Stihl Multioil Bio

Stihl Pty Ltd.

BWES: **3588-545**
Version No: **3.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements
Safety Data Sheet according to the Health and Safety at Work (Hazardous Substances) Regulations 2017

Issue Date: **03/12/2024**
Print Date: **06/12/2024**
L.GHS.AUS/NZ.EN.E

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

Product Identifier

Product name	Stihl Multioil Bio
Chemical Name	Not Applicable
Synonyms	0782 516 8500 A
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 manufacturer or supplier of the safety data sheet

Registered company name	**Stihl Pty Ltd.**
Address	5 Kingston Park Court, Knoxfield, Victoria, 3180, Australia | 9 Bishop Browne Place, East Tamaki, Auckland, 2013 New Zealand
Telephone	AU: +61 3 9215 6666 | NZ: +64 9262 4000
Fax	Not Available
Website	Not Available
Email	enquiries@stihl.com.au

Emergency telephone number

Association / Organisation	**Poisons Information Centre**
Emergency telephone number(s)	131 126 (AU)
Other emergency telephone number(s)	0800 764 766 (NZ)

SECTION 2 Hazards identification

Classification of the substance or mixture

HAZARDOUS CHEMICAL. NON-DANGEROUS GOODS. According to the WHS Regulations and the ADG Code.

Poisons Schedule	Not Applicable
Classification [1]	Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2A, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3
Legend:	*1. Classified by BWES; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

Label elements

Hazard pictogram(s)	
Signal word	**Warning**

Hazard statement(s)

H315	Causes skin irritation.
H319	Causes serious eye irritation.
H335	May cause respiratory irritation.

Precautionary statement(s) Prevention

Page 1 continued...

P271	Use only outdoors or in a well-ventilated area.
P261	Avoid breathing mist/vapours/spray.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P264	Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P337+P313	If eye irritation persists: Get medical advice/attention.
P302+P352	IF ON SKIN: Wash with plenty of water.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

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

Considered a Hazardous Substance according to the criteria of the New Zealand Hazardous Substances New Organisms legislation. Not regulated for transport of Dangerous Goods.

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Legend:	*1. Classified by BWES; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from Regulation (EU) No 1272/2008 - AnnexVI*
Determined by BWESusing GHS/HSNO criteria	6.3A, 6.4A, 6.1E (respiratory tract irritant)

Label elements

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SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
8001-21-6	>90	sunflower oil
27925-02-6	5	ricinoleic acid, homopolymer
10254-57-6	2	4,4'-methylene bis(dibutyldithiocarbamate)
110-25-8	0.099	oleoylsarcosine
Legend:	*1. Classified by BWES; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from Regulation (EU) No 1272/2008 - AnnexVI; 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 the eyes: ▸ Wash out immediately with fresh running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Seek medical attention without delay; if pain persists or recurs 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. For thermal burns: ▸ Decontaminate area around burn. ▸ Consider the use of cold packs and topical antibiotics. For first-degree burns (affecting top layer of skin) ▸ Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides. ▸ Use compresses if running water is not available. ▸ Cover with sterile non-adhesive bandage or clean cloth. ▸ Do NOT apply butter or ointments; this may cause infection. ▸ Give over-the counter pain relievers if pain increases or swelling, redness, fever occur. For second-degree burns (affecting top two layers of skin) ▸ Cool the burn by immerse in cold running water for 10-15 minutes. ▸ Use compresses if running water is not available. ▸ Do NOT apply ice as this may lower body temperature and cause further damage. ▸ Do NOT break blisters or apply butter or ointments; this may cause infection. ▸ Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape. To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort): ▸ Lay the person flat. ▸ Elevate feet about 12 inches. ▸ Elevate burn area above heart level, if possible. ▸ Cover the person with coat or blanket. ▸ Seek medical assistance. For third-degree burns Seek immediate medical or emergency assistance. In the mean time: ▸ Protect burn area cover loosely with sterile, nonstick bandage or, for large areas, a sheet or other material that will not leave lint in wound. ▸ Separate burned toes and fingers with dry, sterile dressings. ▸ Do not soak burn in water or apply ointments or butter; this may cause infection. ▸ To prevent shock see above. ▸ For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway. ▸ Have a person with a facial burn sit up. ▸ Check pulse and breathing to monitor for shock until emergency help arrives.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay.
Ingestion	▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Seek medical advice.

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.

Treat symptomatically.

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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.

SECTION 5 Firefighting measures

Extinguishing media

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

Special hazards arising from the substrate or mixture

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

Advice for firefighters

Fire Fighting	‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear full body protective clothing with breathing apparatus. ‣ Prevent, by any means available, spillage from entering drains or water course. ‣ Use water delivered as a fine spray to control fire and cool adjacent area. ‣ Avoid spraying water onto liquid pools. ‣ **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.
Fire/Explosion Hazard	‣ Combustible. ‣ Slight fire hazard when exposed to heat or flame. ‣ Heating may cause expansion or decomposition leading to violent rupture of containers. ‣ On combustion, may emit toxic fumes of carbon monoxide (CO). ‣ May emit acrid smoke. ‣ Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO_2) acrolein nitrogen oxides (NO_x) sulfur oxides (SO_x) other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes. **CARE**: Water in contact with hot liquid may cause foaming and a steam explosion with wide scattering of hot oil and possible severe burns. Foaming may cause overflow of containers and may result in possible fire.

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	Slippery when spilt. ‣ Remove all ignition sources. ‣ 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	Slippery when spilt. 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. ‣ No smoking, naked lights or ignition sources. ‣ Increase ventilation. ‣ Stop leak if safe to do so. ‣ Contain spill with sand, earth or vermiculite. ‣ Collect recoverable product into labelled containers for recycling. ‣ Absorb remaining product with sand, earth or vermiculite. ‣ Collect solid residues and seal in labelled drums for disposal. ‣ Wash area and prevent runoff into drains. ‣ 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	Rags wet / soaked with unsaturated hydrocarbons / drying oils may auto-oxidise; generate heat and, in-time, smoulder and ignite. This is especially the case where oil-soaked materials are folded, bunched, compressed, or piled together - this allows the heat to accumulate or even accelerate the reaction

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	Oily cleaning rags should be collected regularly and immersed in water, or spread to dry in safe-place away from direct sunlight or stored, immersed, in solvents in suitably closed containers. ▸ **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. ▸ Prevent concentration in hollows and sumps. ▸ **DO NOT** enter confined spaces until atmosphere has been checked. ▸ Avoid smoking, naked lights or ignition sources. ▸ 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. ▸ 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.
Other information	▸ Store in original containers. ▸ Keep containers securely sealed. ▸ No smoking, naked lights or ignition sources. ▸ Store in a cool, dry, well-ventilated area. ▸ Store away from incompatible materials and foodstuff containers. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Glass container is suitable for laboratory quantities ▸ **DO NOT** use aluminium or galvanised containers ▸ Metal can or drum ▸ Packaging as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▸ Avoid reaction with oxidising agents · Materials soaked with plant/ vegetable derived (and rarely, animal) oils may undergo spontaneous combustion · The more unsaturated is the fatty acid component, the more susceptible is the oil to oxidation and spontaneous combustion. · Many vegetable and animal oils absorb oxygen from the air to form oxidation products. This oxidation process produces heat and the resultant increase in temperature accelerates the oxidation process. · Drying oils such as linseed, tung, poppy and sunflower oils and semi-drying oils such as soya bean, tall oil, corn, cotton and castor oils all absorb oxygen readily and thus experience the self-heating process. · Cotton fibres are readily ignited and if contaminated with an oxidisable oil, may ignite unless heat can be dissipated · Vegetable oils and some animal fats undergo undesirable deterioration reactions in the presence of oxygen from the air becoming rancid accompanying off-flavours and smells. The mechanism of autoxidation of vegetable oils is classically regarded as following a number of stages being: · a usually slow initiation phase · a usually rapid propagation · and a termination phase The initiation phase involves the formation of a free radical from a triglyceride molecule in the fat: this may be promoted by the presence of heavy metals in the oil, or by heat or light. The next stage is the reaction of the triglyceride free radical with oxygen to produce a peroxide free radical, which can react with another triglyceride to produce a hydroperoxide and another triglyceride free radical. Steps 2 and 3 can repeat in a chain reaction until two peroxy free radicals collide and neutralise each other. Some drying oils produce cyclic peroxides instead of hydroperoxides. Autooxidation may also occur in saturated fatty acids and their esters. Monohydroperoxides are formed. Although all carbon atoms are subject to oxidation, preferential oxidation appears to occur towards the centre of the molecule. Autoxidation is assisted by higher ambient temperatures (the rate doubling for every ten degrees Centigrade rise) and by the presence of heavy metal ions, especially copper. The degree of unsaturation of the oil is also relevant to shelf-life; oils with a high linolenic fatty acid content (3 double bonds) being more prone that those with a higher saturated fatty acid content. Autoxidation can be minimized by the presence of anti-oxidants, which can act as free-radical inhibitors. Vegetable oils should therefore be stored in a cool place away from heat and light, and should only come into contact with inert (glass of stainless steel) containers which will not leach heavy metals. Blanketing under nitrogen should be considered in bulk storages.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

Ingredient	Original IDLH	Revised IDLH
sunflower oil	Not Available	Not Available
ricinoleic acid, homopolymer	Not Available	Not Available
4,4'-methylene bis(dibutyldithiocarbamate)	Not Available	Not Available
oleoylsarcosine	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
sunflower oil	E	≤ 0.1 ppm
ricinoleic acid, homopolymer	E	≤ 0.1 ppm
4,4'-methylene bis(dibutyldithiocarbamate)	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.*	

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

MATERIAL DATA

Exposure controls

Appropriate engineering controls

Care: Atmospheres in bulk storages and even apparently empty tanks may be hazardous by oxygen depletion. Atmosphere must be checked before entry.

Requirements of State Authorities concerning conditions for tank entry must be met. Particularly with regard to training of crews for tank entry; work permits; sampling of atmosphere; provision of rescue harness and protective gear as needed
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.

Individual protection measures, such as personal protective equipment

Eye and face protection

- Safety glasses with side shields.
- Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent]
- 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].

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.

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	· 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)

Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator
up to 10	1000	A-AUS / Class1 P2	-
up to 50	1000	-	A-AUS / Class 1 P2
up to 50	5000	Airline *	-
up to 100	5000	-	A-2 P2
up to 100	10000	-	A-3 P2
100+			Airline**

* - Continuous Flow ** - Continuous-flow or positive pressure demand
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.

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 Available
pH (as supplied)	Not Available	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Not Available	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

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Heat of Combustion (kJ/g)	Not Available	**Ignition Distance (cm)**	Not Available
Flame Height (cm)	Not Available	**Flame Duration (s)**	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	**Enclosed Space Ignition Deflagration Density (g/m3)**	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	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Inhalation hazard is increased at higher temperatures. Not normally a hazard due to non-volatile nature of product Inhalation of oil droplets/ aerosols may cause discomfort and may produce chemical pneumonitis. Fine mists generated from plant/ vegetable (or more rarely from animal) oils may be hazardous. Extreme heating for prolonged periods, at high temperatures, may generate breakdown products which include acrolein and acrolein-like substances.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual.
Skin Contact	The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either ▸ produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or ▸ produces significant, but moderate, 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. Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. 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	Limited evidence or practical experience suggests, that the material may cause eye irritation in a substantial number of individuals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following. 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. Glyceryl triesters (triglycerides), following ingestion, are metabolised to monoglycerides, free fatty acids and glycerol, all of which are absorbed in the intestinal mucosa and undergo further metabolism. Medium chain triglycerides (C8-C10) appear to have relatively rapid metabolism and elimination from blood and tissues compared to long chain triglycerides (C16-C18). Little or no acute, subchronic or chronic oral toxicity was seen in animal studies unless levels approached a significant percentage of calorific intake. Subcutaneous injections of tricaprylin in rats over a five-week period caused granulomatous reaction characterised by oil deposits surrounded by macrophages. Diets containing substantial levels of tributyrin produced gastric lesions in rats fed for 3-35 weeks; the irritative effect of the substance was thought to be the cause of tissue damage. Dermal application was not associated with significant irritation in rabbit skin; ocular exposures were, at most, mildly irritating to rabbit eyes. No evidence of sensitisation or photosensitisation was seen in a guinea pig maximisation test. Most of the genotoxicity test systems were negative. Tricaprylin, trioctanoin and triolein have been used, historically, as vehicles in carcinogenicity testing of other chemicals. In one study, subcutaneous injection of tricaprylin, in newborn mice, produced more tumours in lymphoid tissue than were seen in untreated animals whereas, in another study, subcutaneous or intraperitoneal injection in 4- to 6-week old female mice produced no tumours. Trioctanoin injected subcutaneously in hamster produced no tumours; when injected intraperitoneally in pregnant rats there was an increase in mammary tumours among the off-spring but similar studies in pregnant hamsters and rabbits showed no tumours in the off-spring. The National Toxicological Program conducted a 2-year study in rats given tricaprylin by gavage. The treatment was associated with a statistically significant dose-related increase in pancreatic acinar cell hyperplasia and adenoma but there were no acinar carcinomas. Tricaprylin is not teratogenic to mice or rats but some reproductive effects were seen in rabbits. A low level of foetal eye abnormalities and a small percentage of abnormal sperm were reported in mice injected with trioctanoin. Trioctanoin was also used as a vehicle control in a sperm abnormality test. Ten male control mice received an intraperitoneal injection of 0.25 ml trioctanoin 0.05 g/kg of benz[a]pyrene (known reproductive toxicant and mutagen) daily for 5 days and sperm from caudae epididymides analysed. Based on these studies there is no sufficient evidence to classify the trioctanoin as reproductive toxicant. In the human body, high levels of triglycerides in the bloodstream have been linked to atherosclerosis, heart disease and stroke. However, the relative negative impact of raised levels of triglycerides compared to that of LDL:HDL ratios is as yet unknown. The risk can be partly accounted for by a strong inverse relationship between triglyceride level and HDL-cholesterol level. But the risk is also due to high triglyceride levels increasing the quantity of small, dense LDL particles

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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.
Synthetic 1,2-diglycerides of short chain (C6, C8, C10) fatty acids are activators of protein kinase C (PKC). PKC is a serine-threonine kinase which also requires calcium ion for its activation. Activated PKC phosphorylates proteins of the cellular signal cascade, which eventually induce expression of growth regulatory genes. This, in turn, may promote the growth of tumours. Structural analogues of the 1,2-diglycerides, such as the phorbol esters, have been shown to strongly promote such an event.
In biochemical signaling, diacylglycerol (DAG) functions as a second messenger signaling lipid, and is a product of the hydrolysis of the phospholipid PIP2 (phosphatidylinositolbisphosphate) by the enzyme phospholipase C (PLC) (a membrane-bound enzyme) that, through the same reaction, produces inositol trisphosphate (IP3). Although inositol trisphosphate (IP3) diffuses into the cytosol, DAG remains within the plasma membrane due to its hydrophobic properties. IP3 stimulates the release of calcium ions from the smooth endoplasmic reticulum, whereas DAG is a physiological activator of protein kinase C (PKC). The production of DAG in the membrane facilitates translocation of PKC from the cytosol to the plasma membrane.
Glyceryl dilaurate, glyceryl diarachidate, glyceryl dibehenate, glyceryl dierucate, glyceryl dihydroxystearate, glyceryl diisopalmitate, glyceryl diisostearate, glyceryl dilinoleate, glyceryl dimyristate, glyceryl dioleate, glyceryl diricinoleate, glyceryl dipalmitate, glyceryl dipalmitoleate, glyceryl distearate, glyceryl palmitate lactate, glyceryl stearate citrate, glyceryl stearate lactate, and glyceryl stearate succinate are diacylglycerols (also known as DAGs, diglycerides or glyceryl diesters) that function as skin conditioning agents-emollients in cosmetics. Only glyceryl dilaurate (up to 5%), glyceryl diisostearate (up to 43%), glyceryl dioleate (up to 2%), glyceryl distearate (up to 7%), and glyceryl stearate lactate (up to 5%) are reported to be in current use. Production proceeds from fully refined vegetable oils, which are further processed using hydrogenation and fractionation techniques, and the end products are produced by reacting selected mixtures of the partly hydrogenated, partly fractionated oils and fats with vegetable-derived glycerine to yield partial glycerides. In the final stage of the production process, the products are purified by deodorization, which effectively removes pesticide residues and lower boiling residues such as residues of halogenated solvents and aromatic solvents. Diglycerides have been approved by the Food and Drug Administration (FDA) for use as indirect food additives. Nominally, these ingredients are 1,3-diglycerides, but are easily isomerised to the 1,2-diglycerides form. The 1,3-diglyceride isomer is not a significant toxicant in acute, short-term, subchronic, or chronic animal tests. Glyceryl dilaurate was a mild primary irritant in albino rabbits, but not a skin sensitiser in guinea pig maximization tests. Diacylglycerol oil was not genotoxic in the Ames test, in mammalian Chinese hamster lung cells, or in a rodent bone marrow micronucleus assay. An eye shadow containing 1.5% glyceryl dilaurate did not induce skin irritation in a single insult patch test, but mild skin irritation reactions to a foundation containing the same concentration were observed. A trade mixture containing an unspecified concentration of glyceryl dibehenate did not induce irritation or significant cutaneous intolerance in a 48-h occlusive patch test. In maximization tests, neither an eye shadow nor a foundation containing 1.5% glyceryl dilaurate was a skin sensitiser. Sensitisation was not induced in subjects patch tested with 50% *w/w* glyceryl dioleate in a repeated insult, occlusive patch test. Glyceryl palmitate lactate (50% *w/v*) did not induce skin irritation or sensitization in subjects patch tested in a repeat-insult patch test. Phototoxicity or photoallergenicity was not induced in healthy volunteers tested with a lipstick containing 1.0% Glyceryl rosinate. Two diacylglycerols, 1-oleoyl-2-acetoyl-sn-glycerol and 1,2-dipalmitoyl-sn-glycerol, did not alter cell proliferation (as determined by DNA synthesis) in normal human dermal fibroblasts in vitro at doses up to 10 μg/ml. In the absence of initiation, Glyceryl distearate induced a moderate hyperplastic response in randomly bred mice of a tumor-resistant strain, and with 9,10-dimethyl-1,2-benzanthracene (DMBA) initiation, an increase in the total cell count was observed. In a glyceryl monoester study, a single application of DMBA to the skin followed by 5% glyceryl stearate twice weekly produced no tumors, but slight epidermal hyperplasia at the site of application. Glyceryl dioleate induced transformation in 3-methylcholanthrene-initiated BALB/3T3 A31-1-1 cloned cells in vitro. A tumour-promoting dosing regimen that consisted of multiple applications of 10 μmol of a 1,2-diacylglycerol (sn-1,2-didecanoylglycerol) to female mice twice daily for 1 week caused more than a 60% decrease in protein kinase C (PKC) activity and marked epidermal hyperplasia. Applications of 10 umol sn-1,2-didecanoylglycerol twice weekly for 1 week caused a decrease in cytosolic PKC activity, an increase in particulate PKC activity, and no epidermal hyperplasia. In studies of the tumour-promoting activity of 1,2-diacylglycerols, dose and the exposure regimen by which the dose is delivered play a role in tumor promotion. The 1,2-diacylglycerol–induced activation of PKC may also relate to the saturation of the fatty acid in the 1 or 2 position; 1,2-Diacylglycerols with two saturated fatty acids are less effective. Also, the activity of 1,2-diacylglycerols may be reduced when the fatty acid moiety in the structure is a long-chain fatty acid. A histological evaluation was performed on human skin from female volunteers (18 to 56 years old) who had applied a prototype lotion or placebo formulation, both containing 0.5% Glyceryl Dilaurate, consecutively for 16 weeks or 21 weeks. Skin irritation was not observed in any of the subjects tested. Biopsies (2 mm) taken from both legs of five subjects indicated no recognizable abnormalities of the skin; the epidermis was normal in thickness, and there was no evidence of scaling, inflammation, or neoplasms in any of the tissues that were evaluated. The available safety test data indicate that diglycerides in the 1,3-diester form do not present any significant acute toxicity risk, nor are these ingredients irritating, sensitizing, or photosensitising. Whereas no data are available regarding reproductive or developmental toxicity, there is no reason to suspect any such toxicity because the dermal absorption of these chemicals is negligible. 1,3-Diglycerides contain 1,2-diglycerides, raising the concern that 1,2-diglycerides could potentially induce hyperplasia. Data regarding the induction of PKC and the tumour promotion potential of 1,2-diacylglycerols increases the level of concern. Most of the diglycerides considered above, however, have fatty acid chains longer than 14 carbons and none have mixed saturated/unsaturated fatty acid moieties. In a 21-week use study of a prototype lotion containing 0.5% glyceryl dilaurate (a 14-carbon chain fatty acid) indicated no evidence of scaling, inflammation, or neoplasms in biopsy specimens. Also, DNA synthesis assays on glyceryl dilaurate and glyceryl distearate indicated that neither chemical altered cell proliferation (as determined by DNA synthesis) in normal human dermal fibroblasts in vitro at doses up to 10 ug/ml. However the concentration of these ingredients can vary (up to 43% for glyceryl diisostearate in lipstick), the frequency of application can be several times daily, and the proportion of diglycerides that are inactive 1,3 isomers versus potentially biologically active 1,2 isomers is unknown; as a precaution it is believed that each use should be examined to ensure the absence of epidermal hyperplasia during product development and testing. In the absence of inhalation toxicity data on the glyceryl diesters it is thought that these ingredients can be used safely in aerosolised products because they are not respirable. Although there are gaps in knowledge about product use, the overall information available on the types of products in which these ingredients are used and at what concentration indicate a pattern of use. Within this overall pattern of use, the CIR Expert Panel considers all ingredients in this group to be safe.
International Journal of Toxicology, Vol. 26, No. 3 Suppl, 1-30 (2007)
Repeated ingestion of linoleic acid, by man produces changes in platelet function tests. Animals tests show weight loss, progressive secondary anaemia, leukopenia and damage to erythrocyte and platelet membranes

	TOXICITY	IRRITATION
Stihl Multioil Bio	Not Available	Not Available
sunflower oil		

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	Oral (Rat) LD50: >10 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]

ricinoleic acid, homopolymer	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

4,4'-methylene bis(dibutyldithiocarbamate)	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: 16000 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]

oleoylsarcosine	**TOXICITY**	**IRRITATION**
	Inhalation (Rat) LC50: 0.575 mg/l4h[1]	Not Available
	Oral (Rat) LD50: >5000 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*

SUNFLOWER OIL

For omega 6 fatty acids and derivatives:
Some medical research suggests that excessive levels of certain omega-6 fatty acids relative to certain omega-3 fatty acids may increase the probability of a number of diseases.
Modern Western diets typically have ratios of omega-6 to omega-3 in excess of 10 to 1, some as high as 30 to 1; the average ratio of omega-6 to omega-3 in the Western diet is 15:1–16.7:1. Humans are thought to have evolved with a diet of a 1-to-1 ratio of omega-6 to omega-3 and the optimal ratio is thought to be 4 to 1 or lower although some sources suggest ratios as low as 1:1). A ratio of 2–3:1 omega 6 to omega 3 helped reduce inflammation in patients with rheumatoid arthritis. A ratio of 5:1 had a beneficial effect on patients with asthma but a 10:1 ratio had a negative effect. A ratio of 2.5:1 reduced rectal cell proliferation in patients with colorectal cancer, whereas a ratio of 4:1 had no effect.
Excess omega-6 fatty acids from vegetable oils interfere with the health benefits of omega-3 fats, in part because they compete for the same rate-limiting enzymes. A high proportion of omega-6 to omega-3 fat in the diet shifts the physiological state in the tissues toward the pathogenesis of many diseases: prothrombotic, proinflammatory and proconstrictive.
Chronic excessive production of omega-6 eicosanoids is correlated with arthritis, inflammation, and cancer. Many of the medications used to treat and manage these conditions work by blocking the effects of the COX-2 enzyme. Many steps in formation and action of omega-6 prostaglandins from omega-6 arachidonic acid proceed more vigorously than the corresponding competitive steps in formation and action of omega-3 hormones from omega-3 eicosapentaenoic acid The COX-1 and COX-2 inhibitor medications, used to treat inflammation and pain, work by preventing the COX enzymes from turning arachidonic acid into inflammatory compounds.The LOX inhibitor medications often used to treat asthma work by preventing the LOX enzyme from converting arachidonic acid into the leukotrienes. Many of the anti-mania medications used to treat bipolar disorder work by targeting the arachidonic acid cascade in the brain.
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.
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

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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. .
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.
Epoxidation of double bonds is a common bioactivation pathway for alkenes. The allylic epoxides, so formed, were found to possess sensitising capacity in vivo and in vitro and to chemically reactive towards a common hexapeptide containing the most common nucleophilic amino acids. Further-more, a SAR study of potentially prohaptenic alkenes demonstrated that conjugated dienes in or in conjunction with a six-membered ring are prohaptens, whereas related alkenes containing isolated double bonds or an acyclic conjugated diene were weak or nonsensitizing compounds.This difference in sensitizing capacity of conjugated dienes as compared to alkenes with isolated double bonds was found to be due to the high reactivity and sensitizing capacity of the allylic epoxides metabolically formed from conjugated dienes.
Allergic Contact Dermatitis—Formation, Structural Requirements,and Reactivity of Skin Sensitizers.
Ann-Therese Karlberg et al: Chem. Res. Toxicol. 2008, 21, pp 53–69
https://ftp.cdc.gov/pub/Documents/OEL/06.%20Dotson/References/Karlberg_2008.pdf
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,

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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.

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

Coronaric and vernolic acids also form non-enzymatically when linoleic acid is exposed to oxygen and/or UV radiation as a result of the spontaneous process of autooxidation. This autoxidation complicates studies in that it is often difficult to determine if these epoxy fatty acids identified in linoleic acid-rich plant and mammalian tissues represent actual tissue contents or are artifacts formed during their isolation and detection

At very high concentrations, the linoleic acid-derived set of optical isomers, coronaric acid (i.e. isoleukotoxin), possesses activities similar to that of other structurally unrelated leukotoxins viz., It is toxic to leukocytes and other cell types and when injected into rodents produce multiple organ failure and respiratory distress. These effects appear due to its conversion to its dihydroxy counterparts, 9S,10R- and 9R,10S-dihydroxy-12(Z)-octadecaenoic acids by soluble epoxide hydrolase. Some studies suggest but have not yet proven that isoleukotoxin, acting primarily if not exclusively through its dihydroxy counterparts, is responsible for or contribute to multiple organ failure, the acute respiratory distress syndrome, and certain other cataclysmic diseases in humans (see epoxygenase section on linoleic acid). Vernolic acid (i.e. leukotoxin) shares a similar metabolic fate in being converted by soluble epoxide hydrolase to its dihydroxide counterparts and toxic actions of these hydroxide counterparts.

At lower concentrations, isoleukotoxin and its dihydroxy counterparts can protect from the toxic actions cited above that occur at higher concentrations of isoleukotoxin and leukotoxin; they may also share with the epoxides of arachidonic acid, i.e. the epoxyeicosatreienoates, anti-hypertension activities

The epoxygenases are known to metabolize linoleic acid, at its 12,13 carbon-carbon double bond to form (+) and (-) epoxy optical isomers viz., the 9S,10R-epoxy-12(Z)-octadecaenoic and 9R,10S-epoxy-12(Z)-octadecaenoic acids; this set of optical isomers is also termed vernolic acid, linoleic acid 9:10-oxide, and leukotoxin. Cytochrome P450 (CYP) subtype CYPC2C9 and the other arachidonic acid-metabolizing CYPs are thought to, likewise, attack linoleic acid at its 9,10 carbon-carbon double bound to form 12S,13R-epoxy-9(Z)-octadecaenoic and 12R,13S-epoxy-9(Z)-octadecaenoic acid optical isomers; this set of optical isomers is also termed coronaric acid, linoleic acid 12,13-oxide, and isoleukotoxin. These linoleic acid-derived leukotoxin and isoleukotoxin sets of optical isomers possess activities similar to that of other leukotoxins such as the pore-forming leukotoxin family of RTX toxin virulence factor proteins

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secreted by gram-negative bacteria, e.g. Aggregatibacter actinomycetemcomitans and E. coli. That is, they are toxic to leukocytes as well as many other cell types and when injected into rodents produce multiple organ failure and respiratory distress. As described above, these effects appear due to the conversion of leukotoxin to its dihydroxy counterparts, 9S,10R- and 9R,10S-dihydroxy-12(Z)-octadecaenoic acids, and isoleukotoxin to its 12R,13S- and 12S,13R-dihydroxy-9(Z)-octadecenoic acid counterparts by soluble epoxide hydrolase. Some studies suggest but have not proven that leukotoxin and isoleukotoxin, acting primarily if not exclusively through their respective dihydroxy counterparts, are responsible for or contribute to multiple organ failure, respiratory distress, and certain other cataclysmic diseases in humans.
Oxidative stress in cells and tissues produces free radical and singlet oxygen oxidations of linoleic acid to generate 13-HpODEs, 9-HpODEs, 13-HODEs, and 9-HODEs; these non-enzymatic reactions produce or are suspected but not proven to produce approximately equal amounts of their S and R stereoisomers. Free radical oxidations of linoleic acid also produce 13-EE-HODE, 9-hydroxy-10E,12-E-octadecadienoic acid, 9-hydroxy-10E,12-Z-octadecadienoic acid, and 11-hydroxy-9Z,12Z-octadecaenoic acid while singlet oxygen attacks on linoleic acid produce (presumably) racemic mixtures of 9-hydroxy-10E,12-Z-octadecadienoic acid, 10-hydroxy-8E,12Z-octadecadienoic acid, and 12-hydroxy-9Z-13-E-octadecadienoic acid. 4-Hydroxynonenal (i.e. 4-hydroxy-2E-nonenal or HNE) is also a peroxidation product of 13-HpODE. Since oxidative stress commonly produces both free radicals and singlet oxygen, most or all of these products may form together in tissues undergoing oxidative stress. Free radical and singlet oxygen oxidations of linoleic acid produce a similar set of 13-HODE metabolites. Studies attribute these oxidations to be major contributors to 13-HODE production in tissues undergoing oxidative stress including in humans sites of inflammation, steatohepatitis, cardiovascular disease-related atheroma plaques, neurodegenerative disease, etc.
For polyunsaturated fatty acids and oils (triglycerides)
Studies on animals have shown a link between polyunsaturated fat and the incidence of tumours. In some of these studies the incidence of tumours increased with increasing intake of polyunsaturated fat, up to about 5% of total energy, near to the middle of the current dietary intake in humans.
The propensity for polyunsaturated fats to oxidise is another possible risk factor. This leads to the generation of free radicals and eventually to rancidity
Research evidence suggests that consuming high amounts of polyunsaturated fat may increase the risk of cancer spreading. Researchers found that linoleic acid in polyunsaturated fats produced increasing membrane phase separation, and thereby increased adherence of circulating tumour cells to blood vessel walls and remote organs.
At least one study in mice has shown that consuming high amounts of polyunsaturated fat (but not monounsaturated fat) may increase the risk of metastasis in cancer.
Lipid peroxides with complex components can damage macromolecules, such as DNA, proteins, and membrane lipids. Some components of lipid peroxides, for example, 4,5(E)-epoxy-2(E)-heptenal (EH) can react with L-lysine and damage proteins . 4,5-epoxy-2-alkenals can react with phenylalanine and cause strecker-type degradation of amino acids. Autoxidized methyl linoleate can decrease DNA synthesis in thymocytes Animals consuming oxidized lipids suffered a wide array of biological consequences, such as decreased feed utilization and performance, oxidative stress and tissue lipid oxidation and, most strikingly, adverse effects on redox indices and shelf life of meat. This manifested in malondialdehyde (MDA) content reduced activities of antioxidant enzymes and elevated transcript levels of oxidative stress-responsive genes
The intestinal mucosa is directly exposed to oxidized fatty acids of dietary origin and this tissue readily experiences redox imbalances and oxidative stress after the ingestion of large amounts of oxidized fat . As the first line of defense, the intestines with abundant gut-associated lymphoid tissues (GALTs) and lymphocytes play an important role in immune defense. The immune response in the intestinal tract is complex and is impaired by any damage to the mucosal barrier. When oxidative stress of the intestines caused by oxidized fat occurs, its immune competence and responsiveness may be compromised by the peroxides they contain
When body insulin levels are low, fatty acids flow from the fat cells into the bloodstream and are taken up by various cells and metabolised in a process called beta-oxidation. The end result of beta-oxidation is a molecule called acetyl-coA, and as more fatty acids are released and metabolised, acetyl-coA levels in the cells rise. Liver cells shunt excess acetyl-coA into "ketogenesis", or the making of ketone bodies. When the rate of synthesis of ketone bodies exceeds the rate of utilisation, their concentration in blood increases; this is known as ketonaemia. This is followed by ketonuria – excretion of ketone bodies in urine. The overall picture of ketonaemia and ketonuria is commonly referred as ketosis. Smell of acetone in breath is a common feature in ketosis
For polyunsaturated fatty acids and oils (triglycerides), products of heating and recycling.*
Culinary oils, when heated, undergo important chemical reaction involving self-sustaining, free radical-mediated oxidative deterioration of polyunsaturated fatty acids (PUFAs). Such by-products may be cytotoxic, mutagenic, reproductive toxins and may produce chronic disease.
Saturated fatty acid (SFA)-rich fats also undergo such reactions but to a substantially lower degree.
Samples of repeatedly used oils collected from fast-food retail outlets and restaurants have confirmed the production of aldehydic lipid oxidation products (LOPs, active aldehydes) 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.
Teratogenic actions. In principle, if aldehydic LOPs induce DNA and chromosomal damage during embryo development, foetal malformations may arise. A study was conducted to investigate the ability of the chain-breaking antioxidant a-tocopherol (a-TOH, vitamin E) to prevent the teratogenic effects of uncontrolled diabetes mellitus in rats (a study based on the hypothesis that diabetic animals have an elevated level of oxidative stress and therefore in vivo lipid peroxidation when expressed relative to that of healthy controls). It found that a PUFA-rich culinary oil (which served as a vehicle for oral administration of a-TOH) increased the rate of malformations and reabsorptions in both normal and diabetic pregnancies. Further investigations revealed that safflower oil subjected to thermal stressing episodes (according to standard frying practices for a period of 20 minutes) markedly enhanced its teratogenic effects. That is, the evidence indicates that the LOPs therein are primarily responsible for these actions.
Further adverse health effects of dietary LOPs. Further documented health effects of LOPs include their pro-inflammatory and gastropathic properties (for the latter, oral administration of the LOP, 4-hydroxy-trans-2-nonenal -HNE- to rats at a dose level of only 0.26 umol·dm-3, a level similar to that of healthy human blood plasma, induced peptic ulcers), and also a significant elevation in systolic blood pressure and an impaired vasorelaxation observed in rats fed pre-heated soy oil
Oxidative degradation process involving culinary oils, can generate extremely toxic conjugated lipid hydroperoxydienes (CHPDs). These are unstable at standard frying temperatures (ca. 180 degrees C) and are degraded to a broad range of secondary products, particularly saturated and unsaturated aldehydes, together with di- and epoxyaldehydes. Such aldehydic fragments also have toxicological properties in humans owing to their high reactivity with critical biomolecules in vivo (proteins such as low-density lipoprotein, amino acids, thiols such as glutathione, DNA, etc.). Despite their reactivities, high levels of CHPDs can remain in PUFA-rich oils which have been subjected to routine frying practices.
Thermally stressed PUFA-containing culinary oils contain high levels of alpha,beta-unsaturated aldehydes (including trans-2-alkenals, and cis,trans- and trans,trans-alka-2,4-dienals, the latter including the mutagen trans,trans-2,4-decadienal), and n-alkanals, together with their CHPD and hydroxydiene precursors .
Toxicological and pathogenic properties of dietary LOPS
Potential influence of dietary LOPS on metabolic pathways. As a consequence of their absorption from the gut into the systemic circulation, LOPs may penetrate cellular membranes, allowing their entry into particular intracellular sites/organelles where many critical metabolic processes occur. Literature evidence indicates that feeding thermally stressed or repeatedly used culinary oils to experimental animals induces significant modifications to key liver microsomal pathways and to the mitochondrial respiratory chain, for example. These effects are likely to occur via reactions of LOPs with key enzymes (and more especially their active sites), for example, the oxidation of active methioninyl and cysteinyl residues by CHPDs, or alteration of critical side-chain amino acid amine or thiol groups with aldehydes via Schiff base or Michael addition reactions.
Atherosclerosis. Investigations have revealed that dietary derived LOPs can accelerate all three stages of the development of atherosclerosis (i.e., endothelial injury, accumulation of plaque, and thrombosis). Animal studies have shown that diets containing thermally stressed, PUFA-laden (and hence LOP-rich) oils exhibit a greater atherogenicity than those containing unheated ones . Because cytotoxic aldehydes can be absorbed, they have the capacity to attack and structurally alter the apolipoprotein B component of low density lipoproteins (LDLs). This mechanism can engender uptake of lipid-loaded LDLs by macrophages, which, in turn, transforms them to foam cells, the accumulation of which is responsible for the development of aortic fatty streaks, a hallmark of the aetiology of atherosclerosis and its pathological sequelae. More recently, our co-investigators found that aldehydic LOPs elevated the expression of the CD36 scavenger receptor of macrophages, a phenomenon that also promotes this process .

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	Mutagenic and carcinogenic properties. Since they are powerful electrophilic alkylating agents, alpha,beta-unsaturated aldehydes can covalently modify DNA base units via a mechanistically complex process that may involve their prior epoxidation in vivo.Such chemically altered bases may therefore be of mutagenic potential. Additionally, these LOPs can inactivate DNA replicating systems, a process that can, at least in principle, elevate the extent of DNA damage. Hence, following cellular uptake, such aldehydes have the potential to cause both DNA and chromosomal damage. Malondialdehyde (MDA) is also generated by thermally stressing culinary oils, although at concentrations much lower than those of the more reactive alpha,beta-unsaturated aldehydes. MDA and other aldehydes arising from lipid peroxidation (especially acrolein) present a serious carcinogenic hazard. Indeed, adenomas and carcinomas of the thyroid gland, together with adenomas of the pancreatic islet cells, were induced in rats by MDA in a prolonged gavage study; nasal and laryngeal cancers arose in rats and hamsters, respectively, during long-term acetaldehyde inhalation experiments. Hence, both these aldehydes satisfied the NIOSH criteria for classification as carcinogens, and therefore it has set exacting limits for their occupational exposure. The most obvious solution to the generation of LOPs in culinary oils during frying is to avoid consuming foods fried in PUFA-rich oils as much as possible. Indeed, consumers, together with those involved in the fast-food sector, could employ culinary oils of only a low PUFA content, or mono-unsaturated fatty acids (MUFA) such as canola (a variety of rape seed oil), olive oil, (both oils are rich in oleic acid) selected palm oils (rich in palmitic acid), or coconut oils (an SFA alternative rich in lauric and myristic acids) - for frying MUFAs such as oleoylglycerol adducts are much more resistant to peroxidative degradation than are PUFAs , and hence markedly lower levels of only selected classes of aldehydes are generated during frying. Previous studies that investigated the prospective health effects or benefits of dietary PUFAs (i.e., those involving feeding trials with humans or animals or, alternatively, related epidemiological ones) should be scrutinized. With hindsight, it seems to us that many of these experimental investigations were flawed since, in addition to some major design faults, they failed to take into account or even consider the nature and concentrations of any cytotoxic LOPs present in the oils or diets involved. Similarly, corresponding epidemiological (or meta-analysis-based) investigations incorporated only the (estimated) total dietary intake of selected PUFAs and further fatty acids, and ignored any LOPs derived or derivable from frying/cooking. Even if PUFA containing culinary oils are unheated, it is virtually impossible to rule out the presence of traces of LOPs within them (analysis of apparently pure PUFAs or their corresponding triglycerides obtained from reputable commercial sources has revealed that these materials contain traces of CHPDs and/or aldehydes As expected, the levels of total aldehydes generated increase proportionately with oil PUFA content, and over half are the more highly cytotoxic alpha,beta-unsaturated classes, which include acrolein and 4-hydroxy-trans-2-nonenal (HNE), as well as 4-hydroperoxy-, 4-hydroxy-, and 4,5-epoxy-trans-2-alkenals. Total alpha,beta-unsaturated aldehyde concentrations in culinary oils (heated at 180 deg C for 30-90 minutes or longer) are often higher than 20 mmol/kg and can sometimes approach 50 mmol/kg. Furthermore, relatively low concentrations of detectable aldehydes and their CHPD precursors are even found in newly purchased unheated culinary oils. Acrylamide (which can exert toxic effects on the nervous system and fertility, and may also be carcinogenic) can also arise from an acrolein source when asparagine-rich foods are deep-fried in PUFA-rich oils. The levels of acrylamide generated in foods during high-temperature cooking/frying processes are substantially lower than those recorded for aldehydes formed in PUFA-rich culinary oils during frying episodes (to date, the very highest reported levels are only ca. 4 ppm, equivalent to 56 umol/kg). Acrolein is just one of the alpha,beta-unsaturated aldehydes generated in thermally stressed PUFA-rich oils: Many others generated in this manner have comparable toxicological properties The foregoing considerations exclude possible toxicological properties of their isomeric CHPD precursors (also present in the high millimolar range in thermally stressed oils) in a typical fried food meal. Indeed, in one early investigation, a single intravenous dose of methyl linoleate hydroperoxide (20 mg/kg) administered to rats gave rise to a high mortality within 24 hours (animals dying from lung damage), although a higher dose given orally was without effect. This observation may reflect the limited in vivo absorption of these particular aldehyde precursors, in contrast to the known absorption of aldehydes. Furthermore, with regard to the risk of inhalation of aldehydes volatilised during frying practices by humans, the maximum US Occupational Safety and Health (OSHA) permissible exposure limit (PEL) for acrolein, which is an (atmospheric) level of 0.1 ppm (equivalent to only 1.8 umol/kg in the fried food model) for a time-weighted long-term (8 hour) exposure, and 0.3 ppm (5.4 umol/kg)for a short-term (15 minute) one. This 15-minute exposure time can be considered to be less than the time taken to consume a typical fried meal The concentrations of aldehydes generated in culinary oils during episodes of heating at 180 deg C represent only what remains in the oil: Owing to their low boiling points, many of the aldehydes generated are volatilized at standard frying temperatures. These represent inhalation health hazards, in view of their inhalation by humans, especially workers in inadequately ventilated fast-food retail outlets. The composition and content of hazardous LOPs available in fried foods depend on the identity of the frying/cooking oil and its PUFA content, the frying conditions employed, the length of the frying process, exposure of the frying medium to atmospheric oxygen, the reactivities of these agents with a range of other biomolecules (e.g., amino acids and proteins), and, to a limited extent, the antioxidant content of the frying matrix. Experiments have shown that shallow frying gives rise to much higher levels of LOPs than deep frying under the same conditions (reflecting the influence of the surface area of the frying medium, its exposure to atmospheric oxygen, and the subsequent dilution of LOPs generated into the bulk medium). In vivo absorption of dietary LOPs Except for direct damage to the gastrointestinal epithelium, the toxicological actions exerted by LOPs depend on their rate and extent of absorption from the gut into the systemic circulation where they may cause damage to essential organs, tissues, and cells. Experiments in rats have demonstrated that trans-2-alkenals, which are generated in PUFA-containing culinary oils during thermal stressing episodes, are absorbed . Following absorption, these cytotoxic agents are metabolized by a process involving the primary addition (Michael addition reaction) of glutathione across their electrophilic carbon-carbon double bonds and finally excreted in the urine as C-3 mercapturate derivatives. * Martin Grootveld, Victor Ruiz Rodado, and Christopher J.L. Silwood Detection, monitoring, and deleterious health effects of lipid oxidation products generated in culinary oils during thermal stressing episodes American Oil Chemists Society, 25 (10), pp. 614-624. November/December 2014 The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
RICINOLEIC ACID, HOMOPOLYMER	for ricinoleic acid Tumours at sites of application recorded. Equivocal tumourigen by RTECS criteria.
4,4'-METHYLENE BIS(DIBUTYLDITHIOCARBAMATE)	Repeated Dose/ Reproductive/ Developmental Effects: A repeat dose toxicity study with screening reproductive and developmental endpoints (OECD 422) has been conducted with the test substance. The parental NOAEL was 1000 ppm; the NOAEL for F1 offspring was greater than 20000 ppm. Genotoxicity: An salmonella/mammalian-microsome plate incorporation mutagenicity assay has been conducted with carbamodithioic acid, dibutyl-,methylene ester. The results of the bacterial mutagenicity test were negative HPV Challenge Program
OLEOYLSARCOSINE	The amino acids alkyl amides most likely dissociate into amino acids and fatty acids in the presence of water. Because most of these amino acids and fatty acids are found in the foods we consume daily, oral toxicity is not expected. In turn, dermal toxicity would not be expected to be different from oral exposure. Data from the previous safety assessments on alpha-amino acids and fatty acids support that these ingredients would not likely be irritants or sensitisers. No irritation was observed in in vitro studies with disodium capryloyl glutamate.Acetyl proline was a mild irritant in another in vitro study. In human studies, acetyl proline, acetyl tyrosinamide, disodium capryloyl glutamate, sodium cocoyl glutamate, and sodium lauroyl glutamate were not dermal irritants. No ocular irritation was observed in in vitro studies of acetyl tyrosinamide, disodium capryloyl glutamate, and sodium lauroyl glutamate. No adverse effects were observed during in-use studies of acetyl hydroxyproline and acetyl tyrosinamide in human subject. Severe irritation was observed in 1 study of sodium cocoyl glutamate at 5%, but was not irritating in another study with an unknown concentration. Sodium cocoyl glutamate and glycinate (fatty acids, C8-14 -even numbered are generally classified as R41/ H318 - Causes severe eye damage - by their suppliers, in spite of contrasting evidence. No sensitisation was observed in human studies with acetyl hydroxyproline, acetyl proline, acetyl tyrosinamide, disodium capryloyl glutamate, sodium cocoyl glutamate, and sodium lauroyl glutamate. Acetyl tyrosinamide, sodium cocoyl glutamate and sodium lauroyl glutamate were not phototoxic in human studies

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	In in vitro studies, acetyl glutamic acid, acetyl proline, acetyl tyrosinamide, disodium capryloyl glutamate, sodium cocoyl glutamate, and sodium lauroyl glutamate were negative for genotoxicity. Acetyl glutamic acid was negative in an in vivo study. The analogue chemicals, acyl sarcosines. raised concern about the possible formation of potentially carcinogenic nitrosated derivatives. For the analogue, the reactive material is likely to be the precursor sarcosine. This chemical varies in that the precursor amine glycine is a primary amine, whereas the precursor amine sarcosine in the analogue material is a secondary amine. Secondary amines are of more concern for nitrosamine formation than primary or tertiary amines. Whereas the nitrogen in chemicals fatty acid glycinates and glutamates is secondary, its functional group is an amide rather than an amine and has different chemical properties. Free amine is not present therefore the possibility of nitrosamine formation is considered to be low. For sarcosine: Motor impairment and respiratory issues have been observed in rats at 10mg/kg. This equates to 800 mg for an 80 kg person. Sarcosine was reported to activate prostate cancer cells and to indicate the malignancy of prostate cancer cells when measured in urine Sarcosine was identified as a differential metabolite that was greatly increased during prostate cancer progression to metastasis and could be detected in urine. Sarcosine levels seemed to control the invasiveness of the cancer. This conclusion has been disputed Sarcosine has been investigated in relation to schizophrenia. Early evidence suggests that intake of 2 g/day sarcosine as add-on therapy to certain antipsychotics (not clozapine) in schizophrenia, sarcosine gives significant additional reductions in both positive and negative symptomatology as well as the neurocognitive and general psychopathological symptoms that are common to the illness. Sarcosine had been tolerated well. It is also under investigation for the possible prevention of schizophrenic illness during the prodromal stage of the disease. It acts as a type 1 glycine transporter inhibitor and a glycine agonist. It increases glycine concentrations in the brain thus causing increased NMDA receptor activation and a reduction in symptoms. A 2011 meta-analysis found adjunctive sarcosine to have a medium effect size for negative and total symptoms. Major depressive disorder is a complex disease and most currently available antidepressants aiming at monoamine neurotransmission exhibit limited efficacy and cognitive effects. N-methyl-D-aspartate receptors (NMDARs), one subtype of glutamate receptor, play an important role in learning and memory, and NMDAR enhancing agents, such as sarcosine (N-methylglycine), have been used as adjunctive therapy of schizophrenia. Preliminary clinic trials indicated that intake of sarcosine improved not only psychotic but also depressive symptoms in patients with schizophrenia, and so may also be a useful supplement for treating depressive type schizoaffective disorders where rapid-acting glutamatergic antidepressants, particularly NMDA antagonists such as esketamine, can promote worsening of psychotic features. Toxicological data are available and well documented for representative toluenesulfonates, xylenesulfonates and cumenesulfonates (including sodium, potassium, ammonium and calcium salts). These data demonstrate that hydrotropes have a low order of acute toxicity by all relevant routes (LC50s range from 100s to 1000s mg/kg), are not genotoxic *in vitro* or *in vivo*, show no evidence of a carcinogenic response (or any other systemic toxicity) in 2-year dermal exposure studies, and failed to induce developmental, teratogenic or fertility (sex organ) effects. Adverse effects after repeated long term dosing of hydrotropes to animals included epidermal hyperplasia at the site of application in dermal studies, and decreased relative spleen weight in females in oral studies. The critical adverse effect and corresponding systemic NOAEL is 763 mg a.i./kg bw based upon decreased relative spleen weight in female rats in a 90-day oral study. The NOAEL for local effects, based on epidermal hyperplasia at the site of application, was 440 mg a.i./kg bw for mice in 90-day dermal studies. Hydrotropes can be classified as a negligible-to-slight irritant to skin and a slight-to-moderate irritant to eyes. The irritation potential of aqueous solutions of hydrotropes depends on concentration, and the irritation is lessened with rinsing. Hydrotropes are not considered to be skin sensitisers. HERA Report (Hydrotropes) September 2005 Hydrotropes in this category were assessed for mutagen/ genotoxic potential in a variety of assays including the mouse micronucleus, Ames, mouse lymphoma, sister chromatid exchange and chromosome aberration assays. No positive results were seen in vitro or in vivo in any of the studies. For both mice and rats exposed dermally for two years, there was no evidence of carcinogenic potential. Examination of the sex organs (such as prostate, testes or ovaries) from animals in 90-day feeding studies and 90-day and two year dermal studies yielded no evidence to suggest that these chemicals have an adverse affect on the reproductive organs.
SUNFLOWER OIL & RICINOLEIC ACID, HOMOPOLYMER & OLEOYLSARCOSINE	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.
SUNFLOWER OIL & RICINOLEIC ACID, HOMOPOLYMER	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

Stihl Multioil Bio	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available
sunflower oil	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	>0.01mg/L	2
	EC50(ECx)	72h	Algae or other aquatic plants	>0.01mg/L	2
	ErC50	72h	Algae or other aquatic plants	>0.01mg/L	2
ricinoleic acid, homopolymer	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available

Continued...

4,4'-methylene bis(dibutyldithiocarbamate)	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	>0.033mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	0.033mg/l	2
	EC50	48h	Crustacea	>0.052mg/l	2
	LC50	96h	Fish	>0.06mg/l	2
oleoylsarcosine	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	NOEC(ECx)	504h	Crustacea	0.102mg/L	2
	EC50	48h	Crustacea	0.43mg/l	2
	LC50	96h	Fish	>0.43mg/l	2
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*				

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
oleoylsarcosine	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
sunflower oil	LOW (LogKOW = 22.86)
4,4'-methylene bis(dibutyldithiocarbamate)	HIGH (LogKOW = 6.73)
oleoylsarcosine	HIGH (LogKOW = 6.83)

Mobility in soil

Ingredient	Mobility
oleoylsarcosine	LOW (Log KOC = 17090)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ **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 or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.

Ensure that the hazardous substance is disposed in accordance with the Hazardous Substances (Disposal) Notice 2017

Disposal Requirements

Packages that have been in direct contact with the hazardous substance must be only disposed if the hazardous substance was appropriately removed and cleaned out from the package. The package must be disposed according to the manufacturer's directions taking into account the material it is made of. Packages which hazardous content have been appropriately treated and removed may be recycled.
The hazardous substance must only be disposed if it has been treated by a method that changed the characteristics or composition of the substance and it is no longer hazardous.
Only dispose to the environment if a tolerable exposure limit has been set for the substance.
Only deposit the hazardous substance into or onto a landfill or sewage facility or incinerator, where the hazardous substance can be handled and treated appropriately.

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (ADG): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Land transport (UN): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Air transport (ICAO-IATA / DGR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Sea transport (IMDG-Code / GGVSee): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

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

Not Applicable

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

Continued...

Product name	Group
sunflower oil	Not Available
ricinoleic acid, homopolymer	Not Available
4,4'-methylene bis(dibutyldithiocarbamate)	Not Available
oleoylsarcosine	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
sunflower oil	Not Available
ricinoleic acid, homopolymer	Not Available
4,4'-methylene bis(dibutyldithiocarbamate)	Not Available
oleoylsarcosine	Not Available

SECTION 15 Regulatory information

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

This substance is to be managed using the conditions specified in an applicable Group Standard

HSR Number	Group Standard
HSR002521	Animal Nutritional and Animal Care Products Group Standard 2020
HSR002530	Cleaning Products Subsidiary Hazard Group Standard 2020
HSR002535	Gases under Pressure Mixtures Subsidiary Hazard Group Standard 2020
HSR002503	Additives Process Chemicals and Raw Materials Subsidiary Hazard Group Standard 2020
HSR002606	Lubricants Lubricant Additives Coolants and Anti freeze Agents Subsidiary Hazard Group Standard 2020
HSR002612	Metal Industry Products Subsidiary Hazard Group Standard 2020
HSR002624	N.O.S. Subsidiary Hazard Group Standard 2020
HSR002638	Photographic Chemicals Subsidiary Hazard Group Standard 2020
HSR002644	Polymers Subsidiary Hazard Group Standard 2020
HSR002647	Reagent Kits Group Standard 2020
HSR002648	Refining Catalysts Group Standard 2020
HSR002653	Solvents Subsidiary Hazard Group Standard 2020
HSR002670	Surface Coatings and Colourants Subsidiary Hazard Group Standard 2020
HSR002684	Water Treatment Chemicals Subsidiary Hazard Group Standard 2020
HSR100425	Pharmaceutical Active Ingredients Group Standard 2020
HSR002600	Leather and Textile Products Subsidiary Hazard Group Standard 2020
HSR002605	Lubricants Low Hazard Group Standard 2020
HSR002544	Construction Products Subsidiary Hazard Group Standard 2020
HSR002549	Corrosion Inhibitors Subsidiary Hazard Group Standard 2020
HSR002552	Cosmetic Products Group Standard 2020
HSR002558	Dental Products Subsidiary Hazard Group Standard 2020
HSR002565	Embalming Products Subsidiary Hazard Group Standard 2020
HSR002571	Fertilisers Subsidiary Hazard Group Standard 2020
HSR002573	Fire Fighting Chemicals Group Standard 2021
HSR002578	Food Additives and Fragrance Materials Subsidiary Hazard Group Standard 2020
HSR002585	Fuel Additives Subsidiary Hazard Group Standard 2020
HSR002596	Laboratory Chemicals and Reagent Kits Group Standard 2020
HSR100580	Tattoo and Permanent Makeup Substances Group Standard 2020
HSR100757	Veterinary Medicines Limited Pack Size Finished Dose Group Standard 2020
HSR100758	Veterinary Medicines Non dispersive Closed System Application Group Standard 2020
HSR100759	Veterinary Medicines Non dispersive Open System Application Group Standard 2020

Please refer to Section 8 of the SDS for any applicable tolerable exposure limit or Section 12 for environmental exposure limit.

sunflower oil is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
New Zealand Inventory of Chemicals (NZIoC)

ricinoleic acid, homopolymer is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
New Zealand Inventory of Chemicals (NZIoC)

4,4'-methylene bis(dibutyldithiocarbamate) is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
New Zealand Inventory of Chemicals (NZIoC)

oleoylsarcosine is found on the following regulatory lists

Continued...

Australian Inventory of Industrial Chemicals (AIIC)
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Land Transport Rule: Dangerous Goods 2005 - Schedule 1 Quantity limits for dangerous goods

Additional Regulatory Information

Not Applicable

Hazardous Substance Location

Subject to the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Hazard Class	Quantities
Not Applicable	Not Applicable

Certified Handler

Subject to Part 4 of the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Class of substance	Quantities
Not Applicable	Not Applicable

Refer Group Standards for further information

Maximum quantities of certain hazardous substances permitted on passenger service vehicles

Subject to Regulation 13.14 of the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Hazard Class	Gas (aggregate water capacity in mL)	Liquid (L)	Solid (kg)	Maximum quantity per package for each classification
Not Applicable	Not Applicable	Not Applicable	Not Applicable	Not Applicable

Tracking Requirements

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (sunflower oil; ricinoleic acid, homopolymer; 4,4'-methylene bis(dibutyldithiocarbamate); oleoylsarcosine)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (ricinoleic acid, homopolymer)
Japan - ENCS	No (sunflower oil)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory 'Active'
Taiwan - TCSI	Yes
Mexico - INSQ	No (sunflower oil; ricinoleic acid, homopolymer; 4,4'-methylene bis(dibutyldithiocarbamate))
Vietnam - NCI	Yes
Russia - FBEPH	No (oleoylsarcosine)
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	03/12/2024
Initial Date	16/05/2024

Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the BWES 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

Continued...

- 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
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration
- MARPOL: International Convention for the Prevention of Pollution from Ships
- IMSBC: International Maritime Solid Bulk Cargoes Code
- IGC: International Gas Carrier Code
- IBC: International Bulk Chemical Code

- 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