# Skin Again Ointment
## SEN Skin

Chemwatch Hazard Alert Code: **2**

Chemwatch: **5423-52**
Version No: **2.1.1.1**
Safety Data Sheet according to WHS and ADG requirements

Issue Date: **07/09/2020**
Print Date: **09/09/2020**
S.GHS.AUS.EN

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

### Product Identifier

| | |
|---|---|
| **Product name** | Skin Again Ointment |
| **Synonyms** | Not Available |
| **Other means of identification** | Not Available |

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

| | |
|---|---|
| **Relevant identified uses** | Hand Sanitiser. For external use only, apply a few pumps of hand sanitiser on palm of hands and rub gently on both hands until completely dry for around 20 seconds.<br>Use according to manufacturer's directions. |

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

| | |
|---|---|
| **Registered company name** | **SEN Skin** |
| **Address** | Level 1/1966 Beach Road Malaga WA 6090 Australia |
| **Telephone** | 9200 5872 |
| **Fax** | Not Available |
| **Website** | www.senskin.com.au |
| **Email** | jill@senskin.com.au |

### Emergency telephone number

| | |
|---|---|
| **Association / Organisation** | **SEN Skin** |
| **Emergency telephone numbers** | 1800 838 064 |
| **Other emergency telephone numbers** | 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.**

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

### Label elements

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

**Page 1 continued...**

### Hazard statement(s)

| | |
|---|---|
| **H319** | Causes serious eye irritation. |

### Precautionary statement(s) Prevention

| | |
|---|---|
| **P280** | Wear protective gloves/protective clothing/eye protection/face protection. |

### 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. |
| **P337+P313** | If eye irritation persists: Get medical advice/attention. |

### 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 |
|---|---|---|
| 8001-21-6 | >60 | sunflower oil |
| Not Available | <60 | vitamin D |
| Not Available | <60 | vitamin B-6, 1 |
| Not Available | <60 | vitamin A |
| Not Available | balance | Ingredients determined not to be hazardous |
| Not Available | | include |
| 68920-03-6 | <60 | Shea butter |

## SECTION 4 First aid measures

### Description of first aid measures

| | |
|---|---|
| **Eye Contact** | If this product comes in contact with the eyes:<br>▸ Wash out immediately with fresh running water.<br>▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.<br>▸ Seek medical attention without delay; if pain persists or recurs seek medical attention.<br>▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. |
| **Skin Contact** | Wipe off excess with absorbent tissue or towel.<br>Seek medical attention if swelling/redness/blistering or irritation occurs. |
| **Inhalation** | ▸ If fumes, aerosols or combustion products are inhaled remove from contaminated area.<br>▸ Other measures are usually unnecessary. |
| **Ingestion** | ▸ If swallowed do **NOT** induce vomiting.<br>▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.<br>▸ Observe the patient carefully.<br>▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.<br>▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.<br>▸ Seek medical advice. |

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

Treat symptomatically.

## SECTION 5 Firefighting measures

**Continued...**

### Extinguishing media

- Water spray or fog.
- Foam.
- Dry chemical powder.
- BCF (where regulations permit).
- 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 |

### Advice for firefighters

| | |
|---|---|
| **Fire Fighting** | ‣ Alert Fire Brigade and tell them location and nature of hazard.<br>‣ Wear breathing apparatus plus protective gloves.<br>‣ Prevent, by any means available, spillage from entering drains or water courses.<br>‣ Use water delivered as a fine spray to control fire and cool adjacent area.<br>‣ **DO NOT** approach containers suspected to be hot.<br>‣ Cool fire exposed containers with water spray from a protected location.<br>‣ If safe to do so, remove containers from path of fire.<br>‣ Equipment should be thoroughly decontaminated after use. |
| **Fire/Explosion Hazard** | ‣ Combustible.<br>‣ Slight fire hazard when exposed to heat or flame.<br>‣ Heating may cause expansion or decomposition leading to violent rupture of containers.<br>‣ On combustion, may emit toxic fumes of carbon monoxide (CO).<br>‣ May emit acrid smoke.<br>‣ Mists containing combustible materials may be explosive.<br>Combustion products include:<br>carbon dioxide ($CO_2$)<br>acrolein<br>other pyrolysis products typical of burning organic material.<br>May emit poisonous fumes.<br>May emit corrosive fumes.<br>**CARE**: Water in contact with hot liquid may cause foaming and a steam explosion with wide scattering of hot oil and possible severe burns. Foaming may cause overflow of containers and may result in possible fire. |
| **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** | Slippery when spilt.<br>‣ Clean up all spills immediately.<br>‣ Avoid contact with skin and eyes.<br>‣ Wear impervious gloves and safety goggles.<br>‣ Trowel up/scrape up.<br>‣ Place spilled material in clean, dry, sealed container.<br>‣ Flush spill area with water. |
| **Major Spills** | **CARE**: Absorbent materials wetted with occluded oil must be moistened with water as they may auto-oxidize, become self heating and ignite.<br>Some oils slowly oxidise when spread in a film and oil on cloths, mops, absorbents may autoxidise and generate heat, smoulder, ignite and burn. In the workplace oily rags should be collected and immersed in water.<br>Slippery when spilt.<br>Minor hazard.<br>‣ Clear area of personnel.<br>‣ Alert Fire Brigade and tell them location and nature of hazard.<br>‣ Control personal contact with the substance, by using protective equipment as required.<br>‣ Prevent spillage from entering drains or water ways.<br>‣ Contain spill with sand, earth or vermiculite.<br>‣ Collect recoverable product into labelled containers for recycling.<br>‣ Absorb remaining product with sand, earth or vermiculite and place in appropriate containers for disposal.<br>‣ Wash area and prevent runoff into drains or waterways. |

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| | |
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| | ▸ If contamination of drains or waterways occurs, advise emergency services. |

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

## SECTION 7 Handling and storage

### Precautions for safe handling

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|---|---|
| **Safe handling** | ▸ Limit all unnecessary personal contact.<br>▸ Wear protective clothing when risk of exposure occurs.<br>▸ Use in a well-ventilated area.<br>▸ Avoid contact with incompatible materials.<br>▸ When handling, **DO NOT** eat, drink or smoke.<br>▸ Keep containers securely sealed when not in use.<br>▸ Avoid physical damage to containers.<br>▸ Always wash hands with soap and water after handling.<br>▸ Work clothes should be laundered separately.<br>▸ Use good occupational work practice.<br>▸ Observe manufacturer's storage and handling recommendations contained within this SDS.<br>▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained. |
| **Other information** | Consider storage under inert gas.<br>▸ Store in original containers.<br>▸ Keep containers securely sealed.<br>▸ No smoking, naked lights or ignition sources.<br>▸ Store in a cool, dry, well-ventilated area.<br>▸ Store away from incompatible materials and foodstuff containers.<br>▸ Protect containers against physical damage and check regularly for leaks.<br>▸ Observe manufacturer's storage and handling recommendations contained within this SDS. |

### Conditions for safe storage, including any incompatibilities

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|---|---|
| **Suitable container** | Supplied in 50ml and 100 ml. |
| **Storage incompatibility** | ▸ Avoid reaction with oxidising agents, bases and strong reducing agents.<br>▸ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates. |

\+ X + O + + +

**X** — Must not be stored together
**0** — May be stored together with specific preventions
**+** — May be stored together

## SECTION 8 Exposure controls / personal protection

### Control parameters

**Occupational Exposure Limits (OEL)**

**INGREDIENT DATA**

Not Available

**Emergency Limits**

| Ingredient | Material name | TEEL-1 | TEEL-2 | TEEL-3 |
|---|---|---|---|---|
| Skin Again Ointment | Not Available | Not Available | Not Available | Not Available |

| Ingredient | Original IDLH | Revised IDLH |
|---|---|---|
| sunflower oil | Not Available | Not Available |
| Shea butter | Not Available | Not Available |

**Occupational Exposure Banding**

| Ingredient | Occupational Exposure Band Rating | Occupational Exposure Band Limit |
|---|---|---|
| sunflower oil | E | ≤ 0.1 ppm |
| ***Notes:*** | *Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's* | |

**Continued...**

| Ingredient | Occupational Exposure Band Rating | Occupational Exposure Band Limit |
|---|---|---|
| | *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.* | |

## Exposure controls

| | |
|---|---|
| **Appropriate engineering controls** | General exhaust is adequate under normal operating conditions. |
| **Personal protection** |  |
| **Eye and face protection** | No special equipment for minor exposure i.e. when handling small quantities.<br>OTHERWISE:<br>▸ Safety glasses with side shields.<br>▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent] |
| **Skin protection** | See Hand protection below |
| **Hands/feet protection** | ▸ Bare skin is cleaned with this material.<br>▸ Application of hand cream / barrier cream after use is recommended. |
| **Body protection** | See Other protection below |
| **Other protection** | No special equipment needed when handling small quantities.<br>**OTHERWISE:**<br>▸ Overalls.<br>▸ Skin cleansing cream.<br>▸ Eyewash unit.<br>▸ Do not spray on hot surfaces. |

## Respiratory protection

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

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

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

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

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

## SECTION 9 Physical and chemical properties

## Information on basic physical and chemical properties

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|---|---|
| **Appearance** | Thick paste cream with a mild sweet floral odour; mixes with water. |

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| | | | |
|---|---|---|---|
| **Physical state** | Non Slump Paste | **Relative density (Water = 1)** | 0.93 @ 20 C |
| **Odour** | Not Available | **Partition coefficient n-octanol / water** | Not Available |
| **Odour threshold** | Not Available | **Auto-ignition temperature (°C)** | Not Available |
| **pH (as supplied)** | 7 | **Decomposition temperature** | Not Available |
| **Melting point / freezing point (°C)** | 45-50 | **Viscosity (cSt)** | >5000 |
| **Initial boiling point and boiling range (°C)** | Not Applicable | **Molecular weight (g/mol)** | Not Applicable |
| **Flash point (°C)** | >95 | **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** | Miscible | **pH as a solution (1%)** | Not Available |
| **Vapour density (Air = 1)** | Not Available | **VOC g/L** | Not Available |

## SECTION 10 Stability and reactivity

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

## SECTION 11 Toxicological information

### Information on toxicological effects

| | |
|---|---|
| **Inhaled** | Inhalation hazard is increased at higher temperatures.<br>Not normally a hazard due to non-volatile nature of product<br>Inhalation of oil droplets or aerosols may cause discomfort and may produce chemical inflammation of the lungs.<br>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.<br>There is some evidence to suggest that the material can cause respiratory irritation in some persons. The body's response to such irritation can cause further lung damage. |
| **Ingestion** | Accidental ingestion of the material may be damaging to the health of the individual. |
| **Skin Contact** | Discontinue use if irritation occurs<br>Not considered an irritant through normal use. |
| **Eye** | Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals.<br>Prolonged eye contact may cause inflammation characterised by a temporary redness of the conjunctiva (similar to windburn). |
| **Chronic** | Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.<br>Principal hazards are accidental eye contact and cleaner overuse. Overuse or obsessive cleaner use may lead to defatting of the skin and may cause irritation, drying, cracking, leading to dermatitis. |

| | | |
|---|---|---|
| **Skin Again Ointment** | **TOXICITY** | **IRRITATION** |
| | Not Available | Not Available |
| **sunflower oil** | **TOXICITY** | **IRRITATION** |
| | Not Available | Not Available |

**Continued...**

| | | |
|---|---|---|
| **Shea butter** | **TOXICITY** | **IRRITATION** |
| | Not Available | Not Available |
| ***Legend:*** | *1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances* | |

| | |
|---|---|
| **SUNFLOWER OIL** | 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.<br>For omega 6 fatty acids and deriavtives:<br>Some medical research suggests that excessive levels of certain omega-6 fatty acids relative to certain omega-3 fatty acids may increase the probability of a number of diseases.<br>Modern Western diets typically have ratios of omega-6 to omega-3 in excess of 10 to 1, some as high as 30 to 1; the average ratio of omega-6 to omega-3 in the Western diet is 15:1–16.7:1. Humans are thought to have evolved with a diet of a 1-to-1 ratio of omega-6 to omega-3 and the optimal ratio is thought to be 4 to 1 or lower although some sources suggest ratios as low as 1:1). A ratio of 2–3:1 omega 6 to omega 3 helped reduce inflammation in patients with rheumatoid arthritis. A ratio of 5:1 had a beneficial effect on patients with asthma but a 10:1 ratio had a negative effect. A ratio of 2.5:1 reduced rectal cell proliferation in patients with colorectal cancer, whereas a ratio of 4:1 had no effect.<br>Excess omega-6 fatty acids from vegetable oils interfere with the health benefits of omega-3 fats, in part because they compete for the same rate-limiting enzymes. A high proportion of omega-6 to omega-3 fat in the diet shifts the physiological state in the tissues toward the pathogenesis of many diseases: prothrombotic, proinflammatory and proconstrictive.<br>Chronic excessive production of omega-6 eicosanoids is correlated with arthritis, inflammation, and cancer. Many of the medications used to treat and manage these conditions work by blocking the effects of the COX-2 enzyme. Many steps in formation and action of omega-6 prostaglandins from omega-6 arachidonic acid proceed more vigorously than the corresponding competitive steps in formation and action of omega-3 hormones from omega-3 eicosapentaenoic acid The COX-1 and COX-2 inhibitor medications, used to treat inflammation and pain, work by preventing the COX enzymes from turning arachidonic acid into inflammatory compounds.The LOX inhibitor medications often used to treat asthma work by preventing the LOX enzyme from converting arachidonic acid into the leukotrienes. Many of the anti-mania medications used to treat bipolar disorder work by targeting the arachidonic acid cascade in the brain.<br>A high consumption of oxidised polyunsaturated fatty acids (PUFAs), which are found in most types of vegetable oil, may increase the likelihood that postmenopausal women will develop breast cancer. Similar effect was observed on prostate cancer, but the study was performed on mice Another "analysis suggested an inverse association between total polyunsaturated fatty acids and breast cancer risk, but individual polyunsaturated fatty acids behaved differently [from each other]. [...] a 20:2 derivative of linoleic acid [...] was inversely associated with the risk of breast cancer"<br>PUFAs are prone to spontaneous oxidation/ peroxidation. The feeding of lipid oxidation products and oxidised fats has been reported to cause adverse biological effects on laboratory animals, including growth retardation, teratogenicity, tissue damage and increased liver and kidney weights, as well as cellular damage to the testes and epididymes, increased peroxidation of membrane and tissue lipids and induction of cytochrome P450 activities in the colon and liver.<br>The propensity for PUFAs to oxidise leads to the generation of free radicals and eventually to rancidity.<br>Culinary oils, when heated, undergo important chemical reaction involving self-sustaining, free radical-mediated oxidative deterioration of PUFAs. Such by-products may be cytotoxic, mutagenic, reproductive toxins and may produce chronic disease. Samples of repeatedly used oils collected from fast-food retail outlets and restaurants have confirmed the production of aldehydic lipid oxidation products (LOPs) at levels exceeding 10 exp-2 moles per kilogram (mol/kg) during “on-site” frying episodes. Volatile emissions from heated culinary oils used in Chinese-style cooking are mutagenic; exposure to such indoor air pollution may render humans more susceptible to contracting lung or further cancers, together with rhinitis and diminished lung function. The high temperatures used in standard (especially Chinese) frying result in fumes that are rich in volatile LOPs, including acrolein. The end products of lipid peroxidation are reactive aldehydes, such as malondialdehyde (MDA) and 4-hydroxynonenal (HNE), the second one being known also as "second messenger of free radicals" and major bioactive marker of lipid peroxidation, due to its numerous biological activities resembling activities of reactive oxygen species. end-products of lipid peroxidation may be mutagenic and carcinogenic malondialdehyde reacts with deoxyadenosine and deoxyguanosine in DNA, forming DNA adducts. Malondialdehyde produces mutagenic effects in several bioassays.<br>Side products of lipid peroxidation can also exert toxic effects, even at sites distant from the primary oxidation site. Such products (typically malondialdehyde and a large group of hydroxyalkenals - alpha-beta-unsaturated aldehydes) may interact with protein thiols (producing intermolecular cross-links) and, as a result produce functional impairment to enzyme systems, receptors and structural proteins. Aldehydes may also inhibit protein biosynthesis and increase osmotic fragility of lysosymes (releasing hydrolytic enzymes) and other subcellular organelles. They may also react with nucleic acids.<br>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.<br>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<br>Lipid peroxidation in cellular membranes may produce several morphological alterations resulting, for example, in membrane |

**Continued...**

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.
Epoxidation of double bonds is a common bioactivation pathway for alkenes. The allylic epoxides formed were found to be sensitizing. Research has shown that conjugated dienes in or in conjunction with a six-membered ring are prohaptens, while related dienes containing isolated double bonds or an acrylic conjugated diene were weak or non-sensitising.
Coronaric and vernolic acids also form non-enzymatically when linoleic acid is exposed to oxygen and/or UV radiation as a result of the spontaneous process of autooxidation. This autoxidation complicates studies in that it is often difficult to determine if these epoxy fatty acids identified in linoleic acid-rich plant and mammalian tissues represent actual tissue contents or are artifacts formed during their isolation and detection
At very high concentrations, the linoleic acid-derived set of optical isomers, coronaric acid (i.e. isoleukotoxin), possesses activities similar to that of other structurally unrelated leukotoxins viz., It is toxic to leukocytes and other cell types and when injected into rodents produce multiple organ failure and respiratory distress. These effects appear due to its conversion to its dihydroxy counterparts, 9S,10R- and 9R,10S-dihydroxy-12(Z)-octadecaenoic acids by soluble epoxide hydrolase. Some studies suggest but have not yet proven that isoleukotoxin, acting primarily if not exclusively through its dihydroxy counterparts, is responsible for or contribute to multiple organ failure, the acute respiratory distress syndrome, and certain other cataclysmic diseases in humans (see epoxygenase section on linoleic acid). Vernolic acid (i.e. leukotoxin) shares a similar metabolic fate in being converted by soluble epoxide hydrolase to its dihydroxide counterparts and toxic actions of these hydroxide counterparts.
At lower concentrations, isoleukotoxin and its dihydroxy counterparts can protect from the toxic actions cited above that occur at higher concentrations of isoleukotoxin and leukotoxin; they may also share with the epoxides of arachidonic acid, i.e. the epoxyeicosatreienoates, anti-hypertension activities

The epoxygenases are known to metabolize linoleic acid, at its 12,13 carbon-carbon double bond to form (+) and (-) epoxy optical isomers viz., the 9S,10R-epoxy-12(Z)-octadecaenoic and 9R,10S-epoxy-12(Z)-octadecaenoic acids; this set of optical isomers is also termed vernolic acid, linoleic acid 9:10-oxide, and leukotoxin. Cytochrome P450 (CYP) subtype CYPC2C9 and the other arachidonic acid-metabolizing CYPs are thought to, likewise, attack linoleic acid at its 9,10 carbon-carbon double bound to form 12S,13R-epoxy-9(Z)-octadecaenoic and 12R,13S-epoxy-9(Z)-octadecaenoic acid optical isomers; this set of optical isomers is also termed coronaric acid, linoleic acid 12,13-oxide, and isoleukotoxin. These linoleic acid-derived leukotoxin and isoleukotoxin sets of optical isomers possess activities similar to that of other leukotoxins such as the pore-forming leukotoxin family of RTX toxin virulence factor proteins secreted by gram-negative bacteria, e.g. Aggregatibacter actinomycetemcomitans and E. coli. That is, they are toxic to leukocytes as well as many other cell types and when injected into rodents produce multiple organ failure and respiratory distress. As described above, these effects appear due to the conversion of leukotoxin to its dihydroxy counterparts, 9S,10R- and 9R,10S-dihydroxy-12(Z)-octadecaenoic acids, and isoleukotoxin to its 12R,13S- and 12S,13R-dihydroxy-9(Z)-octadecenoic acid counterparts by soluble epoxide hydrolase. Some studies suggest but have not proven that leukotoxin and isoleukotoxin, acting primarily if not exclusively through their respective dihydroxy counterparts, are responsible for or contribute to multiple organ failure, respiratory distress, and certain other cataclysmic diseases in humans.
Oxidative stress in cells and tissues produces free radical and singlet oxygen oxidations of linoleic acid to generate 13-HpODEs, 9-HpODEs, 13-HODEs, and 9-HODEs; these non-enzymatic reactions produce or are suspected but not proven to produce approximately equal amounts of their S and R stereoisomers. Free radical oxidations of linoleic acid also produce 13-EE-HODE, 9-hydroxy-10E,12-E-octadecadienoic acid, 9-hydroxy-10E,12-Z-octadecadienoic acid, and 11-hydroxy-9Z,12Z-octadecaenoic acid while singlet oxygen attacks on linoleic acid produce (presumably) racemic mixtures of 9-hydroxy-10E,12-Z-octadecadienoic acid, 10-hydroxy-8E,12Z-octadecadienoic acid, and 12-hydroxy-9Z-13-E-octadecadienoic acid. 4-Hydroxynonenal (i.e. 4-hydroxy-2E-nonenal or HNE) is also a peroxidation product of 13-HpODE. Since oxidative stress commonly produces both free radicals and singlet oxygen, most or all of these products may form together in tissues undergoing oxidative stress. Free radical and singlet oxygen oxidations of linoleic acid produce a similar set of 13-HODE metabolites. Studies attribute these oxidations to be major contributors to 13-HODE production in tissues undergoing oxidative stress including in humans sites of inflammation, steatohepatitis, cardiovascular disease-related atheroma plaques, neurodegenerative disease, etc.
For polyunsaturated fatty acids and oils (triglycerides):
Animal studies have shown a link between polyunsaturated fat and the incidence of tumours, which increased with increasing intake of polyunsaturated fats. This may be partly due to the propensity for polyunsaturated fats to oxidize, leading to generation of free radicals.
Research evidence shows that consuming high amounts of polyunsaturated fat may increase the risk of cancer spreading.
Culinary oils, when heated, leads to self-sustaining oxidation f polyunsaturated fatty acids (PUFAs), which may produce oxidation products that are toxic to the cell and reproduction and which may cause mutations and 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) during frying. Volatile emissions from heated culinary oils used in Chinese-style cooking may cause mutations; exposure to such indoor air pollution may make humans more susceptible to contracting lung or other cancers, together with inflammation of the nose, and reduced lung function. The high temperatures used in standard (especially Chinese) frying result in fumes that are rich in volatile LOPs, including acrolein. Shallow frying appears to generate more LOPs than deep frying.
Birth defects: Animal testing shows that LOPs increase the rate of birth defects, whether or not the mother had diabetes. Further investigation reveals that safflower oil subjected to high temperatures markedly increased its propensity to increase birth defects.
Further adverse health effects of LOPs in the diet: Animal testing shows that other documented effects of LOPs include peptic ulcer and high blood pressure.
Atherosclerosis: Investigations have revealed that LOPs derived from the diet can accelerate all three stages of the development of atherosclerosis, including endothelial injury, accumulation of plaque, and thrombosis.
Mutation- and cancer-causing potential: Since they are powerful alkylating agents, alpha,beta-unsaturated aldehydes can covalently modify DNA base units and therefore cause mutations. These LOPs can inactivate DNA replicating systems, a process that can increase the extent of DNA damage.
Malondialdehyde (MDA) is also generated by thermally stressing culinary oils, although at lower concentrations than alpha,beta-unsaturated aldehydes. MDA and other aldehydes arising from lipid peroxidation (especially acrolein) present a serious cancer hazard.
The most obvious solution to the generation of LOPs in culinary oils during frying is to avoid consuming food in PUFA-rich oils as

**Continued...**

| | |
|---|---|
| | much as possible. Consumers and those involved in the fast-food industry could employ culinary oils of only a low PUFA content, or monounsaturated fatty acids (MUFA) such as canola or olive oil, or coconut oils (a saturated fatty acid).<br>Acrylamide (which can exert toxic effects on the nervous system and fertility, and may also cause cancer) can be generated when asparagines-rich foods are deep-fried in PUFA-rich oils.<br>The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin. |
| **SHEA BUTTER** | Polyunsaturated fats (PUFAs) protect against heart disease by providing more membrane fluidity than monounsaturated fats (MUFAs), but they are more vulnerable to being oxidized and therefore rancid.<br>Foods containing monounsaturated fats reduce low-density lipoprotein (LDL) cholesterol, while possibly increasing high-density lipoprotein (HDL) cholesterol.<br>Levels of oleic, and other monounsaturated fatty acids in red blood cell membranes were positively associated with breast cancer risk. In children, consumption of monounsaturated oils is associated with healthier blood lipid profiles.<br>The diet in Mediterranean countries consists of more total fat than the diets of Northern European countries, but most of the fat is made up of monounsaturated fatty acids from olive oil and omega-3 fatty acids (PUFAs) from fish and vegetables, and very little saturated fat.<br>This product contains partially hydrogenated fatty acids and/ or trans fatty acids.<br>The consumption of trans fats increases the risk of coronary heart disease by raising levels of LDL cholesterol and lowering levels of "good" HDL cholesterol. There is an ongoing debate about a possible differentiation between trans fats of natural origin and trans fats of man-made origin but so far no scientific consensus has been found. Two Canadian studies have shown that the natural trans fat vaccenic acid, found in beef and dairy products, may have an opposite health effect and could actually be beneficial compared to hydrogenated vegetable shortening, or a mixture of pork lard and soy fat, by lowering total and LDL cholesterol and triglyceride levels. In lack of recognized evidence and scientific agreement, nutritional authorities consider all trans fats as equally harmful for health and recommend that consumption of trans fats be reduced to trace amounts.<br>The use of hydrogenated oils in foods has never been completely satisfactory. Because the center arm of the triglyceride is shielded somewhat by the end fatty acids, most of the hydrogenation occurs on the end fatty acids,<br>While full hydrogenation produces largely saturated fatty acids, partial hydrogenation results in the transformation of unsaturated cis fatty acids to trans fatty acids in the oil mixture due to the heat used in hydrogenation. Partially hydrogenated oils and their trans fats have increasingly been viewed as "unhealthy".<br>Trans fat is the common name for unsaturated fat with trans-isomer (E-isomer) fatty acid(s). Because the term refers to the configuration of a double carbon-carbon bond, trans fats are sometimes monounsaturated or polyunsaturated, but never saturated. Trans fats do exist in nature but also occur during the processing of polyunsaturated fatty acids in food production.<br>Trans fats occur naturally in a limited number of cases: vaccenyl and conjugated linoleyl (CLA) containing trans fats occur naturally in trace amounts in meat and dairy products from ruminants.<br>The exact biochemical methods by which trans fats produce specific health problems are a topic of continuing research. One theory is that the human lipase enzyme works only on the cis configuration and cannot metabolise a trans fat. A lipase is a water-soluble enzyme that helps digest, transport, and process dietary lipids such as triglycerides, fats, and oils in most - if not all - living organisms. While the mechanisms through which trans fats contribute to coronary heart disease are fairly well understood, the mechanism for trans fat's effect on diabetes is still under investigation. Trans fatty acids may impair the metabolism of long-chain polyunsaturated fatty acids (LCPUFAs), but maternal pregnancy trans fatty acid intake has been inversely associated with LCPUFAs levels in infants at birth thought to underlie the positive association between breastfeeding and intelligence.<br>There are suggestions that the negative consequences of trans fat consumption go beyond the cardiovascular risk. In general, there is much less scientific consensus asserting that eating trans fat specifically increases the risk of other chronic health problems:<br>It has been suggested that the intake of both trans fats and saturated fats promote the development of Alzheimer disease, although not confirmed in an animal model. It has been found that trans fats impaired memory and learning in middle-age rats. The rats' brains of trans-fat eaters had fewer proteins critical to healthy neurological function. Inflammation in and around the hippocampus, the part of the brain responsible for learning and memory. These are the exact types of changes normally seen at the onset of Alzheimer's, but seen after six weeks, even though the rats were still young.<br>There is a growing concern that the risk of type 2 diabetes increases with trans fat consumption.[52] However, consensus has not been reached. For example, one study found that risk is higher for those in the highest quartile of trans fat consumption.Another study has found no diabetes risk once other factors such as total fat intake and BMI were accounted for.<br>Research indicates that trans fat may increase weight gain and abdominal fat, despite a similar caloric intake. A 6-year experiment revealed that monkeys fed a trans fat diet gained 7.2% of their body weight, as compared to 1.8% for monkeys on a mono-unsaturated fat diet. Although obesity is frequently linked to trans fat in the popular media, this is generally in the context of eating too many calories; there is not a strong scientific consensus connecting trans fat and obesity, although the 6-year experiment did find such a link, concluding that "under controlled feeding conditions, long-term TFA consumption was an independent factor in weight gain. TFAs enhanced intra-abdominal deposition of fat, even in the absence of caloric excess, and were associated with insulin resistance, with evidence that there is impaired post-insulin receptor binding signal transduction.<br>Liver Dysfunction: Trans fats are metabolised differently by the liver than other fats and interfere with delta 6 desaturase. Delta 6 desaturase is an enzyme involved in converting essential fatty acids to arachidonic acid and prostaglandins, both of which are important to the functioning of cells.<br>Infertility in women: One 2007 study found, "Each 2% increase in the intake of energy from trans unsaturated fats, as opposed to that from carbohydrates, was associated with a 73% greater risk of ovulatory infertility...".<br>Major depressive disorder: Spanish researchers analysed the diets of 12,059 people over six years and found those who ate the most trans fats had a 48 per cent higher risk of depression than those who did not eat trans fats. One mechanism may be trans-fats' substitution for docosahexaenoic acid (DHA) levels in the orbitofrontal cortex (OFC). Very high intake of trans-fatty acids (43% of total fat) in mice from 2 to 16 months of age was associated with lowered DHA levels in the brain (p=0.001) When the brains of 15 major depressive subjects who had committed suicide were examined post-mortem and compared against 27 age-matched controls, the suicidal brains were found to have 16% less (male average) to 32% (female average) less DHA in the OFC. The OFC is known to control reward, reward expectation and empathy, which are all negatively impacted in depressive mood disorders, as well as regulating the limbic system><br>Eye Irritation: Practically non-irritating; Low irritation potential (Draize Test). Skin Irritation: Non-irritating to human skin. Mildly |

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| | |
|---|---|
| | irritating (In vitro). Sensitization: Not a skin sensitizer. Mutagenicity: Negative in mutagenicity assays. Non-phototoxic. Sheanut oil is generally recognized as safe (GRAS) in direct food substances. It is used in as a cocoa butter substitute in confections and frostings, coatings of soft candy, and sweet sauces and toppings. It is also used as a margarine or shortening. Components of shea extracts have potential anti-inflammatory, antioxidant, and anti-tumour effects. In a 13-week rat feeding study, shea oleine or hydrogenated shea oleine did not produced adverse effects. No reproductive effects were observed in rats fed shea oleine or hydrogenated shea oleine for up to 20 weeks. No tumorgenic potential or adverse effects to shea oleine in a carcinogenicity study in the offspring of the rats from the reproductive study. Butyrospermum Parkii (Shea) Butter was not a dermal or ocular irritant or dermal sensitizer in non-human studies. This ingredient was not a dermal irritant or sensitizer in human studies at concentrations up to 60%. Butyrospermum Parkii (Shea) Butter was not phototoxic in a guinea pig study. |
| **SUNFLOWER OIL & SHEA BUTTER** | For aliphatic fatty acids (and salts)<br>Acute oral (gavage) toxicity:<br>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.<br>Skin and eye irritation potential, with a few stated exceptions, is chain length dependent and decreases with increasing chain length<br>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.<br>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.<br>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.<br>Eye irritation potential of the ammonium salts does not follow chain length dependence; the C18 ammonium salts are corrosive to the eyes.<br>Dermal absorption:<br>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.<br>Sensitisation:<br>No sensitisation data were located.<br>Repeat dose toxicity:<br>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. .<br>Mutagenicity<br>Aliphatic acids do not appear to be mutagenic or clastogenic in vitro or in vivo<br>Carcinogenicity<br>No data were located for carcinogenicity of aliphatic fatty acids.<br>Reproductive toxicity<br>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.<br><br>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.<br>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).<br>Metabolism:<br>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.<br>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.<br>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).<br>Hence, the situation will be similar for compounds originating from acids and therefore no differences in uptake are anticipated<br>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..<br>Toxicokinetics:<br>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 |

**Continued...**

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.
No significant acute toxicological data identified in literature search.
For group E aliphatic esters (polyol esters):
The polyol esters, including trimethylolpropane (TMP). Pentaerythritol (PE) and dipentaerythritol (diPE) are unique in their chemical characteristics since they lack beta-tertiary hydrogen atoms, thus leading to stability against oxidation and elimination. Therefore their esters with C5-C10 fatty acids have applications as artificial lubricants. Because of their stability at high temperatures, they are also used in 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 lubricating properties.
Acute toxicity: Animal studies show relatively low toxicity by swallowing. These esters are hydrolysed in the gastrointestinal tract, and studies have not shown evidence of these accumulating in body tissues. Acute toxicity by skin contact was also found to be low.
Repeat dose toxicity: According to animal testing, polyol esters show a low level of toxicity following repeated application, either by swallowing or by skin contact.
Reproductive and developmental toxicity: This group should not produce profound reproductive effects in animals.
Genetic toxicity: Tests have shown this group to be inactive. It is unlikely that these substances cause mutations.
Cancer-causing potential: No association between this group of substances and cancer.

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

| | Endpoint | Test Duration (hr) | Species | Value | Source |
|---|---|---|---|---|---|
| **Skin Again Ointment** | Not Available | Not Available | Not Available | Not Available | Not Available |

**Continued...**

| | | | | | |
|---|---|---|---|---|---|
| **sunflower oil** | **Endpoint** | **Test Duration (hr)** | **Species** | **Value** | **Source** |
| | Not Available | Not Available | Not Available | Not Available | Not Available |
| **Shea butter** | **Endpoint** | **Test Duration (hr)** | **Species** | **Value** | **Source** |
| | LC50 | 96 | Fish | >=100mg/L | 2 |
| | EC50 | 48 | Crustacea | >100mg/L | 2 |
| | NOEC | 72 | Algae or other aquatic plants | >100mg/L | 2 |
| ***Legend:*** | *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data* | | | | |

**DO NOT** discharge into sewer or waterways.

### Persistence and degradability

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

### Bioaccumulative potential

| Ingredient | Bioaccumulation |
|---|---|
| | No Data available for all ingredients |

### Mobility in soil

| Ingredient | Mobility |
|---|---|
| | No Data available for all ingredients |

## SECTION 13 Disposal considerations

### Waste treatment methods

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

## SECTION 14 Transport information

### Labels Required

| | |
|---|---|
| **Marine Pollutant** | 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

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

Not Applicable

## SECTION 15 Regulatory information

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

**Continued...**

**sunflower oil is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)

**Shea butter is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)

## ECHA SUMMARY

| Ingredient | CAS number | Index No | ECHA Dossier |
|---|---|---|---|
| sunflower oil | 8001-21-6 | Not Available | Not Available |

| Harmonisation (C&L Inventory) | Hazard Class and Category Code(s) | Pictograms Signal Word Code(s) | Hazard Statement Code(s) |
|---|---|---|---|
| 1 | Not Classified | Not Available | Not Available |

*Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.*

| Ingredient | CAS number | Index No | ECHA Dossier |
|---|---|---|---|
| Shea butter | 68920-03-6 | Not Available | 01-2119937263-39-XXXX |

| Harmonisation (C&L Inventory) | Hazard Class and Category Code(s) | Pictograms Signal Word Code(s) | Hazard Statement Code(s) |
|---|---|---|---|
| 1 | Not Classified | Not Available | Not Available |
| 1 | Not Classified | Not Available | Not Available |
| 1 | Not Classified | Not Available | Not Available |

*Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.*

## National Inventory Status

| National Inventory | Status |
|---|---|
| Australia - AIIC | Yes |
| Australia Non-Industrial Use | No (sunflower oil; Shea butter) |
| Canada - DSL | Yes |
| Canada - NDSL | No (sunflower oil) |
| China - IECSC | Yes |
| Europe - EINEC / ELINCS / NLP | Yes |
| Japan - ENCS | No (sunflower oil; Shea butter) |
| Korea - KECI | Yes |
| New Zealand - NZIoC | Yes |
| Philippines - PICCS | Yes |
| USA - TSCA | Yes |
| Taiwan - TCSI | Yes |
| Mexico - INSQ | No (sunflower oil; Shea butter) |
| Vietnam - NCI | Yes |
| Russia - ARIPS | No (Shea butter) |
| ***Legend:*** | *Yes = All CAS declared ingredients are on the inventory*<br>*No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)* |

## SECTION 16 Other information

| | |
|---|---|
| **Revision Date** | 07/09/2020 |
| **Initial Date** | 07/09/2020 |

## SDS Version Summary

| Version | Issue Date | Sections Updated |
|---|---|---|
| 2.1.1.1 | 07/09/2020 | Supplier Information |

**Continued...**

## Other information

### Ingredients with multiple cas numbers

| Name | CAS No |
|---|---|
| sunflower oil | 8001-21-6, 68555-64-6 |
| Shea butter | 68920-03-6, 91080-23-8, 194043-92-0 |

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

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

### Definitions and abbreviations

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