

Viper WRL Eco Rope Guardian ERG-1

Viper WRL Pty Ltd

Chemwatch Hazard Alert Code: 2

Chemwatch: 85-9136

Version No: 4.1

Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Issue Date: 23/12/2022

Print Date: 28/11/2024

L.GHS.AUS.EN.E

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

Product Identifier

Product name	Viper WRL Eco Rope Guardian ERG-1
Chemical Name	Not Applicable
Synonyms	Not Available
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	Lubricant; consumer use. Use according to manufacturer's directions.
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Details of the manufacturer or supplier of the safety data sheet

Registered company name	Viper WRL Pty Ltd
Address	c/o Unit 2, 14 Stoddart Road Prospect NSW 2148 Australia
Telephone	+61 (0)2 9636 5655
Fax	+61 (0)2 9636 8566
Website	Not Available
Email	sales@viperwrl.com

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone number(s)	Not Available
Other emergency telephone number(s)	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

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

Chemwatch Hazard Ratings

	Min	Max	
Flammability	1	1	
Toxicity	1	1	
Body Contact	2	2	
Reactivity	1	1	
Chronic	0	0	

0 = Minimum
1 = Low
2 = Moderate
3 = High
4 = Extreme

Poisons Schedule	Not Applicable
Classification ^[1]	Serious Eye Damage/Eye Irritation Category 2B
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	Not Applicable
Signal word	Warning

Hazard statement(s)

H320	Causes eye irritation.
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Precautionary statement(s) Prevention

P264	Wash all exposed external body areas thoroughly after handling.
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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.
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P337+P313	If eye irritation persists: Get medical advice/attention.
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Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

Not Applicable

SECTION 3 Composition / information on ingredients**Substances**

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
120962-03-0	20-40	<u>canola oil</u>
8001-79-4	10-30	<u>castor oil</u>
129828-25-7	10-20	<u>canola oil, polymerised, oxidised</u>
54326-11-3	1-10	<u>aluminium hydroxide benzoate stearate</u>
68037-01-4	1-5	<u>1-decene homopolymer, hydrogenated</u>
9003-28-5	1-5	<u>1-butene homopolymer</u>
9011-14-7	<2	<u>methyl methacrylate homopolymer</u>
1327-43-1	<1	<u>magnesium aluminosilicate</u>
Legend:		1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available

SECTION 4 First aid measures**Description of first aid measures**

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ 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	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▶ Other measures are usually unnecessary.
Ingestion	<ul style="list-style-type: none"> ▶ 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

Treat symptomatically.

SECTION 5 Firefighting measures**Extinguishing media**

- ▶ Water spray or fog.
- ▶ Alcohol stable foam.
- ▶ Dry chemical powder.
- ▶ Carbon dioxide.

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
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ 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 courses. ▶ Use water delivered as a fine spray to control fire and cool adjacent area.
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	<ul style="list-style-type: none"> ▶ 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. ▶ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ 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. <p>Combustion products include: carbon dioxide (CO₂) acrolein nitrogen oxides (NO_x) other pyrolysis products typical of burning organic material. May emit poisonous fumes.</p>
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures**Personal precautions, protective equipment and emergency procedures**

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	<p>Slippery when spilt.</p> <ul style="list-style-type: none"> ▶ Clean up all spills immediately. ▶ Avoid contact with skin and eyes. ▶ Wear impervious gloves and safety goggles. ▶ Trowel up/scrape up. ▶ Place spilled material in clean, dry, sealed container. ▶ Flush spill area with water.
Major Spills	<ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Stop leak if safe to do so. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Neutralise/decontaminate residue (see Section 13 for specific agent). ▶ Collect solid residues and seal in labelled drums for disposal. ▶ Wash area and prevent runoff into drains. ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▶ If contamination of drains or waterways occurs, advise emergency services. <p>Slippery when spilt.</p>

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

SECTION 7 Handling and storage**Precautions for safe handling**

Safe handling	<p>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</p> <p>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.</p> <ul style="list-style-type: none"> ▶ 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. ▶ DO NOT allow material to contact humans, exposed food or food utensils. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	<ul style="list-style-type: none"> ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ 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	<ul style="list-style-type: none"> ▶ Glass container is suitable for laboratory quantities ▶ DO NOT use aluminium or galvanised containers ▶ Metal can or drum
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	<ul style="list-style-type: none"> ▶ Packaging as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▶ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection**Control parameters****Occupational Exposure Limits (OEL)****INGREDIENT DATA**

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	aluminium hydroxide benzoate stearate	Stearates	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.

Ingredient	Original IDLH	Revised IDLH
canola oil	Not Available	Not Available
castor oil	Not Available	Not Available
canola oil, polymerised, oxidised	Not Available	Not Available
aluminium hydroxide benzoate stearate	Not Available	Not Available
1-decene homopolymer, hydrogenated	Not Available	Not Available
1-butene homopolymer	Not Available	Not Available
methyl methacrylate homopolymer	Not Available	Not Available
magnesium aluminosilicate	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
canola oil	E	≤ 0.1 ppm
castor oil	E	≤ 0.1 ppm
methyl methacrylate homopolymer	E	≤ 0.01 mg/m ³
Notes:	<i>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.</i>	

MATERIAL DATA

for poly-alpha-olefins (PAOs)
 CEL TWA: 5 mg/m3 (mist); STEL 10 mg/m3 (cf TLV TWA mineral oils, pure, highly and severely refined; inhalable fraction)
 (CEL = Chemwatch Exposure Limit)


The TLV is based on the exposures to aluminium chloride and the amount of hydrolysed acid and the corresponding acid TLV to provide the same degree of freedom from irritation. Workers chronically exposed to aluminium dusts and fumes have developed severe pulmonary reactions including fibrosis, emphysema and pneumothorax. A much rarer encephalopathy has also been described.

Exposure controls

Appropriate engineering controls	<p>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:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>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.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>General exhaust is adequate under normal operating conditions. Local exhaust ventilation may be required in specific circumstances. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.</p>																		
	<table border="1"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>solvent, vapours, degreasing etc., evaporating from tank (in still air).</td> <td>0.25-0.5 m/s (50-100 f/min)</td> </tr> <tr> <td>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)</td> <td>0.5-1 m/s (100-200 f/min.)</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td> <td>2.5-10 m/s (500-2000 f/min.)</td> </tr> </tbody> </table> <p>Within each range the appropriate value depends on:</p> <table border="1"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> </tbody> </table>	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.)	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
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	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	<ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber NOTE: <ul style="list-style-type: none"> ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. ▶ Neoprene rubber gloves 	
Body protection	See Other protection below	
Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ P.V.C apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash unit. 	

Recommended material(s)**GLOVE SELECTION INDEX**

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

The effect(s) of the following substance(s) are taken into account in the **computer-generated** selection:

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Material	CPI
NEOPRENE	A

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

C: Poor to Dangerous Choice for other than short term immersion

NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -

* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.

Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	A-AUS P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	-	A-AUS / Class 1 P2	-
up to 100 x ES	-	A-2 P2	A-PAPR-2 P2 ^

^ - Full-face

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

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

SECTION 9 Physical and chemical properties**Information on basic physical and chemical properties**

Appearance	Brown paste with a hydrocarbon-like odour; not miscible with water.		
Physical state	Non Slump Paste	Relative density (Water = 1)	0.95
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available

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pH (as supplied)	6-8	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)	285	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	<1	VOC g/L	Not Available
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	Product is considered stable and 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	<p>Inhalation hazard is increased at higher temperatures.</p> <p>Not normally a hazard due to non-volatile nature of product</p> <p>Inhalation of oil droplets/ aerosols may cause discomfort and may produce chemical pneumonitis.</p> <p>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.</p> <p>Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs.</p> <p>Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p>
Ingestion	<p>Accidental ingestion of the material may be damaging to the health of the individual.</p> <p>JECFA established an acceptable daily intake (ADI) of 0-25 mg/kg bw for polyglyceryl esters of fatty acids having an average chain length of up to 3 glycerol units and an ADI of 0-7.5 mg/kg bw for polyglyceryl esters of interesterified ricinoleic acid.</p> <p>In the EU, the esters are listed as food additives at concentrations between 5000 and 10,000 mg/kg in certain foods, and up to 7% free glycerol/polyglycerol is allowed (i.e., 700 mg/kg).</p> <p>Ricinoleic acid, the major fatty acid present in castor oil, has a variety of effects on the gastrointestinal tract, including inhibition of water and electrolyte absorption (Donowitz, 1979), stimulation of water secretion into the intestinal lumen (Ammon and Phillips, 1974), and depression of small bowel contractile activity (Ammon et al., 1974). The cathartic action of orally ingested castor oil traditionally has been attributed to irritant or stimulatory effects of ricinoleic acid on the gastrointestinal smooth muscle; the ricinoleic acid is liberated in the small intestine by the action of pancreatic lipase (Stewart and Bass, 1976). Moreover, absorption of ricinoleic acid occurs incompletely; substantial quantities remain in the gastrointestinal tract after oral administration (Stewart and Bass, 1976). Since diet palatability was not affected by the presence of castor oil, the poor absorption of ricinoleic acid and its potential to reduce absorption of other fatty acids could be responsible for the absence of more substantial body weight gains by rats and mice consuming castor oil-containing diets.</p>
Skin Contact	<p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Limited evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p>
Eye	<p>Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals. Repeated or prolonged eye contact may cause inflammation (similar to windburn) characterised by a temporary redness of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p>
Chronic	<p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals.</p> <p>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.</p>

Viper WRL Eco Rope Guardian ERG-1	TOXICITY	IRRITATION
	Not Available	Not Available
canola oil	TOXICITY	IRRITATION
	Not Available	Not Available
castor oil	TOXICITY	IRRITATION
	Oral (Rat) LD50: >4800 mg/kg ^[1]	Eye (Rodent - rabbit): 500mg - Mild
		Skin (Human - man): 50mg/48H - Mild
		Skin (Rodent - guinea pig): 100mg/24H - Mild
		Skin (Rodent - rabbit): 100mg/24H - Severe
		Skin (Rodent - rat): 100mg/24H - Mild
canola oil, polymerised, oxidised	TOXICITY	IRRITATION
	Not Available	Not Available
aluminium hydroxide benzoate stearate	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >2000 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
1-decene homopolymer, hydrogenated	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation (Rat) LC50: 0.9 mg/l4h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >2000 mg/kg ^[1]	
1-butene homopolymer	TOXICITY	IRRITATION
	Not Available	Not Available
methyl methacrylate homopolymer	TOXICITY	IRRITATION
	Not Available	Not Available
magnesium aluminosilicate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[1]	Not Available
	Inhalation (Rat) LC50: >2.08 mg/l4h ^[1]	
	Oral (Rat) LD50: >2000 mg/kg ^[1]	

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

CASTOR OIL

Some tumorigenic effects have been reported in animal studies using castor oil

The castor seed contains ricin, a toxic protein. Heating during the oil extraction process denatures and inactivates the protein. However, harvesting castor beans may not be without risk. Allergenic compounds found on the plant surface can cause permanent nerve damage, making the harvest of castor beans a human health risk.

The United States Food and Drug Administration (FDA) has categorized castor oil as "generally recognized as safe and effective" (GRASE) for over-the-counter use as a laxative with its major site of action the small intestine where it is digested into ricinoleic acid. Despite castor oil being widely used to start labor in pregnant women, to date there is not enough research to show whether it is effective to ripen the cervix or induce labour

Due to its foul taste a heavy dose of castor oil was formerly used as a humiliating punishment for children and adults. Victims of this treatment did sometimes die, as the dehydrating effects of the oil-induced diarrhea; however, even those victims who survived had to bear the humiliation of the laxative effects resulting from excessive consumption of the oil.

Several instances of sensitization to castor oil in cosmetics have been reported, including an allergic reaction to a make-up remover and contact dermatitis caused by use of a lipstick containing castor oil. Hypersensitivity reactions such as angioedema, rhinitis, asthma, and scarlatiniform rashes have been reported in factory workers involved in the extraction of castor oil, or in association with ingesting it.

Relatively few studies of castor oil toxicity have been conducted with experimental animals, and no studies were located concerning its absorption, distribution, metabolism, or excretion. Subcutaneous injection of 0.1 ml of castor oil in adult C57Bl/6 mice, daily for 4 weeks, was associated with the presence of electron dense lipid inclusions in parenchymal cells of the zona fasciculata of the adrenal gland.

Gavage administration of 1 ml/kg to rhesus monkeys, daily for 4 days, caused mild morphological changes in the small intestine, characterized by lipid droplets along the mucosal epithelium and in the underlying lamina propria. This was considered a possible indication that castor oil had reduced lipid metabolism in the intestinal epithelium.

Because of widespread human exposure, large annual production and use, and the lack of studies characterizing the effect of exposures of moderate duration, the subchronic toxicity of castor oil was evaluated by administering diet formulations to F344/N rats and B6C3F1 mice for 13 weeks. Exposure to castor oil in the diet at concentrations up to 10% had no effect on survival of F344/N rats. No significant differences in average food consumption among each sex were observed, although food consumption of male and female rats receiving diets containing 10% castor oil was slightly lower than that of controls. Hematological effects of the castor oil diets among male rats included a slight decrease in MCHC at day 21 in those receiving the 10% diet; a statistically significant decrease in MCV among the 10% group; a decrease in MCH among the 5% and 10% groups; and an increase in platelets among the 1.25%, 5%, and 10% groups. The only change observed among female rats was a statistically significant decrease in reticulocyte counts at day 5 in groups receiving the 0.62% or 10% diets. None of these changes was considered biologically significant.

A treatment- and dose-related increase in the activity of serum alkaline phosphatase was observed in male and female rats at days 5 and 21, and at study termination. Total bile acids were increased among males receiving the higher dietary levels at days 5 and 21 but were not increased at study termination. Other minor changes included increases in albumin observed at study termination in males receiving 5% diets and at day 5 in females receiving 10% diets, and an increase in urea nitrogen at study termination in males that received 0.62% diets and a decrease at day 5 in females that received castor oil at 10% in the diet. Absolute liver weights and the liver-to-body-weight ratio were increased in male rats that received diets containing 10% castor oil. Heart-to-body-weight ratios were increased in groups of male rats

Continued...

receiving 0.62% 2.5%, and 10% diets; however, absolute heart weights were not increased, and the differences in body weight ratios were small and not considered treatment related.

Using light microscopy, it was determined there were no morphologic changes associated with the slight differences in organ weights between groups. In male rats, there was a slight decrease in epididymal weight (6-7%) which occurred in the middle- and high-dose groups, but this was not dose-related. There were no effects on any other male rat reproductive endpoint, or on any female rat reproductive endpoint. Although there was some variation in epididymal weights, their small magnitude and the absence of changes in other endpoints suggested that there was little or no evidence of any reproductive toxicity associated with castor oil exposure. Histopathologic examination revealed an absence of compound-related lesions in any organ or tissue of rats exposed to castor oil in the diet.

In genetic toxicity studies, castor oil (100-10,000 ug/plate) was not mutagenic in Salmonella typhimurium strains TA100, TA1535, TA97, or TA98 when tested with a preincubation protocol in the presence and the absence of exogenous metabolic activation (S9). Castor oil did not induce sister-chromatid exchanges or chromosome aberrations in Chinese hamster ovary cells treated with concentrations up to 5000 Oug/ml with and without S9. No induction of micronuclei was observed in peripheral blood erythrocytes of male and female B6C3F1 mice sampled at the termination of the 13-week study.

Castor oil was found not to be mutagenic or clastogenic in several in vitro genetic toxicity assays, and administration of diets containing up to 10% castor oil was not associated with toxicity to any specific organ, organ system, or tissue in this study

1-DECENE HOMOPOLYMER, HYDROGENATED

(estimated) * Evidence of conjunctival changes ** No evidence of tissue damage [Inland Vacuum Industries] ^ US EPA HPV Challenge program October 2002

For poly-alpha-olefins (PAOs):

PAOs are highly branched isoparaffinic chemicals produced by oligomerisation of 1-octene, 1-decene, and/or 1-dodecene. The crude polyalphaolefin mixture is then distilled into appropriate product fractions to meet specific viscosity specifications and hydrogenated.

Read across data exist for health effects endpoints from the following similar hydrogenated long chain branched alkanes derived from a C8, C10, and/or C12 alpha olefins:

- ▶ Decene homopolymer
- ▶ Decene/dodecene copolymer
- ▶ Octene/decene/dodecene copolymer
- ▶ Dodecene trimer

The data for these structural analogs demonstrated no evidence of health effects. In addition, there is evidence in the literature that alkanes with 30 or more carbon atoms are unlikely to be absorbed when administered orally. The physicochemical data suggest that it is unlikely that significant absorption will occur. If a substance of the size and structure of a typical PAO is absorbed, then the principal mechanisms of absorption after oral administration are likely to be passive diffusion and absorption by way of the lymphatic system. The former requires both good lipid solubility and good water solubility as the substance has to partition from an aqueous environment through a lipophilic membrane into another aqueous environment during absorption. Absorption by way of the lymphatics occurs by mechanisms analogous to those that absorb fatty acids and is limited by the size of the molecule. Lipophilicity generally enhances the ability of chemicals to cross biological membranes. Biotransformation by mixed function oxidases often increases the water solubility of a substance; however, existing data suggest that these substances will not undergo oxidation to more hydrophilic metabolites. Finally, a chemical must have an active functional group that can interact chemically or physically with the target cell or receptor upon reaching it; there are no moieties in PAOs that represent a functional group that may have biological activity. The water solubilities of a C10 dimer PAO and a C12 trimer PAO were determined to be <1 ppb and < 1 ppt respectively. The partition coefficient for a C12 trimer PAO was determined to be log Kow of >7. Given the very low water solubility it is extremely unlikely that PAOs will be absorbed by passive diffusion following oral administration, and the size of the molecules suggest that the extent of lymphatic absorption is likely to be very low. Although PAOs are relatively large lipophilic compounds, and molecular size may be a critical limiting determinant for absorption, there is some evidence that these substances are absorbed. However, the lack of observed toxicity in the studies with PAOs suggests that these products are absorbed poorly, if at all.

Furthermore, a review of the literature regarding the absorption and metabolism of long chain alkanes indicates that alkanes with 30+ carbon atoms are unlikely to be absorbed. For example the absorption of squalane, an analogous C30 product, administered orally to male CD rats was examined - essentially all of the squalane was recovered unchanged in the faeces. At the same time, the hydrophobic properties of PAOs suggest that, should they be absorbed, they would undergo limited distribution in the aqueous systemic circulation and reach potential target organs in limited concentrations.

In addition to the general considerations discussed above, the low volatility of PAOs indicates that, under normal conditions of use or transportation, exposure by the inhalation route is unlikely. In particular, the high viscosity of these substances suggests that it would be difficult to generate a high concentration of respirable particles in the air.

Acute toxicity: PAOs (decene/dodecene copolymer, octene/decene/dodecene homo-polymer, and dodecene trimer) have been adequately tested for acute oral toxicity. There were no deaths when the test materials were administered at doses of 5,000 mg/kg (decene/dodecene copolymer and dodecene trimer) and at 2,000 mg/kg (octene/decene/dodecene copolymer) in rats. Overall, the acute oral LD50 for these substances was greater than the 2000 mg/kg limit dose, indicating a relatively low order of toxicity.

PAOs (decene/dodecene copolymer, octene/decene/dodecene copolymer, and dodecene trimer) have been tested for acute dermal toxicity. No mortality was observed for any substance when administered at the limit dose of 2000 or 5000 mg/kg. Overall, the acute dermal LD50 for these substances was greater than the 2000 mg/kg limit dose, indicating a relatively low order of toxicity.

1-Decene, homopolymer, is absorbed (unexpectedly for a high molecular weight polymer) to a moderate degree in rat skin and is eliminated slowly

PAOs (decene homopolymer, decene/dodecene copolymer, and decene trimer) have been tested for acute inhalation toxicity. Rats were exposed to aerosols of the substances at nominal atmospheric concentrations of 2.5, 5.0, and 5.06 mg/L, respectively, for four hours. These levels were the maximum attainable concentrations under the conditions of the tests, due to the low volatility and high viscosity of the test material. No mortality was noted, and all animals fully recovered following depuration. The lack of mortality at concentrations at or above the limit dose of 2.0 mg/L indicates a relatively low order of toxicity for these substances.

Repeat dose toxicity: Eight repeated-dose toxicity studies using two different animal species, rats and mice, and oral and dermal routes of administration have been conducted with three structural analogs. These data suggest that the structural analogs exhibit a low order of toxicity following repeated applications, due to their similarity in chemical structures and physicochemical properties.

One 28-day oral toxicity study in rats, one 90-day dermal and two 90-day dietary studies in rats, and a dermal carcinogenicity study in mice exist for decene homopolymer. A rat oral combined reproductive toxicity and 91-day systemic toxicity study was also conducted with decene homopolymer. In addition, 28-day rat oral toxicity studies exist for two structurally analogous substances (dodecene trimer and octene/decene/dodecene copolymer); and a 90-day rat dermal toxicity study exists for octene/decene/dodecene copolymer. Results from these studies show a low order of repeated dose toxicity. The dermal NOAEL for systemic toxicity studies was equal to or greater than 2000 mg/kg/day.

The oral NOAEL for 1-decene homopolymer is between 5,000 and 20,000 mg/kg/day in Sprague-Dawley rats.

Rats exposed repeatedly by dermal exposure at doses of 2000 mg/kg decene/dodecene copolymer showed increased incidences of hyperplasia of the sebaceous glands, hyperplasia/hyperkeratosis of the epidermis and dermal inflammation. These symptoms generally subsided within 2 weeks. Males showed decreased body weight gain and altered serum chemistry.

In a 90-day feeding study rats receiving 20000 ppm of 1-decene, homopolymer, hydrogenated did not exhibit any clinical signs of systemic toxicity. Marginal effects on clinical chemistry (glucose and ALT in males; sodium, phosphorus and calcium in females) were seen.

Reproductive toxicity: Data are available for decene homopolymer. Results from these studies show a low order of reproductive/developmental toxicity. The NOAEL for reproductive toxicity was 1000 mg/kg/day, the highest concentration tested. The lack of effects on fertility in this study or effects on reproductive organs in this or other subchronic studies with closely related chemicals indicates that PAOs are unlikely to exert effects on reproduction.

Developmental toxicity: Decene homopolymer (with 10 ppm of an antioxidant) was administered once daily on gestation days 0-19 via dermal application to presumed-pregnant rats at doses of 0, 800, and 2000 mg/kg/day. Dermal administration of the test material did not adversely affect parameters of reproductive performance during gestation, nor did it adversely affect in utero survival and development of the offspring. The NOAEL in this study for developmental parameters was 2000 mg/kg/day.

Genotoxicity: Information for the following PAOs (decene homopolymer, octene/decene/dodecene copolymer, dodecene trimer; and decene/dodecene copolymer [prepared from 10% C12 and 90% C10 alpha olefins; approx. 33% trimer and 51% tetramer, 16% pentamer and higher]) is available. Either bacterial or mammalian gene mutation assays, in vitro chromosomal aberration assays, or in vivo chromosomal aberration assays have been conducted for these substances. Neither mutagenicity nor clastogenicity were exhibited by any of these substances in the referenced in vivo or in vitro tests, with or without metabolic activation.

	<p>Carcinogenicity: While alpha-olefin polymers have similar properties to mineral oils, they do not contain polycyclic aromatic hydrocarbons, or other known possible carcinogens.</p> <p>Decene homopolymer produced no treatment-related tumors in C3H mice treated with a 50 ul/application twice weekly for 104 weeks. In addition, survival (56%) was greater than in any other group, including the untreated control.</p>
<p>METHYL METHACRYLATE HOMOPOLYMER</p>	<p>Polymethyl methacrylate (PMMA) and related cosmetic ingredients methyl methacrylate crosspolymer and methyl methacrylate/glycol dimethacrylate crosspolymer are polymers that function as film formers and viscosity-increasing agents in cosmetics. The Food and Drug Administration (FDA) determination of safety of PMMA use in several medical devices, which included human and animal safety data, was used as the basis of safety of PMMA and related polymers in cosmetics by the Cosmetic Ingredient Review (CIR) Expert Panel. The PMMA used in cosmetics is substantially the same as in medical devices. The Panel concluded that these ingredients are safe as cosmetic ingredients in the practices of use and concentrations as described in this safety assessment J Toxicol. 2011 May;30(3 Suppl):54S-65S. doi: 10.1177/1091581811407352.</p> <p>After the polymerization process, there is the possibility of extra monomer being present within and on the final product. MMA is the residual monomer from polymerization of PMMA. MMA was found to be sensitizing at 25% in guinea pigs. The minimum induction concentration in a guinea pig maximization test was 1 M (1 g MW/L; 88,000 ppm). In a local lymph node assay, methyl methacrylate had an EC3 (stimulation index [SI] of 3 relative to concurrent vehicle treated controls) of 60% w/v in acetone and 90% w/v in olive oil. The author rated methyl methacrylate as a weak contact allergen.</p> <p>Sensitization data also were reviewed in the safety assessment of ethyl methacrylate used in the formulation of nail products. Ethyl methacrylate was found to be "...safe as used when application is accompanied by directions to avoid skin contact because of the sensitizing potential of ethyl methacrylate". The frequency of positive reactions among all patients tested with ethyl methacrylate was 14/22 (64%). The frequency of positive reactions among patients with artificial nails was 7/11 (64%), suggesting that use of artificial nails presented no additional risk. More to the point of considering the potential sensitization of the methyl methacrylate monomer, the frequency of positive reactions among all patients to methyl methacrylate was 7/22 (32%) and among patients with artificial nails was 1/10 (10%). Combining the low frequency of sensitization to methyl methacrylate with the low level of the monomer in PMMA, the risk of sensitization may be considered low. Cross- or co-reactivity of ethyl methacrylate and methyl methacrylate was another concern addressed in the safety assessment of ethyl methacrylate, specifically because of the use of methyl methacrylate in PMMA bone cements and the possibility that an individual sensitized to ethyl methacrylate might then have an allergic reaction to the bone cement in a necessary medical procedure. The Panel concluded that there were no data supporting any sensitization reactions in patients receiving implants cemented with methyl methacrylate and that adverse consequences of cross-reactivity of ethyl methacrylate and methyl methacrylate are not a concern</p> <p>The substance is classified by IARC as Group 3: NOT classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.</p>
<p>Viper WRL Eco Rope Guardian ERG-1 & CANOLA OIL & CASTOR OIL & CANOLA OIL, POLYMERISED, OXIDISED & ALUMINIUM HYDROXIDE BENZOATE STEARATE & 1-BUTENE HOMOPOLYMER & METHYL METHACRYLATE HOMOPOLYMER</p>	<p>No significant acute toxicological data identified in literature search.</p>
<p>Viper WRL Eco Rope Guardian ERG-1 & CANOLA OIL</p>	<p>Epoxidation of double bonds is a common bioactivation pathway for alkenes. The allylic epoxides, so formed, were found to possess sensitizing capacity in vivo and in vitro and to chemically react 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 prohaptenes, 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.</p> <p>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</p> <p>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 conducted 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"</p> <p>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 epididymides, increased peroxidation of membrane and tissue lipids and induction of cytochrome P450 activities in the colon and liver.</p> <p>The propensity for PUFAs to oxidise leads to the generation of free radicals and eventually to rancidity.</p> <p>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.</p> <p>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.</p> <p>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 lysosomes (releasing hydrolytic enzymes) and other subcellular organelles. They may also react with nucleic acids.</p> <p>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.</p> <p>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</p> <p>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.</p> <p>For polyunsaturated fatty acids and oils (triglycerides)</p> <p>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.</p>

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. Autooxidized 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 α -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 μ mol-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.

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

Polyunsaturated fats (PUFAs) protect against cardiovascular disease by providing more membrane fluidity than monounsaturated fats (MUFAs), but they are more vulnerable to lipid peroxidation (rancidity). On the other hand, some monounsaturated fatty acids (in the same way as saturated fats) may promote insulin resistance, whereas polyunsaturated fatty acids may be protective against insulin resistance. Furthermore, one the large scale study found that increasing monounsaturated fat and decreasing saturated fat intake could improve insulin sensitivity, but only when the overall fat intake of the diet was low. Studies have shown that substituting dietary monounsaturated fat for saturated fat is associated with increased daily physical activity and resting energy expenditure. More physical activity was associated with a higher-oleic acid diet (a MUFA) than one of a palmitic acid diet (saturated fat). From the study, it is shown that more monounsaturated fats lead to less anger and irritability.

Foods containing monounsaturated fats reduce low-density lipoprotein (LDL) cholesterol, while possibly increasing high-density lipoprotein (HDL) cholesterol. However, their true ability to raise HDL is still in debate.

Levels of oleic along with other monounsaturated fatty acids in red blood cell membranes were positively associated with breast cancer risk. The saturation index (SI) of the same membranes was inversely associated with breast cancer risk. Monounsaturated fats and low SI in erythrocyte membranes are predictors of postmenopausal breast cancer. Both of these variables depend on the activity of the enzyme delta-9 desaturase (delta-9-d).

In children, consumption of monounsaturated oils is associated with healthier serum lipid profiles.

The Mediterranean Diet is one heavily influenced by monounsaturated fats. People in Mediterranean countries consume more total fat than Northern European countries, but most of the fat is in the form of monounsaturated fatty acids from olive oil and omega-3 fatty acids (PUFAs) from fish, vegetables, and certain meats like lamb, while consumption of saturated fat is minimal in comparison. The diet in Crete is fairly high in total fat (40% of total calories, almost exclusively provided by olive oil - oleic acid) yet affords a remarkable protection from coronary heart disease (and probably colon cancer).

essential fatty acids are leukotriene B4 (LTB4) receptor antagonists, which may account in part for their reported anti-inflammatory activities. Generally speaking, fatty acids with two or more unsaturated sites and chain lengths of 18-22 were potent inhibitors of LTB4 binding to neutrophil membranes.

The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

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.

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For Group E aliphatic esters (polyol esters):

According to a classification scheme described by the American Chemistry Council' Aliphatic Esters Panel, Group E substances are esters of monoacids, mainly common fatty acids, and trihydroxy or polyhydroxyalcohols or polyols, such as pentaerythritol (PE), 2-ethyl-2-(hydroxymethyl)-1,3-propanediol or trimethylolpropane (TMP), and dipentaerythritol (diPE). The Group E substances often are referred to as "polyol esters" The polyol esters are unique in their chemical characteristics since they lack beta-tertiary hydrogen atoms, thus leading to stability against oxidation and elimination. The fatty acids often range from C5-C10 to as high as C18 (e.g., oleic, stearic, isostearic, tall oil fatty acids) in carbon number and generally are derived from naturally occurring sources. Group E esters may have multiple ester linkages and may include mixed esters derived from different carbon-length fatty acid mixtures. The lack of beta-tertiary hydrogen atoms in the structure of the polyol esters makes them characteristically and chemically stable against oxidation and elimination in comparison to other ester classes or groups. For these reasons, trimethylolpropane (TMP) and pentaerythritol (PE) esters with fatty acids of C5 to C10 carbon-chain length have applications as synthetic lubricants for passenger car motor oil and military and civilian jet engines. TMP and PE esters of C18 acids (e.g., isostearic and oleic acids) also have found use in synthetic lubricant applications, including refrigeration lubricants and hydraulic fluids. Because of their higher thermal stability characteristics, they also find use in a variety of high temperature applications such as industrial oven chain oils, high temperature greases, fire resistant transformer coolants and turbine engines

Polyol esters that are extensively esterified also have greater polarity, less volatility and enhanced lubricity characteristics. **Acute toxicity:** Depending on the degree of esterification, the polyol esters can be resistant or slow towards chemical or enzymatic hydrolysis (i.e., esterase or lipases) as a result of steric hindrance. PE and diPE esters that are capable of being enzymatically hydrolyzed will generate pentaerythritol or dipentaerythritol, and the corresponding fatty acids which, for most of the Group E esters, are comprised mainly of oleic, linoleic and stearic acids as well as the fatty acids in the C5-10 carbon-length. Similarly, TMP esters can undergo metabolism to yield trimethylolpropane (2-ethyl-2-hydroxymethyl-1,3-propanediol) and fatty acid constituents. Pentaerythritol and trimethylolpropane have been reported to have a low order of toxicity The acute oral LD50 for these substances was greater than 2000 mg/kg indicating a relatively low order of toxicity. The similarity in the low order of toxicity for these substances is consistent with their similar chemical structure and physicochemical properties.

Metabolic studies of polyglyceryl esters indicated that these esters are hydrolyzed in the gastrointestinal (GI) tract, and utilization and digestibility studies supported the assumption that the fatty acid moiety is metabolized in the normal manner. Analytical studies have

produced no evidence of accumulation of the polyglycerol moiety in body tissues.

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 at 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 pentaerythritol, 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 pentaerythritol, 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.

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Guardian ERG-1 & CANOLA
OIL & METHYL
METHACRYLATE
HOMOPOLYMER**

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.

CANOLA OIL & CASTOR OIL

For aliphatic fatty acids (and salts)

Acute oral (gavage) toxicity:

The acute oral LD50 values in rats for both were greater than >2000 mg/kg bw. Clinical signs were generally associated with poor condition following administration of high doses (salivation, diarrhoea, staining, piloerection and lethargy). There were no adverse effects on body weight in any study. In some studies, excess test substance and/or irritation in the gastrointestinal tract was observed at necropsy.

Skin and eye irritation potential, with a few stated exceptions, is chain length dependent and decreases with increasing chain length. According to several OECD test regimes the animal skin irritation studies indicate that the C6-10 aliphatic acids are severely irritating or corrosive, while the C12 aliphatic acid is irritating, and the C14-22 aliphatic acids generally are not irritating or mildly irritating.

Human skin irritation studies using more realistic exposures (30-minute, 1-hour or 24-hours) indicate that the aliphatic acids have sufficient, good or very good skin compatibility.

Animal eye irritation studies indicate that among the aliphatic acids, the C8-12 aliphatic acids are irritating to the eye while the C14-22 aliphatic acids are not irritating.

Eye irritation potential of the ammonium salts does not follow chain length dependence; the C18 ammonium salts are corrosive to the eyes.

Dermal absorption:

The in vitro penetration of C10, C12, C14, C16 and C18 fatty acids (as sodium salt solutions) through rat skin decreases with increasing chain length. At 86.73 ug C16/cm² and 91.84 ug C18/cm², about 0.23% and less than 0.1% of the C16 and C18 soap solutions is absorbed after 24 h exposure, respectively.

Sensitisation:

No sensitisation data were located.

Repeat dose toxicity:

Repeated dose oral (gavage or diet) exposure to aliphatic acids did not result in systemic toxicity with NOAELs greater than the limit dose of 1000 mg/kg bw.

Mutagenicity

Aliphatic acids do not appear to be mutagenic or clastogenic in vitro or in vivo

Carcinogenicity

No data were located for carcinogenicity of aliphatic fatty acids.

Reproductive toxicity

No effects on fertility or on reproductive organs, or developmental effects were observed in studies on aliphatic acids and the NOAELs correspond to the maximum dose tested. The weight of evidence supports the lack of reproductive and developmental toxicity potential of the aliphatic acids category.

Given the large number of substances in this category, their closely related chemical structure, expected trends in physical chemical properties, and similarity of toxicokinetic properties, both mammalian and aquatic endpoints were filled using read-across to the closest structural analogue, and selecting the most conservative supporting substance effect level.

Structure-activity relationships are not evident for the mammalian toxicity endpoints. That is, the low mammalian toxicity of this category of substances limits the ability to discern structural effects on biological activity. Regardless, the closest structural analogue with the most conservative effect value was selected for read across. Irritation is observed for chain lengths up to a cut-off at or near 12 carbons).

Metabolism:

The aliphatic acids share a common degradation pathway in which they are metabolized to acetyl-CoA or other key metabolites in all living systems. Common biological pathways result in structurally similar breakdown products, and are, together with the physico-chemical properties, responsible for similar environmental behavior and essentially identical hazard profiles with regard to human health.

Differences in metabolism or biodegradability of even and odd numbered carbon chain compounds or saturated/unsaturated compounds are not expected; even- and odd-numbered carbon chain compounds, and the saturated and unsaturated compounds are naturally occurring and are expected to be metabolized and biodegraded in the same manner.

The acid and alkali salt forms of the homologous aliphatic acid are expected to have many similar physicochemical and toxicological properties when they become bioavailable; therefore, data read across is used for those instances where data are available for the acid form but not the salt, and vice versa. In the gastrointestinal tract, acids and bases are absorbed in the undissociated (non-ionised) form by simple diffusion or by facilitated diffusion. It is expected that both the acids and the salts will be present in (or converted to) the acid form in the stomach. This means that for both aliphatic acid or aliphatic acid salt, the same compounds eventually enter the small intestine, where equilibrium, as a result of increased pH, will shift towards dissociation (ionised form).

Hence, the situation will be similar for compounds originating from acids and therefore no differences in uptake are anticipated

Note that the saturation or unsaturation level is not a factor in the toxicity of these substances and is not a critical component of the read across process.

Toxicokinetics:

The turnover of the [14C] surfactants in the rat showed that there was no significant difference in the rate or route of excretion of 14C given by intraperitoneal or subcutaneous administration. The main route of excretion was as ¹⁴CO₂ in the expired air at 6 h after administration. The remaining material was incorporated in the body. Longer fatty acid chains are more readily incorporated than shorter chains. At ca. 1.55 and 1.64 mg/kg bw, 71% of the C16:0 and 56% of the C18:0 was incorporated and 21% and 38% was excreted as ¹⁴CO₂, respectively.

Glycidyl fatty acid esters (GEs), one of the main contaminants in processed oils, are mainly formed during the deodorisation step in the refining process of edible oils and therefore occur in almost all refined edible oils. GEs are potential carcinogens, due to the fact that they readily hydrolyze into the free form glycidol in the gastrointestinal tract, which has been found to induce tumours in various rat tissues.

Therefore, significant effort has been devoted to inhibit and eliminate the formation of GEs

GEs contain a common terminal epoxide group but exhibit different fatty acid compositions. This class of compounds has been reported in edible oils after overestimation of 3-monochloropropane-1,2-diol (3-MCPD) fatty acid esters analysed by an indirect method, 3-MCPD esters have been studied as food processing contaminants and are found in various food types and food ingredients, particularly in refined edible oils. 3-Monochloropropane-1,2-diol (3-MCPD) and 2-monochloropropane-1,3-diol (2-MCPD) are chlorinated derivatives of glycerol (1,2,3-propanetriol). 3- and 2-MCPD and their fatty acid esters are among non-volatile chloropropanols, Glycidol is associated with the formation and decomposition of 3- and 2-MCPD. It forms monoesters with fatty acids (GE) during the refining of vegetable oils. Chloropropanols are formed in HVP during the hydrochloric acid-mediated hydrolysis step of the manufacturing process. In food production, chloropropanols form from the reaction of endogenous or added chloride with glycerol or acylglycerol.

Although harmful effects on humans and animals have not been demonstrated, the corresponding hydrolysates, 3-MCPD and glycidol, have been identified as rodent genotoxic carcinogens, ultimately resulting in the formation of kidney tumours (3-MCPD) and tumours at other tissue sites (glycidol). Therefore, 3-MCPD and glycidol have been categorised as "possible human carcinogens (group 2B) and "probably carcinogenic to humans (group 2A), respectively, by the International Agency for Research on Cancer (IARC).

Diacylglyceride (DAG) based oils produced by one company were banned from the global market due to "high levels" of GEs.

Several reports have also suggested that a bidirectional transformation process may occur not only between glycidol and 3-MCPD but also their esterified forms in the presence of chloride ions. The transformation rate of glycidol to 3-MCPD was higher than that of 3-MCPD to glycidol under acidic conditions in the presence of chloride ion.

Precursors of GEs in refined oils have been identified as partial acylglycerols, that is, DAGs and monoacylglycerides (MAGs); however, whether they also originate from triacylglycerides (TAGs) is still a topic of controversial debates. Several authors noted that pure TAGs were stable during heat treatment (such as 235 deg C) for 3 h and were therefore not involved in the formation of GEs. However, experimental results have shown that small amounts of GEs are present in a heat-treated oil model consisting of almost 100% TAGs. The formation of GEs from TAGs can be attributed to the pyrolysis of TAGs to DAGs and MAGs. In contrast, 3-MCPD esters in refined oils can be obtained from TAG. Presently, the mechanism for the formation of GE intermediates and the relationship between GEs and 3-MCPD esters are still unknown.

For triglycerides:

Carboxylic acid esters will undergo enzymatic hydrolysis by ubiquitously expressed GI esterases. The rate of hydrolysis is dependant on the structure of the ester, and may therefore be rapid or rather slow. Thus, due to hydrolysis, predictions on oral absorption based on the physico-chemical characteristics of the intact parent substance alone may no longer apply.

When considering the hydrolysis product glycerol, absorption is favoured based on passive and active absorption of glycerol.

The Cosmetic Ingredient Review (CIR) Expert Panel has issued three final reports on the safety of 25 triglycerides, i.e., fatty acid triesters of glycerin

High purity is needed for the triglycerides. Previously the Panel published a final report on a diglycerides, and concluded that the ingredients in the diglyceride family are safe in the present practices of use and concentration provided the content of 1,2-diesters is not high enough to induce epidermal hyperplasia. The Panel discussed that there was an increased level of concern because of data regarding the induction of protein kinase C (PKC) and the tumor promotion potential of 1,2-diacylglycerols. The Panel noted that, nominally, glyceryl-1,3-diesters contain 1,2-diesters, raising the concern that 1,2-diesters could potentially induce hyperplasia. The Panel did note that these compounds are more likely to cause these effects when the fatty acid chain length is <=14 carbons, when one fatty acid is saturated and one is not, and when given at high doses, repeatedly. Although minimal percutaneous absorption of triolein has been demonstrated in vivo using guinea pigs (but not hairless mice) and in vitro using full-thickness skin from hairless mice, the Expert Panel recognizes that, reportedly, triolein and tricaprilyn can enhance the skin penetration of other chemicals, and recommends that care should be exercised in using these and other glyceryl triesters in cosmetic products.

The Panel acknowledged that some of the triglycerides may be formed from plant-derived or animal-derived constituents. The Panel thus expressed concern regarding pesticide residues and heavy metals that may be present in botanical ingredients. They stressed that the cosmetics industry should continue to use the necessary procedures to sufficiently limit amounts of such impurities in an ingredient before blending them into cosmetic formulations. Additionally, the Panel considered the risks inherent in using animal-derived ingredients, namely the transmission of infectious agents. Although tallow may be used in the manufacture of glyceryl tallowate and is clearly animal-derived, the Panel notes that tallow is highly processed, and tallow derivatives even more so. The Panel agrees with determinations by the U.S. FDA that tallow derivatives are not risk materials for transmission of infectious agents.

Finally, the Panel discussed the issue of incidental inhalation exposure, as some of the triglycerides are used in cosmetic sprays and could possibly be inhaled. For example, triethylhexanoïn and trisostearin are reported to be used at maximum concentrations of 36% and 30%, respectively, in perfumes, and 14.7% and 10.4%, respectively, in face powders. The Panel noted that in aerosol products, 95% – 99% of droplets/particles would not be respirable to any appreciable amount. Furthermore, droplets/particles deposited in the nasopharyngeal or bronchial regions of the respiratory tract present no toxicological concerns based on the chemical and biological properties of these ingredients. Coupled with the small actual exposure in the breathing zone and the concentrations at which the ingredients are used, the available information indicates that incidental inhalation would not be a significant route of exposure that might lead to local respiratory or systemic effects

Cosmetic Ingredient Review (CIR) : Amended Safety Assessment of Triglycerides as Used in Cosmetics August 2017

Glyceryl triesters are also known as triglycerides; ingested triglycerides are metabolized to monoglycerides, free fatty acids, and glycerol, all of which are absorbed in the intestinal mucosa and undergo further metabolism. Dermal absorption of Triolein in mice was nil; the oil remained at the application site. Only slight absorption was seen in guinea pig skin. Tricaprylin and other glyceryl triesters have been shown to increase the skin penetration of drugs. Little or no acute, subchronic, or chronic oral toxicity was seen in animal studies unless levels approached a significant percentage of caloric intake. Subcutaneous injections of Tricaprylin in rats over a period of 5 weeks caused a granulomatous reaction characterized by oil deposits surrounded by macrophages. Dermal application was not associated with significant irritation in rabbit skin. Ocular exposures were, at most, mildly irritating to rabbit eyes. No evidence of sensitization or photosensitization was seen in a guinea pig maximization test. Most of the genotoxicity test systems were negative. Tricaprylin, Trioctanoïn, and Triolein have historically been used as vehicles in carcinogenicity testing of other chemicals. In one study, subcutaneous injection of Tricaprylin in newborn mice produced more tumors in lymphoid tissue than were seen in untreated animals, whereas neither subcutaneous or intraperitoneal injection in 4- to 6-week-old female mice produced any tumors in another study. Trioctanoïn injected subcutaneously in hamsters produced no tumors. Trioctanoïn injected intraperitoneally in pregnant rats was associated with an increase in mammary tumors in the offspring

Viper WRL Eco Rope Guardian ERG-1

compared to that seen in offspring of untreated animals, but similar studies in pregnant hamsters and rabbits showed no tumors in the offspring. One study of Triolein injected subcutaneously in rats showed no tumors at the injection site. As part of an effort to evaluate vehicles used in carcinogenicity studies, the National Toxicology Program conducted a 2-year carcinogenicity study in rats given Tricaprylin by gavage. This treatment was associated with a statistically significant dose-related increase in pancreatic acinar cell hyperplasia and adenoma, but there were no acinar carcinomas, the incidence of mononuclear leukemia was less, and nephropathy findings were reduced, all compared to corn oil controls. Overall, the study concluded that Tricaprylin did not offer significant advantages over corn oil as vehicles in carcinogenicity studies. Trilaurin was found to inhibit the formation of neoplasms initiated by dimethylbenzanthracene (DMBA) and promoted by croton oil. Tricaprylin was not teratogenic in mice or rats, but some reproductive effects were seen in rabbits. A low level of fetal eye abnormalities and a small percentage of abnormal sperm were reported in mice injected with Trioctanoin as a vehicle control. Clinical tests of Trilaurin at 36.3% in a commercial product applied to the skin produced no irritation reactions. Trilaurin, Tristearin, and Tribehenin at 40%, 1.68%, and 0.38%, respectively, in commercial products were also negative in repeated-insult patch tests. Tristearin at 0.32% in a commercial product induced transient, mild to moderate, ocular irritation after instillation into the eyes of human subjects. Based on the enhancement of penetration of other chemicals by skin treatment with glyceryl triesters, it is recommended that care be exercised in using them in cosmetic products.

Cosmetic Ingredient Review (CIR) Expert Panel: Final Report on the Safety Assessment of Trilaurin etc: Int J Toxicol, 20 Suppl 4, 61-94 2001

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

Viper WRL Eco Rope Guardian ERG-1	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
canola oil	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
castor oil	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	NOEC(ECx)	24h	Crustacea	100mg/l	2
	EC50	48h	Crustacea	100mg/l	2
canola oil, polymerised, oxidised	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
aluminium hydroxide benzoate stearate	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
1-decene homopolymer, hydrogenated	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
1-butene homopolymer	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
methyl methacrylate homopolymer	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
magnesium aluminosilicate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	410mg/l	2
	NOEC(ECx)	96h	Fish	<1.4mg/l	2
	EC50	48h	Crustacea	>10000mg/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*

When spilled this product may act as a typical oil, causing a film, sheen, emulsion or sludge at or beneath the surface of the body of water. The oil film on water surface may physically affect the aquatic organisms, due to the interruption of the oxygen transfer between the air and the water

Oils of any kind can cause:

- ▶ drowning of water-fowl due to lack of buoyancy, loss of insulating capacity of feathers, starvation and vulnerability to predators due to lack of mobility
- ▶ lethal effects on fish by coating gill surfaces, preventing respiration
- ▶ asphyxiation of benthic life forms when floating masses become engaged with surface debris and settle on the bottom and

Continued...

► adverse aesthetic effects of fouled shoreline and beaches

In case of accidental releases on the soil, a fine film is formed on the soil, which prevents the plant respiration process and the soil particle saturation. It may cause deep water infestation.

For aliphatic fatty acids and alcohols:

Environmental fate:

Saturated fatty acids are very stable in air, whereas unsaturated (C=C bonds) fatty acids are susceptible to oxidation.

Unsaturation increases the rate of metabolism although the degree of unsaturation and positioning of double bonds is not highly significant.

The available data indicate all fatty acid salt chain lengths up to and including C18 can be metabolised under aerobic conditions and can be considered to be readily biodegradable

All tests showed that fatty acids and lipids are readily biodegradable

The aliphatic acids are of similar very weak acid strength (approximately pKa 5), i.e., partially dissociate in aqueous solution; the salts of the aliphatic acids are highly dissociated in water solution such that the anion is the same for homologous salts and acids.

Slight (although inconsistent) effects on the trend for decreasing vapour pressure are also observed with the mono-, di- and tri-unsaturated substances as compared to the corresponding saturated substances.

Dicarboxylic acids: Compared to their corresponding single acid substances (C8-10 single component, saturated), the dicarboxylic acids exhibit modestly higher melting/ boiling points and water solubility, and lower partition coefficients and vapour pressures. The trends described above for changes in physical chemical properties with increasing carbon chain length apply.

Salts: As expected, the salts differ in physical / chemical properties as compared to their homologous single component substances. However the trends described above for single components with regard to changes in physical chemical properties with increasing carbon chain length apply

Models also indicate that the aliphatic acids will distribute primarily to soil and water, with lesser amounts to air and sediment. With increasing chain length, the percent distributions to soil and sediment generally increase and the percent distributions to water and air generally decrease.

The rate of degradation of fatty acids was investigated in two non-GLP studies.

The total fatty acids residue exhibits low persistence in soil. From the pattern of peaks decline, it was hypothesised a degradation pathway by the sequential elimination of C2 fragments. Consequently, the major soil metabolites of a given fatty acid would be other fatty acids with shorter chains.

Although mineralisation was not measured in these experiments, formation of CO₂ is the expected terminal step of this process. Fatty acids undergo aerobic biodegradation by the process of beta-oxidation. Beta-oxidation of the parent fatty acid forms acetate and a new fatty acid of two less carbon atoms. This process repeats itself until the compound is completely broken down. The hydrocarbon will eventually be degraded to CO₂ and H₂O. For this reason, the length of the fatty acid chain does not preclude biodegradation, but it may take longer to achieve complete mineralisation. The beta-oxidation sequence does not necessarily require the presence of molecular oxygen, and fatty acid biodegradation may proceed under anaerobic conditions.

Hydrolysis is not an important fate path in the environment due to the fact that the substances lack hydrolysable functional groups. Aliphatic acids are hydrolytically stable in aqueous solution.

Water solubility:

In general, the water solubility of single carbon chain length substances followed a pattern of decreasing solubility as carbon chain length increases, especially at C16 and higher. In addition, greater solubility is seen for dicarboxylic acids as compared to their homologous single acids:

In reviewing the physical/ chemical properties of the aliphatic acids, two predominant trends are clearly evident with increasing alkyl chain length and include: i) increasing melting point, boiling point, and partition coefficient, and ii) decreasing water solubility and vapour pressure. Within a given carbon chain length, melting point increases with increasing saturation and decreases with increasing unsaturation. The noted general trends with increasing alkyl chain length are observed when an entire single component group (12 saturated, 4 mono-unsaturated, 2 di-unsaturated, and 1 tri-unsaturated substances) is evaluated together; that is the degree of saturation or unsaturation does not alter the properties trend. The effect of mono-unsaturation (C14:1 to C22:1) appears to be a slight increase in water solubility and a slight decrease in the partition coefficient, as compared to the corresponding saturated substances; a similar trend is noted for the C18 di- or tri-unsaturated substances.

Fatty acids (including methyl esters) were stable to hydrolysis in the pH range of 1-14. It is not expected that photolysis would significantly contribute to the degradation of fatty acids in water.

According to modelling, the aliphatic acids are subject to photodegradation in air. Estimated half-lives generally increase with decreasing chain length and range from 0.6 hours to 17.5 hours.

Methyl (and other) esters are estimated to exhibit high mobility and the acids very high mobility. Mobility may be expected to be higher for the salts than for the corresponding acids and methyl esters

Biodegradation studies or model estimations for single and multi-component aliphatic acids generally confirm that the extent of biodegradation observed in 28 days meets the ready biodegradability criterion (>60%). When the 10-day window was not met or less than 60%, biodegradation was observed in 28 days, it is likely that the aliphatic acids tested were not fully in solution.

Biodegradability tests demonstrated that pelargonic acid (C9), potassium salts and methyl octanoate / methyl decanoate are readily biodegradable. It can be assumed that both acids and methyl esters fatty acids C7-C18 are readily biodegradable.

No experimental bioaccumulation data appear to be available but log Kow data from various sources are higher than 4, which indicates that fatty acids and natural lipids have a potential for bioaccumulating in aquatic organisms.

Fatty alcohols up to chain length C18 are biodegradable, with length up to C16 biodegrading within 10 days completely. Chains C16 to C18 were found to biodegrade from 62% to 76% in 10 days. Chains greater than C18 were found to degrade by 37% in 10 days. Field studies at waste-water treatment plants have shown that 99% of fatty alcohols lengths C12-C18 are removed.

A review of soaps (including calcium and magnesium salts) states that the available data indicate all fatty acid salt chain lengths up to and including C18 can be metabolised under aerobic conditions and can be considered to be biodegradable. Biodegradability did not appear to be influenced by even or odd chain length, degree of saturation or unsaturation or branching. For example odd/even chain length C8 and C9 are readily biodegradable; Saturation/unsaturation: C18(saturated) and C18 (di-unsaturated) are biodegradable, while C18 (mono-unsaturated) are readily biodegradable; branching or hydroxylation: the C18 hydroxylated substance was readily biodegradable and the C18 methyl branched substance was biodegradable.

Higher water solubility of the potassium, sodium and ammonium salts make these a lower ranked analogy for the aquatic toxicity endpoints for the (non-salt) aliphatic acids (and vice versa), while lower water solubility of the magnesium and calcium salts make these a lower ranked analogy for all other members of the category

The aliphatic acids also undergo biodegradation under anaerobic conditions.

Estimated bioconcentration factor values are calculated using EPI Suite v4.10. The aliphatic acids have BCF values less than 100, indicating a low potential for bioaccumulation

Fate prediction using fugacity modeling has shown that fatty alcohols with chain lengths of C10 and greater in water partition into sediment. Lengths C14 and above are predicted to stay in the air upon release. Modeling shows that each type of fatty alcohol will respond independently upon environmental release

Ecotoxicity

Structure-activity relationships based on carbon chain length are evident in the available data on the aquatic ecotoxicity of substances of this category (aquatic toxicity increases with increasing chain length up to a "cutoff" at or near 12 carbons).

The aliphatic acids category members possess properties indicating a hazard for the environment (acute toxicity to fish: between 1-100 mg/L for carbon chain lengths C6 through C12, and multi-component sodium or potassium salts C16-18; acute toxicity to aquatic invertebrates: between 1 and 100 mg/L for carbon chain lengths C6 through C9 (including sodium salts) and less than 1 mg/L for sodium salts single component aliphatic acids C18 and multi component sodium salt aliphatic acids with carbon chain lengths including C14 through C18; and, acute toxicity to aquatic plants: between 1-100 mg/L for carbon chain length C12, including sodium or ammonium salts).

There are a number of acute data for fatty acids and fatty acid salts to aquatic organisms although there is a predominance of data for fatty acid. There are few toxicity values for terrestrial organisms. Data availability / quality covering all the taxonomic groups for specific fatty acid salt chain lengths is poor. The chronic data set is very limited.

For chain lengths >C12, solubility decreases to a degree where an adverse effect would not be expected in the environment due to reduced bioavailability. Data for longer chain lengths have been generated using solvents which makes interpretation more difficult.

The most of few available data indicate low toxicity towards aquatic organisms with EC/LC50 values above 1000 mg/l. However, EC/LC50 values below 100 mg/l are not unusual either

Fish, invertebrates and algae experience similar levels of toxicity with fatty alcohols although it is dependent on chain length with the shorter chain having greater toxicity potential. Longer chain lengths show no toxicity to aquatic organisms.

The available toxicity data indicated low acute and short-term (for birds only) toxicity to birds and mammals. Given that fatty acids are an essential component of the diet of birds and mammals a low risk is expected. On the basis that fatty acids are readily biodegradable and are an essential component of the diet of birds and mammals, a low reproductive risk is expected.

No toxicity data were available for higher aquatic plants and therefore a risk assessment cannot be performed. As pelargonic acid, fatty acid/salt and C8-C10 methyl esters are used as herbicides and plant growth regulators, a data gap to address the risk to higher aquatic plants was identified

A low risk to natural populations of bees and non-target arthropods was concluded for representative greenhouses uses of potassium salts of fatty acids, fatty acid/salt and C8-C10 methyl esters.

Given that fatty acids are readily biodegradable a low risk to sewage treatment organisms was concluded for all of the representative uses.

For Group E aliphatic esters (polyol esters):

Environmental fate:

Continued...

In general, the polyol esters have molecular weights of greater than 400, have high boiling points greater than >400 C and are expected to be relatively non-volatile, lipophilic (log P > 7) and are relatively water-insoluble.

Biodegradability:

All of the tested polyol esters showed extensive biodegradation in the standard 28-day test and these findings indicate that polyol esters are capable of undergoing metabolic ester cleavage, which leads to the generation of the corresponding fatty acids as well as the polyol alcohols.

The "readily" biodegradability findings observed for some polyol esters (especially pentaerythritol esters and those with natural fatty acids such as oleic acid) indicate that enzymatic cleavage of the ester linkage(s) must be occurring significantly, in order to achieve the high level of biodegradation observed. This would be consistent with the fact that fatty acids (e.g., oleic acids), arising from enzymatic cleavage of the polyol esters, are rapidly biodegraded. In addition, the results are also consistent with the fact the pentaerythritol itself is readily biodegradable (84% biodegradation in 28 days)

Ecotoxicity:

Acute aquatic toxicity studies have been carried out for many polyol esters. There is sufficient information on the aquatic toxicity of many of the Group E polyol esters in fish, invertebrates and algae. In general, the tested polyol esters do not cause acute toxicity to aquatic organisms. In addition, polyol esters have very limited water solubility and these materials are probably not likely to cause toxicity at their maximum water solubility.

Fish LC50 (96 h): Danio rerio >102 mg/l (OECD 203)

Daphnia magna EC50 (48 h): >106 mg/l (OECD 202); NOELR (21 d): >=0.11 g/l

Algae EC50 (72 h): Pseudokirchneriella subcapitata >110 mg/l (OECD 201)

Substances containing unsaturated carbons are ubiquitous in indoor environments. They result from many sources (see below). Most are reactive with environmental ozone and many produce stable products which are thought to adversely affect human health. The potential for surfaces in an enclosed space to facilitate reactions should be considered.

Source of unsaturated substances	Unsaturated substances (Reactive Emissions)	Major Stable Products produced following reaction with ozone.
Occupants (exhaled breath, ski oils, personal care products)	Isoprene, nitric oxide, squalene, unsaturated sterols, oleic acid and other unsaturated fatty acids, unsaturated oxidation products	Methacrolein, methyl vinyl ketone, nitrogen dioxide, acetone, 6MHQ, geranyl acetone, 4OPA, formaldehyde, nonanol, decanal, 9-oxo-nonanoic acid, azelaic acid, nonanoic acid.
Soft woods, wood flooring, including cypress, cedar and silver fir boards, houseplants	Isoprene, limonene, alpha-pinene, other terpenes and sesquiterpenes	Formaldehyde, 4-AMC, pinoaldehyde, pinic acid, pinonic acid, formic acid, methacrolein, methyl vinyl ketone, SOAs including ultrafine particles
Carpets and carpet backing	4-Phenylcyclohexene, 4-vinylcyclohexene, styrene, 2-ethylhexyl acrylate, unsaturated fatty acids and esters	Formaldehyde, acetaldehyde, benzaldehyde, hexanal, nonanal, 2-nonenal
Linoleum and paints/polishes containing linseed oil	Linoleic acid, linolenic acid	Propanal, hexanal, nonanal, 2-heptenal, 2-nonenal, 2-decenal, 1-pentene-3-one, propionic acid, n-butyric acid
Latex paint	Residual monomers	Formaldehyde
Certain cleaning products, polishes, waxes, air fresheners	Limonene, alpha-pinene, terpinolene, alpha-terpineol, linalool, linalyl acetate and other terpenoids, longifolene and other sesquiterpenes	Formaldehyde, acetaldehyde, glycoaldehyde, formic acid, acetic acid, hydrogen and organic peroxides, acetone, benzaldehyde, 4-hydroxy-4-methyl-5-hexen-1-ol, 5-ethenyl-dihydro-5-methyl-2(3H)-furanone, 4-AMC, SOAs including ultrafine particles
Natural rubber adhesive	Isoprene, terpenes	Formaldehyde, methacrolein, methyl vinyl ketone
Photocopier toner, printed paper, styrene polymers	Styrene	Formaldehyde, benzaldehyde
Environmental tobacco smoke	Styrene, acrolein, nicotine	Formaldehyde, benzaldehyde, hexanal, glyoxal, N-methylformamide, nicotinaldehyde, cotinine
Soiled clothing, fabrics, bedding	Squalene, unsaturated sterols, oleic acid and other saturated fatty acids	Acetone, geranyl acetone, 6MHO, 4OPA, formaldehyde, nonanal, decanal, 9-oxo-nonanoic acid, azelaic acid, nonanoic acid
Soiled particle filters	Unsaturated fatty acids from plant waxes, leaf litter, and other vegetative debris; soot; diesel particles	Formaldehyde, nonanal, and other aldehydes; azelaic acid; nonanoic acid; 9-oxo-nonanoic acid and other oxo-acids; compounds with mixed functional groups (=O, -OH, and -COOH)
Ventilation ducts and duct liners	Unsaturated fatty acids and esters, unsaturated oils, neoprene	C5 to C10 aldehydes
"Urban grime"	Polycyclic aromatic hydrocarbons	Oxidized polycyclic aromatic hydrocarbons
Perfumes, colognes, essential oils (e.g. lavender, eucalyptus, tea tree)	Terpinene-4-ol, gamma-terpinene	Formaldehyde, 4-AMC, acetone, 4-hydroxy-4-methyl-5-hexen-1-ol, 5-ethenyl-dihydro-5-methyl-2(3H) furanone, SOAs including ultrafine particles
Overall home emissions	Limonene, alpha-pinene, styrene	Formaldehyde, 4-AMC, pinonaldehyde, acetone, pinic acid, pinonic acid, formic acid, benzaldehyde, SOAs including ultrafine particles

Abbreviations: 4-AMC, 4-acetyl-1-methylcyclohexene; 6MHQ, 6-methyl-5-heptene-2-one, 4OPA, 4-oxopentanal, SOA, Secondary Organic Aerosols

Reference: Charles J Weschler; Environmental Health Perspectives, Vol 114, October 2006

For alkenes (olefins)

Environmental fate:

Ecotoxicity studies conducted with a wide range of products have shown little potential for toxicity to aquatic organisms under expected conditions of use or in the event of an accidental release. Not all alpha olefins are readily biodegradable; however, they will ultimately biodegrade. While the octanol/water partition coefficients of alpha olefins suggest a potential for bioaccumulation of these materials in aquatic organisms, the volatility of these materials (especially for the liquid alpha olefins) and the low-water solubility (indicative of limited bioavailability), would indicate that bioaccumulation will not occur. Under most environmental scenarios, extensive evaporation and subsequent degradation in the atmosphere would preclude bioaccumulation. Therefore, alpha olefins are not expected to be toxic to aquatic organisms, will biodegrade, and will not bioaccumulate. The potential for exposure of aquatic organisms to members of the higher olefins will be influenced by their physico-chemical properties. The predicted or measured water solubilities of these olefins range from 50 mg/L at 20 C for hexene to 0.00015 mg/L at 25 C for 1-octadecene, and to 6.33 [E-23] mg/L at 25 C for C54 alpha olefin, which suggests there is a lower potential for the larger olefins to be bioavailable to aquatic organisms due to their low solubilities. Their vapor pressures range from 230.6 hPa at 25 C for hexene to 0.00009 hPa at 25 C for 1-octadecene, and to 1.13 [E-16] hPa at 25 C for C54 alpha olefin, which suggests the shorter chain olefins will tend to partition to the air at a significant rate and not remain in the other environmental compartments for long periods of time; while the longer chain olefins will tend to partition primarily to water, soil or sediment, depending on water solubility and sorption behavior. The predicted soil adsorption coefficients (Koc) range from 149 for C6 to 230,800 for C18 and to 1.0 [E10] for C54, indicating increasing partitioning to soil/sediment with increasing carbon number. Level I fugacity modelling predicts that the C6-13 olefins would partition primarily to air, while the C16 and longer chain olefins would partition primarily to soil. Results of Level III fugacity modelling suggest that the C6 -8 olefins will partition primarily to the water compartment; and, as the chain length increases beyond C10, soil and sediment become the primary compartments. These chemicals have a very low potential to hydrolyse and do not photodegrade directly. However, in the air, all members of the category are subject to atmospheric oxidation from hydroxyl radical attack, with calculated degradation half-lives of 1.8 to 4.8 hours. C6 -30 olefins have been shown to degrade to an extent of approximately 8-92% in standard 28 day biodegradation tests. These results were not clearly correlated with carbon number or any other identifiable parameter; however, the weight of evidence shows that the members of the higher olefins have potential for degradation in the environment. Volatilisation from water is predicted to occur rapidly (hours to days), with Henry's Law Constants (bond method) ranging from 0.423 (C6) to 10.7 (C18), and to 2.89 [E5] (C54) atm- m3/mol. Consideration of these degradation processes supports the assessment that these substances will degrade relatively rapidly in the environment and not persist. Based on calculated bioconcentration factors, the C6, C7, and C16 and longer chain length category members are not expected to bioaccumulate (BCF: C6 = 44-46, C7 = 236, C16 = 71-92 and >= C18 = 3.2-4.6). Although the C8 - 15 olefins have BCFs ranging from 313 to 2030, and Kow values ranging from 4.13 to 7.49, and thus are considered to have the potential for bioaccumulation, their physico-chemical properties and fate indicate that there would be limited environmental exposure because of volatility, biodegradability and limited solubility.

Ecotoxicity:

Data indicate that acute aquatic toxicity can be observed for C6 through the C10 olefins (C6: EC/LC50 range of 1-10 mg/L; C7-C10: EC/LC50 range of 0.1-1.0 mg/L), and that toxicity increases with increasing carbon number within that range, which is consistent with increasing Kow values (3.07 -5.12). Above a chain length of 10, toxicity is not observed within the limits of solubility. However, data indicate that chronic aquatic toxicity can be observed in the C10 olefins (EC10 = 20.0 ug/L, EC50 = 28.1 ug/L, NOEC = 19.04 ug/L). Data also suggest that aquatic toxicity does not differ with bond location or presence of branching.

For aluminium and its compounds and salts:

Despite its prevalence in the environment, no known form of life uses aluminium salts metabolically. In keeping with its pervasiveness, aluminium is well tolerated by plants and animals. Owing to their prevalence, potential beneficial (or otherwise) biological roles of aluminium compounds are of continuing interest.

Environmental fate:

Aluminium occurs in the environment in the form of silicates, oxides and hydroxides, combined with other elements such as sodium, fluorine and arsenic complexes with organic matter.

Acidification of soils releases aluminium as a transportable solution. Mobilisation of aluminium by acid rain results in aluminium becoming available for plant uptake.

As an element, aluminium cannot be degraded in the environment, but may undergo various precipitation or ligand exchange reactions. Aluminium in compounds has only one oxidation state (+3), and would not undergo oxidation-reduction reactions under environmental conditions. Aluminium can be complexed by various ligands present in the environment (e.g., fulvic and humic acids). The solubility of aluminium in the environment will depend on the ligands present and the pH.

The trivalent aluminium ion is surrounded by six water molecules in solution. The hydrated aluminium ion, [Al(H2O)6]3+, undergoes hydrolysis, in which a stepwise deprotonation of the coordinated water ligands forms bound hydroxide ligands (e.g., [Al(H2O)5(OH)]2+, [Al(H2O)4(OH)2]+). The speciation of aluminium in water is pH dependent. The

hydrated trivalent aluminum ion is the predominant form at pH levels below 4. Between pH 5 and 6, the predominant hydrolysis products are $Al(OH)_2^+$ and $Al(OH)_3$, while the solid $Al(OH)_3$ is most prevalent between pH 5.2 and 8.8. The soluble species $Al(OH)_4^-$ is the predominant species above pH 9, and is the only species present above pH 10. Polymeric aluminum hydroxides appear between pH 4.7 and 10.5, and increase in size until they are transformed into colloidal particles of amorphous $Al(OH)_3$, which crystallise to gibbsite in acid waters. Polymerisation is affected by the presence of dissolved silica; when enough silica is present, aluminum is precipitated as poorly crystallised clay mineral species.

Hydroxyaluminum compounds are considered amphoteric (e.g., they can act as both acids and bases in solution). Because of this property, aluminum hydroxides can act as buffers and resist pH changes within the narrow pH range of 4-5.

Monomeric aluminum compounds, typified by aluminum fluoride, chloride, and sulfate, are considered reactive or labile compounds, whereas polymeric aluminum species react much more slowly in the environment. Aluminum has a stronger attraction for fluoride in an acidic environment compared to other inorganic ligand.

The adsorption of aluminum onto clay surfaces can be a significant factor in controlling aluminum mobility in the environment, and these adsorption reactions, measured in one study at pH 3.0-4.1, have been observed to be very rapid. However, clays may act either as a sink or a source for soluble aluminum depending on the degree of aluminum saturation on the clay surface.

Within the pH range of 5-6, aluminum complexes with phosphate and is removed from solution. Because phosphate is a necessary nutrient in ecological systems, this immobilization of both aluminum and phosphate may result in depleted nutrient states in surface water.

Plant species and cultivars of the same species differ considerably in their ability to take up and translocate aluminum to above-ground parts. Tea leaves may contain very high concentrations of aluminum, >5,000 mg/kg in old leaves. Other plants that may contain high levels of aluminum include Lycopodium (Lycopodiaceae), a few ferns, Symplocos (Symplocaceae), and Orites (Proteaceae). Aluminum is often taken up and concentrated in root tissue. In sub-alpine ecosystems, the large root biomass of the Douglas fir, *Abies amabilis*, takes up aluminum and immobilizes it, preventing large accumulation in above-ground tissue. It is unclear to what extent aluminum is taken up into root food crops and leafy vegetables. An uptake factor (concentration of aluminum in the plant/concentration of aluminum in soil) of 0.004 for leafy vegetables and 0.00065 for fruits and tubers has been reported, but the pH and plant species from which these uptake factors were derived are unclear. Based upon these values, however, it is clear that aluminum is not taken up in plants from soil, but is instead biodiluted.

Aluminum concentrations in rainbow trout from an alum-treated lake, an untreated lake, and a hatchery were highest in gill tissue and lowest in muscle. Aluminum residue analyses in brook trout have shown that whole-body aluminum content decreases as the fish advance from larvae to juveniles. These results imply that the aging larvae begin to decrease their rate of aluminum uptake, to eliminate aluminum at a rate that exceeds uptake, or to maintain approximately the same amount of aluminum while the body mass increases. The decline in whole-body aluminum residues in juvenile brook trout may be related to growth and dilution by edible muscle tissue that accumulated less aluminum than did the other tissues.

The greatest fraction of the gill-associated aluminum was not sorbed to the gill tissue, but to the gill mucus. It is thought that mucus appears to retard aluminum transport from solution to the membrane surface, thus delaying the acute biological response of the fish. It has been reported that concentrations of aluminum in whole-body tissue of the Atlantic salmon exposed to high concentrations of aluminum ranging from 3 ug/g (for fish exposed to 33 ug/L) to 96 ug/g (for fish exposed to 264 ug/L) at pH 5.5. After 60 days of exposure, BCFs ranged from 76 to 190 and were directly related to the aluminum exposure concentration. In acidic waters (pH 4.6-5.3) with low concentrations of calcium (0.5-1.5 mg Ca/L), labile aluminum between 25 and 75 ug/L is toxic. Because aluminum is toxic to many aquatic species, it is not bioaccumulated to a significant degree (BCF <300) in most fish and shellfish; therefore, consumption of contaminated fish does not appear to be a significant source of aluminum exposure in humans.

Bioconcentration of aluminum has also been reported for several aquatic invertebrate species. BCF values ranging from 0.13 to 0.5 in the whole-body were reported for the snail. Bioconcentration of aluminum has also been reported for aquatic insects.

Ecotoxicity:

Freshwater species pH >6.5

Fish: Acute LC50 (48-96 h) 5 spp: 0.6 (*Salmo salar*) - 106 mg/L; Chronic NOEC (8-28 d): 7 spp, NOEC, 0.034-7.1 mg/L. The lowest measured chronic figure was an 8-d LC50 of 0.17 mg/L for *Micropterus* sp.

Amphibian: Acute LC50 (4 d): *Bufo americanus*, 0.86-1.66 mg/L; Chronic LC50 (8-d) 2.28 mg/L

Crustaceans LC50 (48 h): 1 sp 2.3-36.9 mg/L; Chronic NOEC (7-28 d) 3 spp, 0.136-1.72 mg/L

Algae EC50 (96 h): population growth, 0.46-0.57 mg/L; 2 spp, chronic NOEC, 0.8-2.0 mg/L

Freshwater species pH <6.5 (all between pH 4.5 and 6.0)

Fish LC50 (24-96 h): 4 spp, 0.015 (*S. trutta*) - 4.2 mg/L; chronic data on *Salmo trutta*, LC50 (21-42 d) 0.015- 0.105 mg/L

Amphibians LC50 (4-5 d): 2 spp, 0.540-2.670 mg/L (absolute range 0.40-5.2 mg/L)

Alga: 1 sp NOEC growth 2.0 mg/L

Among freshwater aquatic plants, single-celled plants are generally the most sensitive to aluminium. Fish are generally more sensitive to aluminium than aquatic invertebrates.

Aluminium is a gill toxicant to fish, causing both ionoregulatory and respiratory effects.

The bioavailability and toxicity of aluminium is generally greatest in acid solutions. Aluminium in acid habitats has been observed to be toxic to fish and phytoplankton. Aluminium is generally more toxic over the pH range 4.4-5.4, with a maximum toxicity occurring around pH 5.0-5.2. The inorganic single unit aluminium species ($Al(OH)_2^+$) is thought to be the most toxic. Under very acid conditions, the toxic effects of the high H^+ concentration appear to be more important than the effects of low concentrations of aluminium; at approximately neutral pH values, the toxicity of aluminium is greatly reduced. The solubility of aluminium is also enhanced under alkaline conditions, due to its amphoteric character, and some researchers found that the acute toxicity of aluminium increased from pH 7 to pH 9. However, the opposite relationship was found in other studies. The uptake and toxicity of aluminium in freshwater organisms generally decreases with increasing water hardness under acidic, neutral and alkaline conditions. Complexing agents such as fluoride, citrate and humic substances reduce the availability of aluminium to organisms, resulting in lower toxicity. Silicon can also reduce aluminium toxicity to fish.

Drinking Water Standards:

aluminium: 200 ug/l (UK max.)

200 ug/l (WHO guideline)

chloride: 400 mg/l (UK max.)

250 mg/l (WHO guideline)

fluoride: 1.5 mg/l (UK max.)

1.5 mg/l (WHO guideline)

nitrate: 50 mg/l (UK max.)

50 mg/l (WHO guideline)

sulfate: 250 mg/l (UK max.)

Soil Guideline: none available.

Air Quality Standards: none available.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
1-decene homopolymer, hydrogenated	LOW	LOW
methyl methacrylate homopolymer	LOW (Half-life = 56 days)	LOW (Half-life = 0.4 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
canola oil	LOW (LogKOW = 22.65)
castor oil	LOW (LogKOW = 18.1)
1-decene homopolymer, hydrogenated	HIGH (LogKOW = 5.116)
methyl methacrylate homopolymer	LOW (LogKOW = 1.2751)

Mobility in soil

Ingredient	Mobility
1-decene homopolymer, hydrogenated	LOW (Log KOC = 1724)
methyl methacrylate homopolymer	LOW (Log KOC = 10.14)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ 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.
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SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (ADG): 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

Product name	Group
canola oil	Not Available
castor oil	Not Available
canola oil, polymerised, oxidised	Not Available
aluminium hydroxide benzoate stearate	Not Available
1-decene homopolymer, hydrogenated	Not Available
1-butene homopolymer	Not Available
methyl methacrylate homopolymer	Not Available
magnesium aluminosilicate	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
canola oil	Not Available
castor oil	Not Available
canola oil, polymerised, oxidised	Not Available
aluminium hydroxide benzoate stearate	Not Available
1-decene homopolymer, hydrogenated	Not Available
1-butene homopolymer	Not Available
methyl methacrylate homopolymer	Not Available
magnesium aluminosilicate	Not Available

SECTION 15 Regulatory information

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

canola oil is found on the following regulatory lists

Not Applicable

castor oil is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

canola oil, polymerised, oxidised is found on the following regulatory lists

Not Applicable

aluminium hydroxide benzoate stearate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

1-decene homopolymer, hydrogenated is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

1-butene homopolymer is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

methyl methacrylate homopolymer is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

magnesium aluminosilicate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (canola oil; canola oil, polymerised, oxidised)
Canada - DSL	Yes
Canada - NDSL	No (castor oil; canola oil, polymerised, oxidised; aluminium hydroxide benzoate stearate; 1-decene homopolymer, hydrogenated; 1-butene homopolymer; methyl methacrylate homopolymer; magnesium aluminosilicate)
China - IECSC	No (canola oil, polymerised, oxidised)
Europe - EINEC / ELINCS / NLP	No (canola oil; canola oil, polymerised, oxidised; 1-butene homopolymer; methyl methacrylate homopolymer)
Japan - ENCS	No (canola oil; canola oil, polymerised, oxidised; magnesium aluminosilicate)
Korea - KECI	No (canola oil, polymerised, oxidised; aluminium hydroxide benzoate stearate)
New Zealand - NZIoC	No (canola oil, polymerised, oxidised)
Philippines - PICCS	No (canola oil; canola oil, polymerised, oxidised; aluminium hydroxide benzoate stearate)
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory 'Active'
Taiwan - TCSI	No (canola oil, polymerised, oxidised)
Mexico - INSQ	No (canola oil; canola oil, polymerised, oxidised; aluminium hydroxide benzoate stearate)
Vietnam - NCI	No (canola oil, polymerised, oxidised)
Russia - FBEPH	No (canola oil; canola oil, polymerised, oxidised; aluminium hydroxide benzoate stearate; 1-butene homopolymer; magnesium aluminosilicate)
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	23/12/2022
Initial Date	10/10/2017

SDS Version Summary

Version	Date of Update	Sections Updated
3.1	01/11/2019	One-off system update. NOTE: This may or may not change the GHS classification
4.1	23/12/2022	Classification review due to GHS Revision change.

Other information

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

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

Definitions and abbreviations

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

- ▶ LOD: Limit Of Detection
- ▶ OTV: Odour Threshold Value
- ▶ BCF: BioConcentration Factors
- ▶ BEI: Biological Exposure Index
- ▶ 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

- ▶ AIC: 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

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TEL (+61 3) 9572 4700.