trimethylolpropane trioleate	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
fatty acids, soya, methyl esters	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

The wide-ranging effects of antimony compounds have made it difficult to recommend an exposure standard which characterises the toxicology of these substances. One criteria, reflecting the irritant properties of antimony pentachloride, produced a calculated value of 5.0 mg/m3 (as antimony), which on the basis of experience was felt to be too high but did act as an "out-rider". The present value reflects this thinking.
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

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.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
Employers may need to use multiple types of controls to prevent employee overexposure.

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

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

Within each range the appropriate value depends on:

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

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

Personal protection

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Eye and face protection	▸ Safety glasses with side shields. ▸ Chemical goggles. ▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	▸ Wear chemical protective gloves, e.g. PVC. ▸ Wear safety footwear or safety gumboots, e.g. Rubber The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Body protection	See Other protection below
Other protection	▸ Overalls. ▸ P.V.C apron. ▸ Barrier cream. ▸ Skin cleansing cream. ▸ Eye wash unit.

Respiratory protection

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	A-AUS P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	-	A-AUS / Class 1 P2	-
up to 100 x ES	-	A-2 P2	A-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 dioxide(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

- Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Liquid with a characteristic odour;

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Physical state	Liquid	**Relative density (Water = 1)**	Not Available
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Applicable
pH (as supplied)	Not Applicable	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	201	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	157	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Applicable	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Applicable	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Applicable	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Immiscible	**pH as a solution (Not Available%)**	Not Applicable
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	▸ Unstable in the presence of incompatible materials. ▸ Product is considered stable. ▸ 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	The material is not thought to produce adverse health effects or irritation of the respiratory tract (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting. Inhalation of antimony and its compounds may produce respiratory and gastrointestinal tract discomfort with sore throat, shallow respiration, coughing, headaches, breathing difficulties, dizziness, weight loss, gingivitis, anaemia, eosinophilia and enzyme inhibition. Inflammation of the upper and lower respiratory tract may occur. Pulmonary congestion and oedema may also occur. Other symptoms include rhinitis, eye irritation, vomiting and diarrhoea, weight loss, dysomnia, hair loss and haematological disorders. Death due to circulatory failure has been described, with pathology showing acute congestion of the heart (myocardial failure), liver and kidneys.
Ingestion	The acute toxicity of thiocarbamates is generally low. When administered in high doses, signs such as anorexia, squinting, hypersalivation, lachrymation, piloerection, laboured breathing, ataxia, hypothermia, incoordination, depression, pareses and muscular fibrillation may occur. While thiocarbamates and their metabolites can be found in certain organs such as liver and kidney, accumulation does not take place because of their rapid metabolism. Lethal doses of some thiocarbamates have produced muscle weakness and ascending paralysis progressing to respiratory paralysis and death in animals. Exposure to small quantities of thiocarbamates and intake of small quantities of ethanol may produce flushing, breathing difficulties, nausea and vomiting and lowered blood pressure. Sensitisation to alcohol may last as long as 6-14 days following exposure. Thiocarbamates are reversible cholinesterase inhibitors (they bind to cholinesterase active sites but are easily replaced by acetylcholine). In contrast the carbamates are effective cholinesterase inhibitors. Acetylcholine inhibition is the principal toxicological effect of concern in carbamates and is often the end-point used to assess risk. Although thiocarbamates are not particularly effective cholinesterase inhibitors they appear to be direct acting neurotoxic agents. Because the principal toxic effects are neurotoxicity (clinical signs, behavioural effects, and/ or changes in motor activity) and neuropathology, these effects are often used for end-point selection in risk assessments rather than cholinesterase inhibition. The material has **NOT** been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern. Ingestion of antimony compounds may produce violent irritation of the nose, throat, stomach and gastrointestinal tract, vomiting, purging with bloody stools, slow shallow respiration, pulmonary oedema, convulsions, loss of consciousness, coma, and death due to circulatory or respiratory failure. Early signs of antimony intoxication include: fatigue, muscle weakness, myopathy, nausea, low back pain, headache, and metallic taste. Later symptoms include blood disorders (haemolytic anemia, myoglobinuria, haematuria) and renal failure. The substance may cause cough, salivation, nausea, and diarrhoea. It may also cause dizziness, laryngitis, anaemia, muscular and neuralgic pains. Other symptoms of overexposure may include tightness of the chest, pain, swelling of the cervical glands, pustular eruptions ("antimony spots"), particularly on the scrotum, difficult urination, sexual disorders, nervousness, loss of sleep, leukopenia, unconsciousness and death. [NIOSHTIC] The minimum lethal dose in man, of antimony, is 130 mg (although 15000 mg has been survived). Antimony is a strong irritant and emetic but the emetic dose (30 mg by mouth) is dangerously high if vomiting fails to occur. Trivalent compounds are generally more lethal than pentavalent derivatives. The trivalent antimony compounds are cardiotoxic. The insoluble salts however are less likely to produce significant toxic effects. Antimony salts are poorly absorbed from the gastrointestinal tract. Most trivalent compounds of antimony are slowly excreted in the urine and bile. Antimony compounds are used medicinally, but therapeutic dose is close to the toxic dose.

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	Antimony poisoning closely parallels arsenic poisoning although vomiting is probably more prominent as absorption from the gastrointestinal tract is generally lower. Like arsenic, antimony has a high affinity for sulfhydryl groups on many enzymes. Antimony is conjugated with glutathione and excreted in urine and feces. Therefore, excessive exposure to antimony has the potential to deplete intracellular glutathione pools. Temporary changes in heart rhythm occurs amongst humans while poisoned animals exhibit severe heart damage. Periodic medical examinations covering lungs, skin, nervous system, heart and gastro-intestinal tract are recommended for occupationally exposed workers. [ILO Encyclopedia]
Skin Contact	Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions. Limited evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. Skin contact with antimony compounds may result in redness and severe irritation with the formation of itchy papules, pustules, skin lesions/ small septic blisters (antimony spots) within a few hours. Rhinitis may also result from dermal contact. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	Although the liquid is not thought to be an irritant (as classified by EC Directives), direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn).
Chronic	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. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Exposure to the material may cause concerns for human fertility, on the basis that similar materials provide some evidence 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. . Repeated or prolonged exposure to antimony and its compounds may produce stomatitis, dry throat, metallic taste, gingivitis, septal and laryngeal perforation, laryngitis, headache, dyspnea, indigestion, nausea, vomiting, diarrhoea, anorexia, anaemia, weight loss, pain and chest tightness, sleeplessness, muscular pain and weakness, dizziness, pharyngitis,tracheitis, bronchitis, pneumonitis, benign pneumoconiosis (with obstructive lung disease and emphysema) and haematological disorders. Degenerative changes of the liver and kidney may occur. Symptoms can be variable, and may including fatigue, myopathy (muscle aches and inflammation), hypotension, angina and immune dysregulation and hypertrophy of splenic follicles. Antimony's deposition in body tissues and its detrimental effects depend upon the oxidation state of the element. The trivalent antimony compounds are cardiotoxic. Trivalent antimony affects liver functions, impairs enzymes, and may interfere with sulfur chemistry. If antimony impairs phosphofructokinase (PFK), then purine metabolism may be disrupted, resulting in elevated blood and/or urine levels of hypoxanthine, uric acid and possibly ammonia. Pentavalent antimony deposits in bone, kidney, and in organs of the endocrine system. Chronic exposure to antimony compounds may result in itchiness, papules and pustules around sweat and sebaceous glands ("antimony spots"), but rarely around the face, and dermatitis. Collapse and sudden death due to anaphylactic-type reactions have occurred. Therapeutic doses given intravenously cause nausea, vomiting, cough and abdominal pain and diarrhoea. Other side-effects include anorexia, chest, muscle and joint pains, pruritus, skin rashes, dizziness and oedema. Renal and hepatic damage occur rarely and haemolytic anaemia has been reported. Continuous treatment with small doses of antimony may give rise to subacute poisoning similar to chronic arsenic poisoning. Smelter workers often show skin rashes on the forearms and thighs resembling chicken pox pustules. Workers exposed to inorganic antimony compounds show a benign pneumoconiosis and obstructive lung disease - these are probably non-specific. Woman appear to more susceptible to systemic effects following exposure. Antimony crosses the placenta, is present in amniotic fluids, and is excreted in breast milk. There are suggestions that exposure may produce an increased incidence of spontaneous late abortions, premature births, and gynecological problems among female antimony smelter workers. An excess of deaths from lung cancer has been reported in smelter workers with more than 7 years exposure to relatively high levels of dust and fume. Animal studies demonstrate that the dust may produce pathological changes in cardiac muscle and may produce an interstitial pneumonitis and endogenous pneumonia. One animal study has also suggested that inhalation of the dust by rats induced a significantly increased incidence of carcinogenic tumours of the lungs and thorax. Increased chromosome defects occur when human lymphocytes are incubated with a soluble antimony salt. The inhalation data suggests that the myocardium is a target of antimony toxicity. It is possible that antimony affects circulating glucose by interfering with enzymes of the glycogenolysis and gluconeogenesis pathways. The mechanism of action of antimony remains unclear. However, some studies suggest that antimony combines with sulfhydryl groups including those in several enzymes important for tissue respiration Some thiocarbamates have an effect on sperm morphology and therefore reproduction. However no teratogenic effects have been observed. Adequate data on the carcinogenicity of thiocarbamates are not available. A case has been reported of a female kitchen worker who developed urticaria on her wrists after wearing a certain brand of gloves containing zinc diethyldithiocarbamate (ZDC). Patch testing revealed sensitivity to ZDC. Symptoms disappeared when other gloves were used (1). DNA base-substitution mutagenicity has been demonstrated using Salmonella(2). (1) Helander& Makela, Contact Dermatitis, 9, pp 327-328, 1983 (2) Hedenstedt etal, Mutation Research, 68, 313-325, 1979 Some dithiocarbamates have been reported to have teratogenic and/or carcinogenic potential and to affect male reproductive capacity. Ethylene(bis)dithiocarbamates are metabolically converted in animals to ethylene thiourea (ETU), a known carcinogen, teratogen and antithyroid agent. The principal systemic effects in animals after subchronic or chronic exposure to ETU include depression of body weight gains, antithyroid effects, changes in the liver, and increased serum cholesterol secondary to the antithyroid effect. The mechanism by which thioureas exert the latter effect involves the inhibition of iodine uptake and activation by the thyroid. At low doses, a physiological and biological compensation mechanism maintains normal levels of circulating thyroid hormone. Prolonged exposure to high doses of thyroid inhibitors causes severe hypertrophy and hyperplasia resulting in reduced levels of circulating thyroid hormone. Rats given 0.25% maneb or zineb (the zinc equivalent) in the diet for 2 years developed thyroid hyperplasia and nodular goiter. Acute non-specific decreases in immunological reactivity have also been recorded in rats. Dogs given daily doses of maneb - manganese ethylene(bis)dithiocarbamate - (200 mg/kg for several months) developed neurological disease (tremors, weakness, gastrointestinal disturbance, posterior incoordination, hypotonus and paresis progressing to flaccid paraplegia). This may result from the release of carbon disulfide from dithiocarbamates in the acid environment of the stomach. Dithiocarbamates produce an isothiocyanate radical (-N=C=S) in fungi and other microorganisms; this inactivates SH groups in amino acids contained within individual cells, thus producing biocidal activity.

Otis Bio-CLP	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

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antimony diamyldithiocarbamate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >16000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; >16400 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]
fatty acids, soya, methyl esters	**TOXICITY**	**IRRITATION**
	Oral (Rabbit) LD50; 2000 mg/kg[2]	Not Available
naphthenic distillate, heavy, hydrotreated (severe)	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50; 2.18 mg/l4h[2]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; >5000 mg/kg[2]	
trimethylolpropane trioleate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[2]	Not Available
	Oral (Rat) LD50; >2000 mg/kg[2]	
Legend:	*1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*	

FATTY ACIDS, SOYA, METHYL ESTERS

SFS Stepan Bio-Diesel SB-D
Toxicity studies for the same project demonstrated no mortalities and few toxic effects on rats and rabbits with up to 5000 mg/kg of biodiesel. Petroleum diesel showed no mortalities at the same concentration either, however toxic effects such as hair loss and urinary discolouring were noted with concentrations of >2000 mg/l in rabbits.
Research contends that exhaust from pure canola oil biodiesel is more lethal for human epithelial cells than that from traditional diesel. Epithelial cells, which are found in the lining of the airways and lungs, provide the body's first line of defence against viruses and particles capable of invading the body. The research found that the ultrafine size of fuel exhaust particles from refined and blended canola oil could lead to respiratory health problems.
Increased use of renewable energy sources raise concerns about health effects of new emissions. The relative cardiopulmonary health effects of exhausts from (1) 100% soy biofuel (B100), (2) 20% soy biofuel + 80% low sulfur petroleum diesel (B20), and (3) 100% petroleum diesel (B0) in rats have been investigated.. Normotensive Wistar–Kyoto (WKY) and spontaneously hypertensive rats were exposed to these three exhausts at 0, 50, 150 and 500 ug/m3, 4 h/day for 2 days or 4 weeks (5 days/week). In addition, WKY rats were exposed for 1 day and responses were analysed 0 h, 1 day or 4 days later for time-course assessment. The presence of pigmented macrophages in the lung alveoli was clearly evident with all three exhausts without apparent pathology. Overall, exposure to all three exhausts produced only modest effects in most endpoints analysed in both strains. BALF gamma-glutamyl transferase (GGT) activity was the most consistent marker and was increased in both strains, primarily with B0 (B0>B100>B20). This increase was associated with only modest increases in BALF neutrophils. Small and very acute increases occurred in aorta mRNA markers of vasoconstriction and thrombosis with B100 but not B0 in WKY rats. Comparative evaluations show modest cardiovascular and pulmonary effects at low concentrations of all exhausts: B0 causing more pulmonary injury and B100 more acute vascular effects. BALF GGT activity could serve as a sensitive biomarker of inhaled pollutants
In another study cardiovascular and inflammatory toxicity of diesel and biodiesel particles wre assessed. Mice were exposed to fuels for 1 h. Heart rate (HR), heart rate variability, and blood pressure were obtained before exposure, as well as 30 and 60 min after exposure. After 24 h, bronchoalveolar lavage, blood, and bone marrow were collected to evaluate inflammation. Biodiesel was more toxic than diesel because it promoted cardiovascular alterations as well as pulmonary and systemic inflammation.
A high consumption of oxidised polyunsaturated fatty acids (PUFAs), which are found in most types of vegetable oil, may increase the likelihood that postmenopausal women will develop breast cancer. Similar effect was observed on prostate cancer, but the study was performed on mice
Another "analysis suggested an inverse association between total polyunsaturated fatty acids and breast cancer risk, but individual polyunsaturated fatty acids behaved differently [from each other]. [...] a 20:2 derivative of linoleic acid [...] was inversely associated with the risk of breast cancer"
PUFAs are prone to spontaneous oxidation/ peroxidation. The feeding of lipid oxidation products and oxidised fats has been reported to cause adverse biological effects on laboratory animals, including growth retardation, teratogenicity, tissue damage and increased liver and kidney weights, as well as cellular damage to the testes and epididymes, increased peroxidation of membrane and tissue lipids and induction of cytochrome P450 activities in the colon and liver.
The propensity for PUFAs to oxidise leads to the generation of free radicals and eventually to rancidity.
Culinary oils, when heated, undergo important chemical reaction involving self-sustaining, free radical-mediated oxidative deterioration of PUFAs. Such by-products may be cytotoxic, mutagenic, reproductive toxins and may produce chronic disease. Samples of repeatedly used oils collected from fast-food retail outlets and restaurants have confirmed the production of aldehydic lipid oxidation products (LOPs) at levels exceeding 10 exp-2 moles per kilogram (mol/kg) during “on-site” frying episodes. Volatile emissions from heated culinary oils used in Chinese-style cooking are mutagenic; exposure to such indoor air pollution may render humans more susceptible to contracting lung or further cancers, together with rhinitis and diminished lung function. The high temperatures used in standard (especially Chinese) frying result in fumes that are rich in volatile LOPs, including acrolein.
The end products of lipid peroxidation are reactive aldehydes, such as malondialdehyde (MDA) and 4-hydroxynonenal (HNE), the second one being known also as "second messenger of free radicals" and major bioactive marker of lipid peroxidation, due to its numerous biological activities resembling activities of reactive oxygen species. end-products of lipid peroxidation may be mutagenic and carcinogenic malondialdehyde reacts with deoxyadenosine and deoxyguanosine in DNA, forming DNA adducts. Malondialdehyde produces mutagenic effects in several bioassays.
Side products of lipid peroxidation can also exert toxic effects, even at sites distant from the primary oxidation site. Such products (typically malondialdehyde and a large group of hydroxyalkenals - alpha-beta-unsaturated aldehydes) may interact with protein thiols (producing intermolecular cross-links) and, as a result produce functional impairment to enzyme systems, receptors and structural proteins. Aldehydes may also inhibit protein biosynthesis and increase osmotic fragility of lysosymes (releasing hydrolytic enzymes) and other subcellular organelles. They may also react with nucleic acids.
The toxicity of lipid hydroperoxides to animals is best illustrated by the lethal phenotype of glutathione peroxidase 4 (GPX4) knockout mice. These animals do not survive past embryonic day 8, indicating that the removal of lipid hydroperoxides is essential for mammalian life.
Peroxidised linoleic acid applied to the shaved skin of guinea pigs, in a patch test experiment, produced necrosis and bleeding. When the abdominal skin of guinea pig was patched for 8 days with a cream containing 25 nmol (in terms of malondialdehyde) of lipid peroxides per gram, a thickening of the epidermis was found
Lipid peroxidation in cellular membranes may produce several morphological alterations resulting, for example, in membrane aggregation, deformation or breakage. This may result in the release of hydrolytic enzymes which in turn may degrade functional macromolecules and cause secondary damage. In addition membrane-bound enzyme systems may be disrupted.
Group A aliphatic monoesters (fatty acid esters) According to a classification scheme described by the American Chemistry Council' Aliphatic Esters Panel, Group A substances are simple monoesters derived from a monofunctional alcohol, such as 2-ethylhexyl alcohol (C8-alcohol) or tridecyl alcohol (C13 alcohol) and fatty acids such as palmitic, stearic, oleic or linoleic acid. Metabolism of the parent esters is expected to yield

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	the corresponding fatty acids and alcohols. The fatty acids are naturally occurring and have a low order of toxicity. Group A substances are rather lipophilic (log Kow 10-15) in character due to the large number of carbon numbers in the ester molecule (e.g., 24,26, 31 carbons) and have relatively high boiling points. Owing to the non-volatile nature of these esters, their vapour pressures are very low and difficult to determine experimentally. Water solubility is also very low. Mammalian Toxicity: Acute Toxicity. Many higher fatty acid esters, such as the stearates, oleates and palmitates, have been cleared for use in the food industry ; thus, their general physiological response and toxicity are very low. Many of the higher fatty acid esters are considered safe for use in cosmetics. Available acute toxicity data indicate that the fatty acid esters in Group A, in general, have a low order of toxicity [e.g., palmitic acid, 2-ethylhexyl ester (LD50 > 5 g/kg) and tall oil fatty acid 2-ethylhexyl ester (LD50 > 64 g/kg)]. Consistent with that, available data spanning the carbon range of C22 to C34 indicate that the alkyl fatty acid esters are not toxic by oral administration [rat LD50 (oral) > 5g/kg, with range from 5 g/kg to 64 kg/kg]. Butyl stearate is tolerated by rats without lethal effects at oral doses of 32 g/kg while octyl oleate has a reported LD50 of >40 ml/kg. In addition, many alkyl fatty acid esters, such as the stearates, oleates and palmitates, have been demonstrated to be not toxic by dermal administration Because of the low volatility of these substances, inhalation exposure at toxicological significant levels is not expected. Repeated Dose Toxicity. 28-Day oral gavage studies in rats with decyl oleate (CAS 3687-46-5) at doses of 100,500 and 1000 mg/kg showed no toxicity as noted with respect to clinical symptoms, biochemistry, hematology, gross lesions or tissue/organ histopathology. The NOAEL was estimated to be 1000 mg/kg. Similarly, octyl or (2-ethylhexyl) stearate showed a NOAEL of 1000 mg/kg in 28-day oral gavage studies in rats. In chronic two-year feeding studies with butyl stearate at concentrations of 1.25% or 6.25% in the diet, exposed rats showed no significant difference from control animals with respect to growth, survival, blood counts or other haematological parameters. Besides the two substances above, various other long-chain fatty acid esters have also been studied for their repeated dose toxicity and the findings support a low order of toxicity. Genotoxicity: Genetic Toxicity (Salmonella). Fatty acid, C 16- 18 saturated and C 18 unsaturated, 2-ethylhexanoate (CAS 85049-37-2); octyl stearate (CAS 109-36-4); and decyl oleate (CAS 3687-46-5)] were shown to be negative in the Ames assay. Since the monoesters are similar in chemical structure and carbon-number range, it is unlikely that esters in Group A will induce point mutation. In addition, the chemistry of the long-chain fatty acids does not suggest the likelihood that these substances or their constituent substructures (i.e., fatty acids, alcohols) are reactive or electrophilic in nature. Genetic Toxicity (Chromosomal Aberrations). The chemistry of the long-chain fatty acid esters does not suggest the likelihood that these substances or their constituent substructures (i.e., fatty acids, alcohols) are reactive or electrophilic in nature. Therefore, the likelihood that the fatty acid monoesters may cause chromosomal mutation is very low. Reproductive toxicity: Assessment of reproductive effects of alkyl fatty acid esters in Group A is based primarily on studies with butyl stearate. Fertility, litter size and survival of offspring were normal in rats fed diets containing 6.25% butyl stearate for 10 weeks. However, growth was reduced in offspring during the pre-weaning and post-weaning periods. No gross lesions were noted among the offspring killed at the end of the 21-day post-weaning periods These results indicate that long-chain fatty acid esters do not cause reproductive toxicity in rats..Given the relative low order of toxicity for long-chain fatty acid esters and their relative non-electrophilic and non-reactive nature, it seems unlikely that the long-chain fatty acid esters would present serious reproductive concerns. Developmental Toxicity/ Teratogenicity. Assessment of developmental effects for the long-chain fatty acid esters in this group was based primarily on data reported for fatty acid, C16-18, 2-ethylhexyl ester (CAS 91031-48-0). In oral gavage studies in rats administered doses of 100,300 and 1000 mg/kg during gestation, the maternal NOAEL was > 1000 mg/kg and the NOAEL for teratogenicity was >1000 mg/kg. Based on these findings and the fact Group A substances, are very chemically similar to the structure of the tested material, read-across assessment is thought to be appropriate.
NAPHTHENIC DISTILLATE, HEAVY, HYDROTREATED (SEVERE)	Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies with similar materials using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies. Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length,with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent that iso- or cyclo-paraffins. The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption,is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver. 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.

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	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. 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. Toxicity to reproduction: 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. Developmental toxicity, teratogenicity: 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. 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 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. Highly and Severely Refined Distillate Base Oils **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. When tested for skin and eye irritation, the materials have been reported as “non-irritating” to “moderately irritating” Testing in guinea pigs for sensitization has been negative **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. ▸ 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, ▸ 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 ▸ 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. **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. **Genotoxicity**: *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. *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. **Carcinogenicity:** Highly & severely refined base oils are not carcinogens, when given either orally or dermally. **NOTE:** Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA. The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.
TRIMETHYLOLPROPANE TRIOLEATE	* REACh SDS For aliphatic fatty acids (and salts) Acute oral (gavage) toxicity: The acute oral LD50 values in rats for both were greater than >2000 mg/kg bw Clinical signs were generally associated with poor condition following administration of high doses (salivation, diarrhoea, staining, piloerection and lethargy).There were no adverse effects on body weight in any study In some studies, excess test substance and/or irritation in the gastrointestinal tract was observed at necropsy. Skin and eye irritation potential, with a few stated exceptions, is chain length dependent and decreases with increasing chain length According to several OECD test regimes the animal skin irritation studies indicate that the C6-10 aliphatic acids are severely irritating or corrosive, while the C12 aliphatic acid is irritating, and the C14-22 aliphatic acids generally are not irritating or mildly irritating. Human skin irritation studies using more realistic exposures (30-minute,1-hour or 24-hours) indicate that the aliphatic acids have sufficient, good or very good skin compatibility. Animal eye irritation studies indicate that among the aliphatic acids, the C8-12 aliphatic acids are irritating to the eye while the C14-22 aliphatic acids are not irritating. Eye irritation potential of the ammonium salts does not follow chain length dependence; the C18 ammonium salts are corrosive to the eyes. Dermal absorption: The in vitro penetration of C10, C12, C14, C16 and C18 fatty acids (as sodium salt solutions) through rat skin decreases with increasing chain length. At 86.73 ug C16/cm2 and 91.84 ug C18/cm2, about 0.23% and less than 0.1% of the C16 and C18 soap solutions is absorbed after 24 h exposure, respectively. Sensitisation: No sensitisation data were located. Repeat dose toxicity: Repeated dose oral (gavage or diet) exposure to aliphatic acids did not result in systemic toxicity with NOAELs greater than the limit dose of 1000 mg/kg bw. .

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Mutagenicity
Aliphatic acids do not appear to be mutagenic or clastogenic in vitro or in vivo
Carcinogenicity
No data were located for carcinogenicity of aliphatic fatty acids.
Reproductive toxicity
No effects on fertility or on reproductive organs, or developmental effects were observed in studies on aliphatic acids and the NOAELs correspond to the maximum dose tested. The weight of evidence supports the lack of reproductive and developmental toxicity potential of the aliphatic acids category.

Given the large number of substances in this category, their closely related chemical structure, expected trends in physical chemical properties, and similarity of toxicokinetic properties, both mammalian and aquatic endpoints were filled using read-across to the closest structural analogue, and selecting the most conservative supporting substance effect level.
Structure-activity relationships are not evident for the mammalian toxicity endpoints.That is, the low mammalian toxicity of this category of substances limits the ability to discern structural effects on biological activity. Regardless, the closest structural analogue with the most conservative effect value was selected for read across. Irritation is observed for chain lengths up to a cut-off" at or near 12 carbons).
Metabolism:
The aliphatic acids share a common degradation pathway in which they are metabolized to acetyl-CoA or other key metabolites in all living systems. Common biological pathways result in structurally similar breakdown products, and are, together with the physico-chemical properties, responsible for similar environmental behavior and essentially identical hazard profiles with regard to human health.
Differences in metabolism or biodegradability of even and odd numbered carbon chain compounds or saturated/ unsaturated compounds are not expected; even-and odd-numbered carbon chain compounds, and the saturated and unsaturated compounds are naturally occurring and are expected to be metabolized and biodegraded in the same manner.
The acid and alkali salt forms of the homologous aliphatic acid are expected to have many similar physicochemical and toxicological properties when they become bioavailable; therefore,data read across is used for those instances where data are available for the acid form but not the salt, and vice versa. In the gastrointestinal tract, acids and bases are absorbed in the undissociated (non-ionised) form by simple diffusion or by facilitated diffusion. It is expected that both the acids and the salts will be present in (or converted to) the acid form in the stomach. This means that for both aliphatic acid or aliphatic acid salt,the same compounds eventually enter the small intestine, where equilibrium, as a result of increased pH, will shift towards dissociation (ionised form).
Hence, the situation will be similar for compounds originating from acids and therefore no differences in uptake are anticipated
Note that the saturation or unsaturation level is not a factor in the toxicity of these substances and is not a critical component of the read across process..
Toxicokinetics:
The turnover of the [14C] surfactants in the rat showed that there was no significant difference in the rate or route of excretion of 14C given by intraperitoneal or subcutaneous administration. The main route of excretion was as 14CO2 in the expired air at 6 h after administration. The remaining material was incorporated in the body. Longer fatty acid chains are more readily incorporated than shorter chains. At ca. 1.55 and 1.64 mg/kg bw, 71% of the C16:0 and 56% of the C18:0 was incorporated and 21% and 38% was excreted as 14CO2, respectively.

Glycidyl fatty acid esters (GEs), one of the main contaminants in processed oils, are mainly formed during the deodorisation step in the refining process of edible oils and therefore occur in almost all refined edible oils. GEs are potential carcinogens, due to the fact that they readily hydrolyze into the free form glycidol in the gastrointestinal tract, which has been found to induce tumours in various rat tissues. Therefore, significant effort has been devoted to inhibit and eliminate the formation of GEs
GEs contain a common terminal epoxide group but exhibit different fatty acid compositions. This class of compounds has been reported in edible oils after overestimation of 3-monochloropropane-1,2-diol (3-MCPD) fatty acid esters analysed by an indirect method , 3-MCPD esters have been studied as food processing contaminants and are found in various food types and food ingredients, particularly in refined edible oils.
3-Monochloropropane-1,2-diol (3-MCPD) and 2-monochloropropane-1,3-diol (2-MCPD) are chlorinated derivatives of glycerol (1,2,3-propanetriol). 3- and 2-MCPD and their fatty acid esters are among non-volatile chloropropanols, Glycidol is associated with the formation and decomposition of 3- and 2-MCPD. It forms monoesters with fatty acids (GE) during the refining of vegetable oils. Chloropropanols are formed in HVP during the hydrochloric acid-mediated hydrolysis step of the manufacturing process. In food production, chloropropanols form from the reaction of endogenous or added chloride with glycerol or acylglycerol.
Although harmful effects on humans and animals have not been demonstrated, the corresponding hydrolysates, 3-MCPD and glycidol, have been identified as rodent genotoxic carcinogens, ultimately resulting in the formation of kidney tumours (3-MCPD) and tumours at other tissue sites (glycidol). Therefore, 3-MCPD and glycidol have been categorised as ‘‘possible human carcinogens (group 2B) and “probably carcinogenic to humans (group 2A), respectively, by the International Agency for Research on Cancer (IARC).
Diacylglyceride (DAG) based oils produced by one company were banned from the global market due to “high levels” of GEs.
Several reports have also suggested that a bidirectional transformation process may occur not only between glycidol and 3-MCPD but also their esterified forms in the presence of chloride ions. The transformation rate of glycidol to 3-MCPD was higher than that of 3-MCPD to glycidol under acidic conditions in the presence of chloride ion.
Precursors of GEs in refined oils have been identified as partial acylglycerols, that is, DAGs and monoacylglycerides (MAGs); however, whether they also originate from triacylglycerides (TAGs) is still a topic of controversial debates. Several authors noted that pure TAGs were stable during heat treatment (such as 235 deg C) for 3 h and were therefore not involved in the formation of GEs. However, experimental results have shown that small amounts of GEs are present in a heat-treated oil model consisting of almost 100% TAGs. The formation of GEs from TAGs can be attributed to the pyrolysis of TAGs to DAGs and MAGs. In contrast, 3-MCPD esters in refined oils can be obtained from TAG . Presently, the mechanism for the formation of GE intermediates and the relationship between GEs and 3-MCPD esters are still unknown.
For Group E aliphatic esters (polyol esters):
According to a classification scheme described by the American Chemistry Council' Aliphatic Esters Panel, Group E substances are esters of monoacids, mainly common fatty acids, and trihydroxy or polyhydroxyalcohols or polyols, such as pentaerythritol (PE), 2-ethyl-2-(hydroxymethyl)-1,3-propanediol or trimethylolpropane (TMP), and dipentaerythritol (diPE). The Group E substances often are referred to as "polyol esters" The polyol esters are unique in their chemical characteristics since they lack beta-tertiary hydrogen atoms, thus leading to stability against oxidation and elimination. The fatty acids often range from C5-C10 to as high as C18 (e.g., oleic, stearic, isostearic, tall oil fatty acids) in carbon number and generally are derived from naturally occurring sources. Group E esters may have multiple ester linkages and may include mixed esters derived from different carbon-length fatty acid mixtures. The lack of beta-tertiary hydrogen atoms in the structure of the polyol esters makes them characteristically and chemically stable against oxidation and elimination in comparison to other ester classes or groups. For these reasons, trimethylolpropane (TMP) and pentaerythritol (PE) esters with fatty acids of C5 to C10 carbon-chain length have applications as synthetic lubricants for passenger car motor oil and military and civilian jet engines. TMP and PE esters of C18 acids (e.g., isostearic and oleic acids) also have found use in synthetic lubricant applications, including refrigeration lubricants and hydraulic fluids. Because of their higher thermal stability characteristics, they also find use in a variety of high temperature applications such as industrial oven chain oils, high temperature greases, fire resistant transformer coolants and turbine engines
Polyol esters that are extensively esterified also have greater polarity, less volatility and enhanced lubricity characteristics. **Acute toxicity:**
Depending on the degree of esterification, the polyol esters can be resistant or slow towards chemical or enzymatic hydrolysis (i.e., esterase or lipases) as a result of steric hindrance. PE and diPE esters that are capable of being enzymatically hydrolyzed will generate pentaerythritol or dipentaerythritol, and the corresponding fatty acids which, for most of the Group E esters, are comprised mainly of oleic, linoleic and stearic acids as well as the fatty acids in the C5-10 carbon-length. Similarly, TMP esters can undergo metabolism to yield trimethylolpropane (2-ethyl-2-hydroxymethyl-1,3-propanediol) and fatty acid constituents. Pentaerythritol and trimethylolpropane have been reported to have a low order of toxicity The acute oral LD50 for these substances was greater than 2000 mg/kg indicating a relatively low order of toxicity. The similarity in the low order of toxicity for these substances is consistent with their similar chemical structure and physicochemical properties.
Metabolic studies of polyglyceryl esters indicated that these esters are hydrolyzed in the gastrointestinal (GI) tract, and utilization and digestibility studies supported the assumption that the fatty acid moiety is metabolized in the normal manner. Analytical studies have produced no evidence of accumulation of the polyglycerol moiety in body tissues.

Continued...

	In an acute dermal toxicity study in rats, the LD50 of 1,2,3-propanetriol, homopolymer, diisooctadecanoate was>5000 mg/kg Low toxicity was reported in acute oral studies. In rats, the LD50 >2000 mg/kg for polyglyceryl-3 caprate, polyglyceryl-3 caprylate, polyglyceryl-4 caprate, diisostearoyl polyglyceryl-3 dimer dilinoleate, and the LD50 was >5000 mg/kg for polyglyceryl-3 iso-stearate, polyglyceryl-3-oleate, polyglyceryl-2 diisostearate and polyglyceryl-3 diisostearate. The ability to enhance skin penetration was examined for several of the polyglyceryl fatty acid esters. **Repeat dose toxicity:** Polyol esters are generally well tolerated by rats in 28-day oral toxicity studies. NOAEL for these substances was 1000 mg/kg/day in Sprague-Dawley rats. The TMP ester of heptanoic and octanoic acid did not produce signs of overt systemic toxicity at any dose levels tested (i.e., 100, 300, and 1000 mg/kg/day). There were no treatment-related clinical in-life, functional observation battery, or gross postmortem findings. There were no treatment related mortality, and no adverse effects on body weight, food consumption, clinical laboratory parameters, or organ weights. However, there were increased numbers of hyaline droplets in the proximal cortical tubular epithelium of the 300 and 1000 mg/kg/day in male rats. Based on these findings (hyaline droplets), the NOAEL for this polyol ester was established at 100 mg/kg/day for male rats. Hyaline droplet formation observed in the male kidneys is believed to be a sex/species condition specific to only male rats, which has little relevance to humans. The results from these repeated dose dermal toxicity studies suggest that polyol esters exhibit a low order of toxicity following repeated application. This may be attributable to similarities in their chemical structures, physicochemical properties, and common metabolic pathways (i.e., esters can be enzymatically hydrolyzed to the corresponding polyalcohol and the corresponding fatty acids) The polyol, hexanedioic acid, mixed esters with decanoic acid, heptanoic acid, octanoic acid and PE, was applied to the skin of groups of 10 (male and female) rats for five days a week for four (4) weeks at dose levels of 0, 125, 500 and 2000 mg/kg/day. Treated animals exhibited no signs indicative of systemic toxicity. No visible signs of irritation were observed a treatment sites. Microscopically, treated skin (viz., greater than or equal to 500 mg/kg/day) exhibited a dose-related increased incidence and severity of hyperplasia and hyperkeratosis of the epidermis and sebaceous gland hyperplasia. These effects were reversible. None of the minor changes in haematology and serum chemistry parameters were considered biologically significant. High dose females (2000 mg/kg/day) exhibited a significant increase in relative adrenal and brain weights when compared to the controls. These differences were attributed to the lower final body weight of the female animals. The NOAEL in this study for systemic toxicity was established as 500 mg /kg/day and 125 mg/kg/day for skin irritation. Two 28-day study conducted with fatty acids, C5-10, esters with pentaerythritol (CAS RN: 68424-31-7) and dipentaerythritol ester of n-C5/iso-C9 acids (CAS RN: 647028-25-9) showed no signs of overt toxicity. The 90-day study pentaerythritol ester of pentanoic acids and isononanoic acid (CAS RN: 146289-36-3) did not show any signs of overt toxicity. However, increased kidney and liver weights in the male animals was observed. In conclusion, since the effects observed are not considered to be systemic and relevant for humans, the NOAEL was found to exceed 1000 mg/kg bw for all substances based on the result from the 28 and 90-day studies. **Reproductive and developmental toxicity:** Since metabolism of the polyol esters can occur, leading to the generation of the corresponding fatty acids and the polyol alcohol (such as pentaerthyritol, trimethylolpropane, and dipentaerythritol), the issue of whether these metabolites may pose any potential reproductive/developmental toxicity concerns is important.. However, the polyol alcohols such as pentaerthyritol, trimethylolpropane, and dipentaerythritol, would be expected to undergo further metabolism, conjugation and excretion in the urine. Available evidence indicates that these ester hydrolysates (i.e., hydrolysis products), primarily fatty acids (e.g., heptanoic, octanoic, and decanoic acids) and secondarily the polyol alcohols should exhibit a low order of reproductive toxicity. it can be concluded that this group of high molecular weight polyol esters should not produce profound reproductive effects in rodents. **Genotoxicity:** Polyols tested for genetic activity in the Salmonella assay, have been found to be inactive. Several polyol esters have been adequately tested for chromosomal mutation in the in vitro mammalian chromosome aberration assay, and all were inactive. Two TMP esters were also tested for in vivo chromosomal aberration in rats, and both demonstrated no activity. Thus, it is unlikely that these substances are chromosomal mutagens. **Carcinogenicity:** In a 2-yr study, 28 male and 28 female rats were fed 5% polyglyceryl ester in the diet. No adverse effects on body weight, feed consumption, haematology values, or survival rate were noted. Liver function tests and renal function tests performed at 59 and 104 wks of the study were comparable between the test group and a control group fed 5% ground nut oil. The carcass fat contained no polyglycerol, and the levels of free fatty acid, unsaponifiable residue and fatty acid composition of carcass fat were not different from the controls. Organ weights, tumour incidence and tumour distribution were similar in control and test groups. A complete histological examination of major organs showed nothing remarkable
Otis Bio-CLP & ANTIMONY DIAMYLDITHIOCARBAMATE & NAPHTHENIC DISTILLATE, HEAVY, HYDROTREATED (SEVERE)	No significant acute toxicological data identified in literature search.

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

Otis Bio-CLP

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

antimony diamyldithiocarbamate

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

fatty acids, soya, methyl esters

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

naphthenic distillate, heavy, hydrotreated (severe)

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	504h	Crustacea	>1mg/l	1

Continued...

	ErC50	72h	Algae or other aquatic plants	>1000mg/l	1
	EC50	48h	Crustacea	>1000mg/l	1
	EC50	96h	Algae or other aquatic plants	>1000mg/l	1

trimethylolpropane trioleate	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available

Legend: *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 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*

Toxic 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.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
	No Data available for all ingredients	No Data available for all ingredients

Bioaccumulative potential

Ingredient	Bioaccumulation
	No Data available for all ingredients

Mobility in soil

Ingredient	Mobility
	No Data available for all ingredients

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal

Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.
A Hierarchy of Controls seems to be common - the user should investigate:
- Reduction
- Reuse
- Recycling
- Disposal (if all else fails)

This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.
- **DO NOT** allow wash water from cleaning or process equipment to enter drains.
- It may be necessary to collect all wash water for treatment before disposal.
- In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.
- Where in doubt contact the responsible authority.
- Recycle wherever possible.
- Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified.
- Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or incineration in a licensed apparatus (after admixture with suitable combustible material).
- Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

SECTION 14 Transport information

Labels Required

Marine Pollutant

HAZCHEM •3Z

Land transport (ADG)

UN number 3082

Continued...

UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains antimony diamyldithiocarbamate)
Transport hazard class(es)	Class: 9 Subrisk: Not Applicable
Packing group	III
Environmental hazard	Environmentally hazardous
Special precautions for user	Special provisions: 274 331 335 375 AU01 Limited quantity: 5 L

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

Air transport (ICAO-IATA / DGR)

UN number	3082
UN proper shipping name	Environmentally hazardous substance, liquid, n.o.s. * (contains antimony diamyldithiocarbamate)
Transport hazard class(es)	ICAO/IATA Class: 9 ICAO / IATA Subrisk: Not Applicable ERG Code: 9L
Packing group	III
Environmental hazard	Environmentally hazardous
Special precautions for user	Special provisions: A97 A158 A197 A215 Cargo Only Packing Instructions: 964 Cargo Only Maximum Qty / Pack: 450 L Passenger and Cargo Packing Instructions: 964 Passenger and Cargo Maximum Qty / Pack: 450 L Passenger and Cargo Limited Quantity Packing Instructions: Y964 Passenger and Cargo Limited Maximum Qty / Pack: 30 kg G

Sea transport (IMDG-Code / GGVSee)

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

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

Not Applicable

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

Product name	Group
antimony diamyldithiocarbamate	Not Available
fatty acids, soya, methyl esters	Not Available
naphthenic distillate, heavy, hydrotreated (severe)	Not Available
trimethylolpropane trioleate	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
antimony diamyldithiocarbamate	Not Available
fatty acids, soya, methyl esters	Not Available
naphthenic distillate, heavy, hydrotreated (severe)	Not Available
trimethylolpropane trioleate	Not Available

Continued...

SECTION 15 Regulatory information

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

antimony diamyldithiocarbamate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

fatty acids, soya, methyl esters is found on the following regulatory lists

Not Applicable

naphthenic distillate, heavy, hydrotreated (severe) is found on the following regulatory lists

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

trimethylolpropane trioleate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (fatty acids, soya, methyl esters)
Canada - DSL	Yes
Canada - NDSL	No (antimony diamyldithiocarbamate; fatty acids, soya, methyl esters; naphthenic distillate, heavy, hydrotreated (severe))
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (fatty acids, soya, methyl esters)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (antimony diamyldithiocarbamate; fatty acids, soya, methyl esters)
Vietnam - NCI	No (antimony diamyldithiocarbamate)
Russia - FBEPH	No (fatty acids, soya, methyl esters; trimethylolpropane trioleate)
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.*

SECTION 16 Other information

Revision Date	17/01/2022
Initial Date	17/01/2022

SDS Version Summary

Version	Date of Update	Sections Updated
2.1	17/01/2022	Appearance, Ingredients, Physical Properties

Other information

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

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

Definitions and abbreviations

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

Continued...

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