Ranvet	Chemwatch Hazard Alert Code: 2
Chemwatch: 4787-83	Issue Date: 12/10/2021
Version No: 8.1	Print Date: 07/18/2022
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements	L.GHS.AUS.EN.E

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

### **Product Identifier**

Product name	Ranvet's Filybol
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	Non-virilising anabolic combination for fillies, mares, colts and stallions.	

## Details of the supplier of the safety data sheet

Registered company name	Ranvet
Address	10-12 Green Street Banksmeadow NSW 2019 Australia
Telephone	+61 2 9666 1744
Fax	+61 2 9666 1755
Website	http://www.ranvet.com.au/other_msds.htm
Email	info@ranvet.com.au

#### Emergency telephone number

Association / Organisation	Ranvet
Emergency telephone numbers	+61 425 061 584
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.

## ChemWatch Hazard Ratings

	Min	Max	
Flammability	1		
Toxicity	1 📃		0 = Minimum
Body Contact	1 📃		1 = Low
Reactivity	1 📃		2 = Moderate
Chronic	2		3 = High 4 = Extreme

Poisons Schedule	S4
Classification <sup>[1]</sup>	Carcinogenicity Category 2, Reproductive Toxicity Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 2
Legend:	1. Classified by Chernwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

#### Label elements



Signal word

Warning

### Hazard statement(s)

H351	Suspected of causing cancer.
H361f	Suspected of damaging fertility.

H411 Toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention	
P201	Obtain special instructions before use.
P280	Wear protective gloves and protective clothing.
P273	Avoid release to the environment.

### Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P391	Collect spillage.

### Precautionary statement(s) Storage

P405 Store locked up.

P501

#### Precautionary statement(s) Disposal

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

Not Applicable

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

#### Substances

See section below for composition of Mixtures

## Mixtures

CAS No	%[weight]	Name
8002-03-7	<100	peanut oil
360-70-3	<10	nandrolone decanoate
3593-85-9	<10	methandriol dipropionate
Not Available	balance	Ingredients determined not to be hazardous
Legend:	1. Classified by Chemwatch; 2. C Classification drawn from C&L * I	lassification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. EU IOELVs available

#### **SECTION 4 First aid measures**

Description of first aid measures		
Eye Contact	<ul> <li>If this product comes in contact with the eyes:</li> <li>Wash out immediately with fresh running water.</li> <li>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.</li> <li>Seek medical attention without delay; if pain persists or recurs seek medical attention.</li> <li>Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.</li> </ul>	
Skin Contact	<ul> <li>If skin contact occurs:</li> <li>Immediately remove all contaminated clothing, including footwear.</li> <li>Flush skin and hair with running water (and soap if available).</li> <li>Seek medical attention in event of irritation.</li> </ul>	
Inhalation	<ul> <li>If fumes, aerosols or combustion products are inhaled remove from contaminated area.</li> <li>Other measures are usually unnecessary.</li> </ul>	
Ingestion	<ul> <li>If swallowed do NOT induce vomiting.</li> <li>If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.</li> <li>Observe the patient carefully.</li> <li>Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.</li> <li>Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.</li> <li>Seek medical advice.</li> </ul>	

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

Treat symptomatically.

- TREATMENT for androgen-anabolic steroid intoxication:
- For acute massive ingestions of androgenic-anabolic steroids, decontamination of the gastrointestinal tract with oral administration of activated charcoal may reduce absorption if employed within 1 or 2 hours of ingestion.
- Activated charcoal is used empirically as a general adsorbent, because there is no evidence to support or refute its efficacy.
- Because there appears to be minimal risk from ingesting pharmacological doses of androgen-anabolic steroids, gastrointestinal decontamination should not be indicated in these cases.
- + For intramuscular injections of androgenic-anabolic steroids, cleansing and examination of the injection site would be advisable.
- Complications associated with androgenic-anabolic steroid use may require symptomatic treatment, such as lorazepam, diazepam, or haloperidol for excessively aggressive and combative patients.
- There are no specific antagonists for acute or chronic exposure to androgen-anabolic steroids. Discontinuance of further use should be emphasised.
- Management of a withdrawal syndrome associated with these steroids have been described but the risks and benefits of such an approach have not been reported. CLINICAL TOXICOLOGY: Ford, Delaney, Ling and Erickson: 2001 Publisher: W.B. Saunders Company

## 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		
Advice for firefighters			
Fire Fighting	<ul> <li>Alert Fire Brigade and tell them location and nature of hazard.</li> <li>Wear breathing apparatus plus protective gloves.</li> <li>Prevent, by any means available, spillage from entering drains or water courses.</li> <li>Use water delivered as a fine spray to control fire and cool adjacent area.</li> <li>DO NOT approach containers suspected to be hot.</li> <li>Cool fire exposed containers with water spray from a protected location.</li> <li>If safe to do so, remove containers from path of fire.</li> <li>Equipment should be thoroughly decontaminated after use.</li> </ul>		
Fire/Explosion Hazard	<ul> <li>Combustible.</li> <li>Slight fire hazard when exposed to heat or flame.</li> <li>Heating may cause expansion or decomposition leading to violent rupture of containers.</li> <li>On combustion, may emit toxic fumes of carbon monoxide (CO).</li> <li>May emit acrid smoke.</li> <li>Mists containing combustible materials may be explosive.</li> <li>Combustion products include:</li> <li>carbon dioxide (CO2)</li> <li>acrolein</li> <li>other pyrolysis products typical of burning organic material.</li> <li>May emit corrosive fumes.</li> <li>CARE: Water in contact with hot liquid may cause foaming and a steam explosion with wide scattering of hot oil and possible severe burns.</li> </ul>		
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	<ul> <li>Slippery when spilt.</li> <li>Remove all ignition sources.</li> <li>Clean up all spills immediately.</li> <li>Avoid breathing vapours and contact with skin and eyes.</li> <li>Control personal contact with the substance, by using protective equipment.</li> <li>Contain and absorb spill with sand, earth, inert material or vermiculite.</li> <li>Wipe up.</li> <li>Place in a suitable, labelled container for waste disposal.</li> </ul>
Major Spills	<ul> <li>Slippery when spilt.</li> <li>CARE: Absorbent materials wetted with occluded oil must be moistened with water as they may auto-oxidize, become self heating and ignite.</li> <li>Some oils slowly oxidise when spread in a film and oil on cloths, mops, absorbents may autoxidise and generate heat, smoulder, ignite and burn.</li> <li>In the workplace oily rags should be collected and immersed in water.</li> <li>Moderate hazard.</li> <li>Clear area of personnel and move upwind.</li> <li>Alert Fire Brigade and tell them location and nature of hazard.</li> <li>Wear breathing apparatus plus protective gloves.</li> <li>Prevent, by any means available, spillage from entering drains or water course.</li> <li>No smoking, naked lights or ignition sources.</li> <li>Increase ventilation.</li> <li>Stop leak if safe to do so.</li> <li>Contain spill with sand, earth or vermiculite.</li> <li>Collect recoverable product into labelled containers for recycling.</li> <li>Absorb remaining product with sand, earth or vermiculite.</li> <li>Collect solid residues and seal in labelled drums for disposal.</li> <li>Wash area and prevent runoff into drains.</li> <li>If contamination of drains or waterways occurs, advise emergency services.</li> </ul>

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

## **SECTION 7 Handling and storage**

Precautions for safe handling	
Safe handling	• DO NOT allow clothing wet with material to stay in contact with skin 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

	<ul> <li>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.</li> <li>Avoid all personal contact, including inhalation.</li> <li>Wear protective clothing when risk of exposure occurs.</li> <li>Use in a well-ventilated area.</li> <li>Prevent concentration in hollows and sumps.</li> <li>DO NOT enter confined spaces until atmosphere has been checked.</li> <li>Avoid contact with incompatible materials.</li> <li>When handling, DO NOT eat, drink or smoke.</li> <li>Keep containers securely sealed when not in use.</li> <li>Avoid physical damage to containers.</li> <li>Always wash hands with soap and water after handling.</li> <li>Work clothes should be laundered separately.</li> <li>Use good occupational work practice.</li> <li>Observe manufacturer's storage and handling recommendations contained within this SDS.</li> <li>Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.</li> </ul>
Other information	<ul> <li>Store in original containers.</li> <li>Keep containers securely sealed.</li> <li>No smoking, naked lights or ignition sources.</li> <li>Store in a cool, dry, well-ventilated area.</li> <li>Store away from incompatible materials and foodstuff containers.</li> <li>Protect containers against physical damage and check regularly for leaks.</li> <li>Observe manufacturer's storage and handling recommendations contained within this SDS.</li> </ul>
Conditions for safe storage, in	cluding any incompatibilities
Suitable container	<ul> <li>Glass container is suitable for laboratory quantities</li> <li>Metal can or drum</li> <li>Packaging as recommended by manufacturer.</li> <li>Check all containers are clearly labelled and free from leaks.</li> </ul>
Storage incompatibility	<ul> <li>Materials soaked with plant/ vegetable derived (and rarely, animal) oils may undergo spontaneous combustion</li> <li>The more unsaturated is the fatty acid component, the more susceptible is the oil to oxidation and spontaneous combustion.</li> <li>Many vegetable and animal oils absorb oxygen from the air to form oxidation products. This oxidation process produces heat and the resultant increase in temperature accelerates the oxidation process.</li> <li>Drying oils such as linseed, tung, poppy and sunflower oils and semi-drying oils such as soya bean, tall oil, corn, cotton and castor oils all absorb oxygen readily and thus experience the self-heating process.</li> <li>Cotton fibres are readily ignited and if contaminated with an oxidisable oil, may ignite unless heat can be dissipated</li> <li>Vegetable oils and some animal fats undergo undesirable deterioration reactions in the presence of oxygen from the air becoming rancid accompanying off-flavours and smells.</li> <li>The mechanism of autoxidation of vegetable oils is classically regarded as following a number of stages being: <ul> <li>a usually slow initiation phase</li> <li>a usually rapid propagation</li> <li>and a termination phase</li> </ul> </li> <li>The initiation phase involves the formation of a free radical from a triglyceride molecule in the fat: this may be promoted by the presence of heavy metals in the oil, or by heat or light. The next stage is the reaction of the triglyceride free radical. Steps 2 and 3 can repeat in a chain reaction until two peroxy free radicals collide and neutralise each other.</li> <li>Some drying oils produce cyclic peroxides instead of hydroperoxides.</li> <li>Autoxidation may also occur in saturated fatty acids and their esters. Monohydroperoxides are formed. Although all carbon atoms are subject to oxidation, preferential oxidation appears to occur towards the centre of the molecule.</li> <li>Autoxidation is assisted by higher ambient temperatures (the rate doubling for every ten degrees Centigrade rise) a</li></ul>

anti-oxidants, which can act as free-radical inhibitors. Vegetable oils should therefore be stored in a cool place away from heat and light, and should only come into contact with inert (glass of stainless steel) containers which will not leach heavy metals. Blanketing under nitrogen should be considered in bulk storages.

Avoid reaction with oxidising agents

### **SECTION 8 Exposure controls / personal protection**

#### **Control parameters**

INGREDIENT DATA

```
Not Available
```

Ingredient

Notes:

Emergency Limits

Ingredient	TEEL-1	TEEL-2		TEEL-3
Ranvet's Filybol	Not Available	Not Available		Not Available
Ingredient	Original IDLH		Revised IDLH	
peanut oil	Not Available		Not Available	
nandrolone decanoate	Not Available		Not Available	
methandriol dipropionate	Not Available		Not Available	

### Occupational Exposure Banding

I Exposure	Band	Rating
	I Exposure	I Exposure Band

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.

**Occupational Exposure Band Limit** 

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit	
peanut oil	E	≤ 0.1 ppm	
nandrolone decanoate	E	≤ 0.01 mg/m³	
methandriol dipropionate	E	≤ 0.01 mg/m³	
Notes:	Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.		

#### MATERIAL DATA

Sensory irritants are chemicals that produce temporary and undesirable side-effects on the eyes, nose or throat. Historically occupational exposure standards for these irritants have been based on observation of workers' responses to various airborne concentrations. Present day expectations require that nearly every individual should be protected against even minor sensory irritation and exposure standards are established using uncertainty factors or safety factors of 5 to 10 or more. On occasion animal no-observable-effect-levels (NOEL) are used to determine these limits where human results are unavailable. An additional approach, typically used by the TLV committee (USA) in determining respiratory standards for this group of chemicals, has been to assign ceiling values (TLV C) to rapidly acting irritants and to assign short-term exposure limits (TLV STELs) when the weight of evidence from irritation, bioaccumulation and other endpoints combine to warrant such a limit. In contrast the MAK Commission (Germany) uses a five-category system based on intensive odour, local irritation, and elimination half-life. However this system is being replaced to be consistent with the European Union (EU) Scientific Committee for Occupational Exposure Limits (SCOEL); this is more closely allied to that of the USA.

OSHA (USA) concluded that exposure to sensory irritants can:

- cause inflammation
- cause increased susceptibility to other irritants and infectious agents
- lead to permanent injury or dysfunction
- permit greater absorption of hazardous substances and
- ▶ acclimate the worker to the irritant warning properties of these substances thus increasing the risk of overexposure.

#### for androgenic anabolic steroid

IARC Group 2A - probable human carcinogen

Cases of benign hepatoma, peliosis hepatitis, primary hepatocellular carcinoma and hepatic cholangiocarcinoma have been associated with the use of steroidal androgens, notably oxymetholone.

#### Exposure controls

	None required when handling small quantities. <b>OTHERWISE:</b> <b>Care:</b> Atmospheres in bulk storages and even apparently en before entry. Requirements of State Authorities concerning conditions for work permits; sampling of atmosphere; provision of rescue h Engineering controls are used to remove a hazard or place a be highly effective in protecting workers and will typically be The basic types of engineering controls are: Process controls which involve changing the way a job activit Enclosure and/or isolation of emission source which keeps a "adds" and "removes" air in the work environment. Ventilation ventilation system must match the particular process and ch Employers may need to use multiple types of controls to pre-	apply tanks may be hazardous by oxygen depletion. Atmosph tank entry must be met. Particularly with regard to training o arness and protective gear as needed barrier between the worker and the hazard. Well-designed independent of worker interactions to provide this high level ty or process is done to reduce the risk. selected hazard "physically" away from the worker and ven n can remove or dilute an air contaminant if designed proper emical or contaminant in use. vent employee overexposure.	ere must be checked f crews for tank entry; engineering controls can of protection. tilation that strategically rly. The design of a		
	Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. An approved self contained breathing apparatus (SCBA) may be required in some situations. Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.				
	Type of Contaminant:	Air Speed:			
Appropriate engineering controls	solvent, vapours, degreasing etc., evaporating from tank (i	0.25-0.5 m/s (50-100 f/min.)			
	aerosols, fumes from pouring operations, intermittent cont drift, plating acid fumes, pickling (released at low velocity i	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 ge very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)			
	Within each range the appropriate value depends on:				
	Lower end of the range	Upper end of the range			
	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents			
	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity	-		
	3: Intermittent, low production.	3: High production, heavy use			
	4: Large hood or large air mass in motion	4: Small hood-local control only			
	Simple theory shows that air velocity falls rapidly with distance with the square of distance from the extraction point (in simp accordingly, after reference to distance from the contaminati 1-2 m/s (200-400 f/min) for extraction of solvents generated producing performance deficit within the extraction apparent	ce away from the opening of a simple extraction pipe. Veloci le cases). Therefore the air speed at the extraction point sho ng source. The air velocity at the extraction fan, for example in a tank 2 meters distant from the extraction point. Other m is make it essential that theoretical air velocities are multipl	ty generally decreases build be adjusted, , should be a minimum of echanical considerations, ied by factors of 10 or		



more when extraction systems are installed or used.

Ranvet's	Filvbol
11011101010	

Eye and face protection	<ul> <li>No special equipment for minor exposure i.e. when handling small quantities.</li> <li>OTHERWISE:</li> <li>Safety glasses with side shields.</li> <li>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]</li> </ul>
Skin protection	See Hand protection below
Hands/feet protection	<ul> <li>Wear general protective gloves, eg. light weight rubber gloves.</li> <li>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</li> <li>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</li> <li>Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</li> <li>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: <ul> <li>requency and duration of contact,</li> <li>ohemical resistance of glove material,</li> <li>glove thickness and</li> </ul> </li> <li>dexterity</li> </ul> <li>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</li> <li>When only brief contact is expected, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.</li> <li>When only burief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.</li> <li>Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.</li> <li>Contaminated gloves should be replaced.</li> <li>As defined in ASTM F-739-96 in any application, gloves are rated as: <ul> <li>Excellent when breakthrough time &lt; 20 min</li> <li>Fair when breakthrough time &lt; 20 min</li> <li>F</li></ul></li>
Body protection	See Other protection below
Other protection	<ul> <li>Overalls.</li> <li>Barrier cream</li> <li>Eyewash unit.</li> </ul>

### **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

## Information on basic physical and chemical properties

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

## **SECTION 10 Stability and reactivity**

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

## **SECTION 11 Toxicological information**

### Information on toxicological effects

Inhaled	The material is not thought to produce either adverse health effects or irritation of the respiratory tract following inhalation (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting. Not normally a hazard due to non-volatile nature of product Inhalation of oil droplets/ aerosols may cause discomfort and may produce chemical pneumonitis. Fine mists generated from plant/ vegetable (or more rarely from animal) oils may be hazardous. Extreme heating for prolonged periods, at high temperatures, may generate breakdown products which include acrolein and acrolein-like substances.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. Side-effects associated with both natural and synthetic androgens include increases in nitrogen retention and skeletal weight, sodium, calcium, potassium, inorganic phosphates, and water retention, oedema, increased vascularisation of the skin, hypercalcaemia, and increased bone growth. Large and repeated doses in early puberty may cause closure of the epiphyses and stop linear growth. Jaundice, cholestatic hepatitis and abnormal liver-function tests may occur. Liver effects occur mainly with 17alpha alkylated derivatives. Other effects may include hypersensitivity characterised by skin manifestations and anaphylactoid reactions. In men, large doses suppress spermatogenesis and cause degenerative changes in seminiferous tubules. Excessive stimulation may be evidence of excessive doses especially in elder men. In women androgens inhibit the activity of the anterior pituitary which results in the suppression of ovarian activity and menstruation. Continued administration of large doses may produce symptoms of virilism including male-pattern hirsutism, deepening of the voice, atrophy of the breasts and endometrial tissue, acne, seborrhea, and hypertrophy of the clitoris. Libido may be increased and lactation suppressed.
Skin Contact	<ul> <li>The material may produce moderate skin irritation; limited evidence or practical experience suggests, that the material either:</li> <li>produces moderate inflammation of the skin in a substantial number of individuals following direct contact and/or</li> <li>produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period.</li> <li>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.</li> <li>Open cuts, abraded or irritated skin should not be exposed to this material</li> <li>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects.</li> <li>Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</li> </ul>
Eye	Although the liquid is not thought to be an irritant (as classified by EC Directives), direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn).

levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects.

are not secondary non-specific consequences of the other toxic effects

On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in

Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose

Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or

There is some evidence that human exposure to the material may result in developmental toxicity. This evidence is based on animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which

High doses of the androgens over prolonged periods may produce hepatic tumours. Androgens may also accelerate growth of malignant neoplasms of the prostate. The prolonged use of 17alpha alkylated derivatives in particular has been associated with the development of hepatic adenomas and hepatocellular carcinoma. In pregnant women, androgens may cause virilisation of the external genitalia of the female foetus. Large and repeated doses of androgens in early puberty may cause closure of the epiphyses (growth areas at the end of long bones) and prevent linear growth. Elderly males may become over-stimulated. In females, the inhibitory action of testosterone on the activity of the pituitary results in suppression of ovarian activity. In men, large doses suppress spermatogenesis and cause degenerative changes in the seminiferous tubules creating a form of sterility. Androgens may accelerate the growth of malignant tumours of the prostate. Liver cancers (hepatic

respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.

angiosarcoma) have developed in patients undergoing androgen therapies for prolonged periods. This effect has also been noted amongst body builders self-administering anabolic steroids. Peliosis hepatitis, a condition in which liver and sometimes splenic tissue is replaced with blood-filled cysts, has been reported in patients receiving androgenic steroid therapy. These cysts are sometimes present with minimal hepatic dysfunction but at other times they have been associated with liver failure. They are often not recognised until life-threatening liver failure or intra-abdominal haemorrhage develops. Glyceryl triesters (triglycerides), following ingestion, are metabolised to monoglycerides, free fatty acids and glycerol, all of which are absorbed in the intestinal mucosa and undergo further metabolism. Medium chain triglycerides (C8-C10) appear to have relatively rapid metabolism and elimination from blood and tissues compared to long chain triglycerides (C16-C18). Little or no acute, subchronic or chronic oral toxicity was seen in animal studies unless levels approached a significant percentage of calorific intake. Subcutaneous injections of tricaprylin in rats over a five-week period caused granulomatous reaction characterised by oil deposits surrounded by macrophages. Diets containing substantial levels of tributyrin produced gastric lesions in rats fed for 3-35 weeks; the irritative effect of the substance was thought to be the cause of tissue damage. Dermal application was not associated with significant irritation in rabbit skin; ocular exposures were, at most, mildly irritating to rabbit eyes. No evidence of sensitisation or photosensitisation was seen in a guinea pig maximisation test. Most of the genotoxicity test systems were negative. Tricaprylin, trioctanoin and triolein have been used, historically, as vehicles in carcinogenicity testing of other chemicals. In one study, subcutaneous injection of tricaprylin, in newborn mice, produced more tumours in lymphoid tissue than were seen in untreated animals whereas, in another study, subcutaneous or intraperitoneal injection in 4- to 6-week old female mice produced no tumours. Trioctanoin injected subcutaneously in hamster produced no tumours; when injected intraperitoneally in pregnant rats there was an increase in mammary tumours among the off-spring but similar studies in pregnant hamsters and rabbits showed no tumours in the off-spring. The National Toxicological Program conducted a 2-year study in rats given tricaprylin by gavage. The treatment was associated with a statistically significant dose-related increase in pancreatic acinar cell hyperplasia and adenoma but there were no acinar carcinomas. Tricaprylin is not teratogenic to mice or rats but some reproductive effects were seen in rabbits. A low level of foetal eye abnormalities and a small percentage of abnormal sperm were reported in mice injected with trioctanoin. Trioctanoin was also used as a vehicle control in a sperm abnormality test. Ten male control mice received an intraperitoneal injection of 0.25 ml trioctanoin 0.05 g/kg of benz[a]pyrene (known reproductive toxicant and mutagen) daily for 5 days and sperm from caudae epididymides analysed. Based on these studies there is no sufficient evidence to classify the trioctanoin as reproductive toxicant. In the human body, high levels of triglycerides in the bloodstream have been linked to atherosclerosis, heart disease and stroke. However, the relative negative impact of raised levels of triglycerides compared to that of LDL:HDL ratios is as yet unknown. The risk can be partly accounted for by a strong inverse relationship between triglyceride level and HDL-cholesterol level. But the risk is also due to high triglyceride levels increasing the quantity of small, dense LDL particles Synthetic 1,2-diglycerides of short chain (C6, C8, C10) fatty acids are activators of protein kinase C (PKC). PKC is a serine-threonine kinase which also requires calcium ion for its activation. Activated PKC phosphorylates proteins of the cellular signal cascade, which eventually induce expression of growth regulatory genes. This, in turn, may promote the growth of tumours. Structural analogues of the 1,2-diglycerides, such as the phorbol esters, have been shown to strongly promote such an event. In biochemical signaling, diacylglycerol (DAG) functions as a second messenger signaling lipid, and is a product of the hydrolysis of the phospholipid PIP2 (phosphatidylinositolbisphosphate) by the enzyme phospholipase C (PLC) (a membrane-bound enzyme) that, through the same reaction, produces inositol trisphosphate (IP3). Although inositol trisphosphate (IP3) diffuses into the cytosol, DAG remains within the plasma membrane due to its hydrophobic properties. IP3 stimulates the release of calcium ions from the smooth endoplasmic reticulum, whereas DAG is a physiological activator of protein kinase C (PKC). The production of DAG in the membrane facilitates translocation of PKC from the cytosol to the plasma membrane. Glyceryl dilaurate, glyceryl diarachidate, glyceryl dibehenate, glyceryl dierucate, glyceryl dihydroxystearate, glyceryl diisopalmitate, glyceryl disostearate, glyceryl dilinoleate, glyceryl dimyristate, glyceryl dioleate, glyceryl diricinoleate, glyceryl dipalmitate, glyceryl dipalmitoleate, glyceryl distearate, glyceryl palmitate lactate, glyceryl stearate citrate, glyceryl stearate lactate, and glyceryl stearate succinate are diacylglycerols (also known as DAGs, diglycerides or glyceryl diesters) that function as skin conditioning agents-emollients in cosmetics. Only glyceryl dilaurate (up to 5%), glyceryl diisostearate (up to 43%), glyceryl dioleate (up to 2%), glyceryl distearate (up to 7%), and glyceryl stearate lactate (up to 5%) are reported to be in current use. Production proceeds from fully refined vegetable oils, which are further processed using hydrogenation and fractionation techniques, and the end products are produced by reacting selected mixtures of the partly hydrogenated, partly fractionated oils and fats with vegetable-derived glycerine to yield partial glycerides. In the final stage of the production process, the products are purified by deodorization, which effectively removes pesticide residues and lower boiling residues such as residues of halogenated solvents and aromatic solvents. Diglycerides have been approved by the Food and Drug Administration (FDA) for use as indirect food additives. Nominally, these ingredients are 1,3-diglycerides, but are easily isomerised to the 1,2-diglycerides form. The 1,3-diglyceride isomer is not a significant toxicant in acute, short-term, subchronic, or chronic animal tests. Glyceryl dilaurate was a mild primary irritant in albino rabbits, but not a skin sensitiser in guinea pig maximization tests. Diacylglycerol oil was not genotoxic in the Ames test, in mammalian Chinese hamster lung cells, or in a rodent bone marrow micronucleus assay. An eye shadow containing 1.5% glyceryl dilaurate did not induce skin irritation in a single insult patch test, but mild skin irritation reactions to a foundation containing the same concentration were observed. A trade mixture containing an unspecified concentration of glyceryl dibehenate did not induce irritation or significant cutaneous intolerance in a 48-h occlusive patch test. In maximization tests, neither an eye shadow nor a foundation containing 1.5% glyceryl dilaurate was a skin sensitiser. Sensitisation was not induced in subjects patch tested with 50% w/w glyceryl dioleate in a repeated insult, occlusive patch test. Glyceryl palmitate lactate (50% w/v) did not induce skin irritation or sensitization in subjects patch tested in a repeat-insult patch test. Phototoxicity or photoallergenicity was not induced in healthy volunteers tested with a lipstick containing 1.0% Glyceryl rosinate. Two diacylglycerols, 1-oleoyl-2-acetoyl-sn-glycerol and 1,2-dipalmitoylsn-glycerol, did not alter cell proliferation (as determined by DNA synthesis) in normal human dermal fibroblasts in vitro at doses up to 10 µg/ml. In the absence of initiation, Glyceryl distearate induced a moderate hyperplastic response in randomly bred mice of a tumor-resistant strain, and with 9,10-dimethyl-1,2-benzanthracene (DMBA) initiation, an increase in the total cell count was observed. In a glyceryl monoester study, a single application of DMBA to the skin followed by 5% glyceryl stearate twice weekly produced no tumors, but slight epidermal hyperplasia at the site of application. Glyceryl dioleate induced transformation in 3-methylcholanthrene-initiated BALB/3T3 A31-1-1 cloned cells in vitro. A tumourpromoting dosing regimen that consisted of multiple applications of 10 µmol of a 1,2-diacylglycerol (sn-1,2-didecanoylglycerol) to female mice twice daily for 1 week caused more than a 60% decrease in protein kinase C (PKC) activity and marked epidermal hyperplasia. Applications of 10 umol sn-1,2-didecanoylglycerol twice weekly for 1 week caused a decrease in cytosolic PKC activity, an increase in particulate PKC activity, and no epidermal hyperplasia. In studies of the tumour-promoting activity of 1,2-diacylglycerols, dose and the exposure regimen by which the dose is delivered play a role in tumor promotion. The 1,2-diacylglycerol-induced activation of PKC may also relate to the saturation of the fatty acid in the 1 or 2 position; 1,2-Diacylglycerols with two saturated fatty acids are less effective. Also, the activity of 1,2-diacylglycerols may be reduced when the fatty acid mojety in the structure is a long-chain fatty acid. A histological evaluation was performed on human skin from female volunteers (18 Continued...

Chronic

biochemical systems.

to 56 years old) who had applied a prototype lotion or placebo formulation, both containing 0.5% Glyceryl Dilaurate, consecutively for 16 weeks or 21 weeks. Skin irritation was not observed in any of the subjects tested. Biopsies (2 mm) taken from both leas of five subjects indicated no recognizable abnormalities of the skin: the epidermis was normal in thickness, and there was no evidence of scaling, inflammation, or neoplasms in any of the tissues that were evaluated. The available safety test data indicate that diglycerides in the 1,3-diester form do not present any significant acute toxicity risk, nor are these ingredients irritating, sensitizing, or photosensitising. Whereas no data are available regarding reproductive or developmental toxicity, there is no reason to suspect any such toxicity because the dermal absorption of these chemicals is negligible. 1,3-Diglycerides contain 1,2-diglycerides, raising the concern that 1,2-diglycerides could potentially induce hyperplasia. Data regarding the induction of PKC and the tumour promotion potential of 1,2-diacylglycerols increases the level of concern. Most of the diglycerides considered above, however, have fatty acid chains longer than 14 carbons and none have mixed saturated/unsaturated fatty acid moieties. In a 21-week use study of a prototype lotion containing 0.5% glyceryl dilaurate (a 14-carbon chain fatty acid) indicated no evidence of scaling, inflammation, or neoplasms in biopsy specimens. Also, DNA synthesis assays on glyceryl dilaurate and glyceryl distearate indicated that neither chemical altered cell proliferation (as determined by DNA synthesis) in normal human dermal fibroblasts in vitro at doses up to 10 ug/ml. However the concentration of these ingredients can vary (up to 43% for glyceryl diisostearate in lipstick), the frequency of application can be several times daily, and the proportion of diglycerides that are inactive 1,3 isomers versus potentially biologically active 1,2 isomers is unknown; as a precaution it is believed that each use should be examined to ensure the absence of epidermal hyperplasia during product development and testing. In the absence of inhalation toxicity data on the glyceryl diesters it is thought that these ingredients can be used safely in aerosolised products because they are not respirable. Although there are gaps in knowledge about product use, the overall information available on the types of products in which these ingredients are used and at what concentration indicate a pattern of use. Within this overall pattern of use, the CIR Expert Panel considers all ingredients in this group to be safe.

International Journal of Toxicology, Vol. 26, No. 3 Suppl, 1-30 (2007)

Nandrolone may cause cholestatic jaundice, virilisation, oedema, congestive heart failure, iron deficiency anaemia, hypercalcaemia, muscle cramps, gastrointestinal disturbances, retention of fluids, excitation, chills and toxic confusion. Chronic effects of exposure include peliosis hepatitis and blood lipid changes.

Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).

Ranvet's Filybol	TOXICITY Not Available	IRRITATION Not Available
	ΤΟΧΙΟΙΤΥ	IRRITATION
peanut oil	Not Available	Skin (human) : 300 mg/3d I - mild Skin (rabbit):100 mg/24h-moderate
nandrolone decanoate	ΤΟΧΙΟΙΤΥ	IRRITATION
	Not Available	Not Available
methandriol dipropionate	τοχιςιτγ	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

Equivocal tumorigenic agent by RTECS criteria. Highly refined peanut oil is different from peanuts, peanut butter, and peanut flour when it comes to allergy. This is because most peanut oil undergoes a refining process, in which it is purified, refined, bleached, and deodorized. When peanut oil is correctly processed and becomes highly refined, the proteins in the oil, which are the components in the oil that can cause allergic reaction, are removed. Sixty two adults with peanut allergy where given refined peanut oil (up to 10 ml). None of the volunteers had allergic reactions to refined peanut oil. But crude unrefined peanut oil did cause reactions in 6 out of the 62 peanut allergic volunteers. These reactions were not severe. Theoretically it is possible that traces of peanut protein which cannot cause a reaction would nevertheless sensitive someone, or increase the sensitivity of someone who is already allergic.

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.

PEANUT OIL

but they are more vulnerable to lipid peroxidation (rancidity). On the other hand, some monounsaturated fatty acids (in the same way as saturated fatts) 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. 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).

For omega 6 fatty acids and derivatives:

Some medical research suggests that excessive levels of certain omega-6 fatty acids relative to certain omega-3 fatty acids may increase the probability of a number of diseases.

Modern Western diets typically have ratios of omega-6 to omega-3 in excess of 10 to 1, some as high as 30 to 1; the average ratio of omega-6 to omega-3 in the Western diet is 15:1–16.7:1. Humans are thought to have evolved with a diet of a 1-to-1 ratio of omega-6 to omega-3 and the optimal ratio is thought to be 4 to 1 or lower although some sources suggest ratios as low as 1:1). A ratio of 2–3:1 omega 6 to omega 3 helped

pathogenesis of many diseases: prothrombotic, proinflammatory and proconstrictive.

reduce inflammation in patients with rheumatoid arthritis. A ratio of 5:1 had a beneficial effect on patients with asthma but a 10:1 ratio had a negative effect. A ratio of 2.5:1 reduced rectal cell proliferation in patients with colorectal cancer, whereas a ratio of 4:1 had no effect. Excess omega-6 fatty acids from vegetable oils interfere with the health benefits of omega-3 fats, in part because they compete for the same rate-limiting enzymes. A high proportion of omega-6 to omega-3 fat in the diet shifts the physiological state in the tissues toward the

Chronic excessive production of omega-6 eicosanoids is correlated with arthritis, inflammation, and cancer. Many of the medications used to treat and manage these conditions work by blocking the effects of the COX-2 enzyme. Many steps in formation and action of omega-6 prostaglandins from omega-6 arachidonic acid proceed more vigorously than the corresponding competitive steps in formation and action of omega-3 hormones from omega-3 eicosapentaenoic acid The COX-1 and COX-2 inhibitor medications, used to treat inflammation and pain, work by preventing the COX enzymes from turning arachidonic acid into inflammatory compounds. The LOX inhibitor medications often used to treat asthma work by preventing the LOX enzyme from converting arachidonic acid into the leukotrienes. Many of the anti-mania medications used to treat bipolar disorder work by targeting the arachidonic acid cascade in the brain. A high consumption of oxidised polyunsaturated fatty acids (PUFAs), which are found in most types of vegetable oil, may increase the likelihood that postmenopausal women will develop breast cancer. Similar effect was observed on prostate cancer, but the study was performed on mice Another "analysis suggested an inverse association between total polyunsaturated fatty acids and breast cancer risk, but individual polyunsaturated fatty acids behaved differently [from each other]. [...] a 20:2 derivative of linoleic acid [...] was inversely associated with the risk of breast cancer" PUFAs are prone to spontaneous oxidation/ peroxidation. The feeding of lipid oxidation products and oxidised fats has been reported to cause adverse biological effects on laboratory animals, including growth retardation, teratogenicity, tissue damage and increased liver and kidney weights, as well as cellular damage to the testes and epididymes, increased peroxidation of membrane and tissue lipids and induction of cytochrome P450 activities in the colon and liver. The propensity for PUFAs to oxidise leads to the generation of free radicals and eventually to rancidity. Culinary oils, when heated, undergo important chemical reaction involving self-sustaining, free radical-mediated oxidative deterioration of PUFAs. Such by-products may be cytotoxic, mutagenic, reproductive toxins and may produce chronic disease. Samples of repeatedly used oils collected from fast-food retail outlets and restaurants have confirmed the production of aldehydic lipid oxidation products (LOPs) at levels exceeding 10 exp-2 moles per kilogram (mol/kg) during "on-site" frying episodes. Volatile emissions from heated culinary oils used in Chinese-style cooking are mutagenic; exposure to such indoor air pollution may render humans more susceptible to contracting lung or further cancers, together with rhinitis and diminished lung function. The high temperatures used in standard (especially Chinese) frying result in fumes that are rich in volatile LOPs, including acrolein. The end products of lipid peroxidation are reactive aldehydes, such as malondialdehyde (MDA) and 4-hydroxynonenal (HNE), the second one being known also as "second messenger of free radicals" and major bioactive marker of lipid peroxidation, due to its numerous biological activities resembling activities of reactive oxygen species. end-products of lipid peroxidation may be mutagenic and carcinogenic malondialdehyde reacts with deoxyadenosine and deoxyguanosine in DNA, forming DNA adducts. Malondialdehyde produces mutagenic effects in several bioassavs Side products of lipid peroxidation can also exert toxic effects, even at sites distant from the primary oxidation site. Such products (typically malondialdehyde and a large group of hydroxyalkenals - alpha-beta-unsaturated aldehydes) may interact with protein thiols (producing intermolecular cross-links) and, as a result produce functional impairment to enzyme systems, receptors and structural proteins. Aldehydes may also inhibit protein biosynthesis and increase osmotic fragility of lysosymes (releasing hydrolytic enzymes) and other subcellular organelles. They may also react with nucleic acids. The toxicity of lipid hydroperoxides to animals is best illustrated by the lethal phenotype of glutathione peroxidase 4 (GPX4) knockout mice. These animals do not survive past embryonic day 8, indicating that the removal of lipid hydroperoxides is essential for mammalian life. Peroxidised linoleic acid applied to the shaved skin of guinea pigs, in a patch test experiment, produced necrosis and bleeding. When the abdominal skin of guinea pig was patched for 8 days with a cream containing 25 nmol (in terms of malondialdehyde) of lipid peroxides per gram, a thickening of the epidermis was found Lipid peroxidation in cellular membranes may produce several morphological alterations resulting, for example, in membrane aggregation, deformation or breakage. This may result in the release of hydrolytic enzymes which in turn may degrade functional macromolecules and cause secondary damage. In addition membrane-bound enzyme systems may be disrupted. For 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 a treatment sites. Microscopically, treated skin (viz., greater than or equal to 500 mg/kg/day) exhibited a dose-related increased incidence and severity of hyperplasia and hyperkeratosis of the epidermis and sebaceous gland hyperplasia. These effects were reversible. None of the minor changes in haematology and serum chemistry parameters were considered biologically significant. High dose females (2000 mg/kg/day) exhibited a significant increase in relative adrenal and brain weights when compared to the controls. These differences were attributed to the lower final body weight of the female animals. The NOAEL in this study for systemic toxicity was established as 500 mg /kg/day and 125 mg/kg/day for skin irritation.

Two 28-day study conducted with fatty acids, C5-10, esters with pentaerythritol (CAS RN: 68424-31-7) and dipentaerythritol ester of n-C5/iso-C9 acids (CAS RN: 647028-25-9) showed no signs of overt toxicity. The 90-day study pentaerythritol ester of pentanoic acids and isononanoic acid (CAS RN: 146289-36-3) did not show any signs of overt toxicity. However, increased kidney and liver weights in the male animals was observed. In conclusion, since the effects observed are not considered to be systemic and relevant for humans, the NOAEL was found to exceed 1000 mg/kg bw for all substances based on the result from the 28 and 90-day studies.

Reproductive and developmental toxicity: Since metabolism of the polyol esters can occur, leading to the generation of the corresponding fatty acids and the polyol alcohol (such as pentaerthyritol, trimethylolpropane, and dipentaerythritol), the issue of whether these metabolites may pose any potential reproductive/developmental toxicity concerns is important. However, the polyol alcohols such as pentaerthyritol, trimethylolpropane, and dipentaerythritol, such as pentaerthyritol, trimethylolpropane, and dipentaerythritol, would be expected to undergo further metabolism, conjugation and excretion in the urine. Available evidence indicates that these ester hydrolysates (i.e., hydrolysis products), primarily fatty acids (e.g., heptanoic, octanoic, and decanoic acids) and secondarily the polyol alcohols should exhibit a low order of reproductive toxicity. It can be concluded that this group of high molecular weight polyol esters should not produce profound reproductive effects in rodents.

Genotoxicity: Polyols tested for genetic activity in the Salmonella assay, have been found to be inactive. Several polyol esters have been adequately tested for chromosomal mutation in the in vitro mammalian chromosome aberration assay, and all were inactive. Two TMP esters were also tested for in vivo chromosomal aberration in rats, and both demonstrated no activity. Thus, it is unlikely that these substances are chromosomal mutagens.

**Carcinogenicity:** In a 2-yr study, 28 male and 28 female rats were fed 5% polyglyceryl ester in the diet. No adverse effects on body weight, feed consumption, haematology values, or survival rate were noted. Liver function tests and renal function tests performed at 59 and 104 wks of the study were comparable between the test group and a control group fed 5% ground nut oil. The carcass fat contained no polyglycerol, and the levels of free fatty acid, unsaponifiable residue and fatty acid composition of carcass fat were not different from the controls. Organ weights, tumour incidence and tumour distribution were similar in control and test groups. A complete histological examination of major organs showed nothing remarkable

For polyunsaturated fatty acids and oils (triglycerides)

Studies on animals have shown a link between polyunsaturated fat and the incidence of tumours. In some of these studies the incidence of tumours increased with increasing intake of polyunsaturated fat, up to about 5% of total energy, near to the middle of the current dietary intake in humans.

The propensity for polyunsaturated fats to oxidise is another possible risk factor. This leads to the generation of free radicals and eventually to rancidity

Research evidence suggests that consuming high amounts of polyunsaturated fat may increase the risk of cancer spreading. Researchers found that linoleic acid in polyunsaturated fats produced increasing membrane phase separation, and thereby increased adherence of circulating tumour cells to blood vessel walls and remote organs.

At least one study in mice has shown that consuming high amounts of polyunsaturated fat (but not monounsaturated fat) may increase the risk of metastasis in cancer.

Lipid peroxides with complex components can damage macromolecules, such as DNA, proteins, and membrane lipids. Some components of lipid peroxides, for example, 4,5(E)-epoxy-2(E)-heptenal (EH) can react with L-lysine and damage proteins . 4,5-epoxy-2-alkenals can react with phenylalanine and cause strecker-type degradation of amino acids. Autoxidized methyl linoleate can decrease DNA synthesis in thymocytes Animals consuming oxidized lipids suffered a wide array of biological consequences, such as decreased feed utilization and performance, oxidative stress and tissue lipid oxidation and, most strikingly, adverse effects on redox indices and shelf life of meat. This manifested in malondialdehyde (MDA) content reduced activities of antioxidant enzymes and elevated transcript levels of oxidative stress-responsive genes The intestinal mucosa is directly exposed to oxidized fatty acids of dietary origin and this tissue readily experiences redox imbalances and oxidative stress after the ingestion of large amounts of oxidized fat . As the first line of defense, the intestinal with abundant gut-associated lymphoid tissues (GALTs) and lymphocytes play an important role in immune defense. The immune response in the intestinal tract is complex and is impaired by any damage to the mucosal barrier. When oxidative stress of the intestines caused by oxidized fat occurs, its immune competence and responsiveness may be compromised by the peroxides they contain

When body insulin levels are low, fatty acids flow from the fat cells into the bloodstream and are taken up by various cells and metabolised in a process called beta-oxidation. The end result of beta-oxidation is a molecule called acetyl-coA, and as more fatty acids are released and metabolised, acetyl-coA levels in the cells rise. Liver cells shunt excess acetyl-coA into "ketogenesis", or the making of ketone bodies. When the rate of synthesis of ketone bodies exceeds the rate of utilisation, their concentration in blood increases; this is known as ketonaemia. This is followed by ketonuria – excretion of ketone bodies in urine. The overall picture of ketonaemia and ketonuria is commonly referred as ketosis. Smell of acetone in breath is a common feature in ketosis

For polyunsaturated fatty acids and oils (triglycerides), products of heating and recycling.\*

Culinary oils, when heated, undergo important chemical reaction involving self-sustaining, free radical-mediated oxidative deterioration of polyunsaturated fatty acids (PUFAs). Such by-products may be cytotoxic, mutagenic, reproductive toxins and may produce chronic disease. Saturated fatty acid (SFA)-rich fats also undergo such reactions but to a substantially lower degree.

Samples of repeatedly used oils collected from fast-food retail outlets and restaurants have confirmed the production of aldehydic lipid oxidation products (LOPs, active aldehydes) at levels exceeding 10 exp-2 moles per kilogram (mol/kg) during "on-site" frying episodes. Volatile emissions from heated culinary oils used in Chinese-style cooking are mutagenic; exposure to such indoor air pollution may render humans more susceptible to contracting lung or further cancers, together with rhinitis and diminished lung function. The high temperatures used in standard (especially Chinese) frying result in fumes that are rich in volatile LOPs, including acrolein.

Teratogenic actions. In principle, if aldehydic LOPs induce DNA and chromosomal damage during embryo development, foetal malformations may arise. A study was conducted to investigate the ability of the chain-breaking antioxidant a-tocopherol (a-TOH, vitamin E) to prevent the teratogenic effects of uncontrolled diabetes mellitus in rats (a study based on the hypothesis that diabetic animals have an elevated level of oxidative stress and therefore in vivo lipid peroxidation when expressed relative to that of healthy controls). It found that a PUFA-rich culinary oil (which served as a vehicle for oral administration of a-TOH) increased the rate of malformations and reabsorptions in both normal and diabetic pregnancies. Further investigations revealed that safflower oil subjected to thermal stressing episodes (according to standard frying practices for a period of 20 minutes) markedly enhanced its teratogenic effects. That is, the evidence indicates that the LOPs therein are primarily responsible for these actions.

Further adverse health effects of dietary LOPs. Further documented health effects of LOPs include their pro-inflammatory and gastropathic properties (for the latter, oral administration of the LOP, 4-hydroxy-trans-2-nonenal -HNE- to rats at a dose level of only 0.26 umol-dm-3, a level similar to that of healthy human blood plasma, induced peptic ulcers), and also a significant elevation in systolic blood pressure and an impaired vasorelaxation observed in rats fed pre-heated soy oil

Oxidative degradation process involving culinary oils, can generate extremely toxic conjugated lipid hydroperoxydienes (CHPDs). These are unstable at standard frying temperatures (ca. 180 degrees C) and are degraded to a broad range of secondary products, particularly saturated and unsaturated aldehydes, together with di- and epoxyaldehydes. Such aldehydic fragments also have toxicological properties in humans owing to their high reactivity with critical biomolecules in vivo (proteins such as low-density lipoprotein, amino acids, thiols such as glutathione, DNA, etc.). Despite their reactivities, high levels of CHPDs can remain in PUFA-rich oils which have been subjected to routine frying practices. Thermally stressed PUFA-containing culinary oils contain high levels of alpha,beta-unsaturated aldehydes (including trans-2-alkenals, and cis,trans- and trans,trans-1,4-denals, 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 cytotxic agents are metabolized by a process involving the primary addition (Michael addition reaction) of glutathione across their electrophilic carbon-carbon double bonds and finally excreted in the urine as C-3 mercapturate derivatives. \* Martin Grootveld, Victor Ruiz Rodado, and Christopher J.L. Silwood

Detection, monitoring, and deleterious health effects of lipid oxidation products generated in culinary oils during thermal stressing episodes American Oil Chemists Society, 25 (10), pp. 614-624. November/December 2014

The material may 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.

PEANUT OIL & METHANDRIOL DIPROPIONATE

No significant acute toxicological data identified in literature search.

Page 13 of 16 Ranvet's Filybol

NANDROLONE DECANOATE & METHANDRIOL DIPROPIONATE	Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis). for androgenic anabolic steroid IARC Group 2A - probable human carcinogen Cases of benign hepatoma, peliosis hepatitis, primary hepatocellular carcinoma and hepatic cholangiocarcinoma have been associated with the use of steroidal androgens, notably oxymetholone.		
Acute Toxicity	×	Carcinogenicity	✓
Skin Irritation/Corrosion	×	Reproductivity	✓
Serious Eye Damage/Irritation	×	STOT - Single Exposure	×
Respiratory or Skin sensitisation	×	STOT - Repeated Exposure	×
Mutagenicity	×	Aspiration Hazard	×
Logand: 🔰 – Data aither not available or doos not fill the criteria for classification			

Data available to make classification

### **SECTION 12 Ecological information**

#### Toxicity Endpoint Test Duration (hr) Species Value Source Ranvet's Filvbol Not Not Not Not Available Not Available Available Available Available Endpoint Test Duration (hr) Species Value Source peanut oil Not Not Not Not Available Not Available Available Available Available Endpoint Test Duration (hr) Species Value Source nandrolone decanoate Not Not Not Not Available Not Available Available Available Available Test Duration (hr) Species Endpoint Value Source methandriol dipropionate Not Not Not Not Available Not Available Available Available Available Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Legend: Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan)

- Bioconcentration Data 8. Vendor Data

Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

Unsaturated vegetable oils are often used in paints which upon "drying" produce a polymeric network formed of the constituent fatty acids.

During the drying process, a number of compounds are produced that do not contribute to the polymer network. These include unstable hydroperoxide (ROOH) the major by-product of the reaction of oxygen with unsaturated fatty acids. The hydroperoxides quickly decompose, forming carbon dioxide and water, as well as a variety of aldehydes, acids and hydrocarbons. Many of these compounds are volatile, and in an unpigmented oil, they would be quickly lost to the environment. However, in paints, such volatiles may react with lead, zinc, copper or iron compounds in the pigment, and remain in the paint film as coordination complexes or salts. A large number of the original ester bonds in the oil molecules undergo hydrolysis releasing individual fatty acids. Some portion of the free fatty acids react with metals in the pigment, producing metal carboxylates. Together, the various non-cross-linking substances associated with the polymer network constitute the mobile phases. Unlike the molecules that are part of the network itself, they are capable of moving and diffusing within the film, and can be removed using heat or a solvent. The mobile phase may play a role in plasticising the paint film, preventing it from becoming too brittle.

One simple technique for monitoring the early stages of the drying process is to measure weight change in an oil film over time. Initially, the film becomes heavier, as it absorbs large amounts of oxygen. Then oxygen uptake ceases, and the weight of the film declines as volatile compounds are lost to the environment.

As the oil ages, a further transition occurs. Carboxyl groups in the polymers of the stationary phase lose a hydrogen ion, becoming negatively charged, and form complexes with metal cations present in the pigment. The original network, with its nonpolar, covalent bonds is replaced by an ionomeric structure, held together by ionic interactions. At present, the structure of these ionomeric networks is not well understood.

DO NOT discharge into sewer or waterways.

#### Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
nandrolone decanoate	HIGH	HIGH
methandriol dipropionate	HIGH	HIGH

#### **Bioaccumulative potential**

Ingredient	Bioaccumulation
nandrolone decanoate	LOW (LogKOW = 7.7499)
methandriol dipropionate	HIGH (LogKOW = 6.9385)

#### Mobility in soil

Ingredient	Mobility
nandrolone decanoate	LOW (KOC = 1137000)
methandriol dipropionate	LOW (KOC = 344500)

### **SECTION 13 Disposal considerations**

Waste treatment methods	
Product / Packaging disposal	<ul> <li>Containers may still present a chemical hazard/ danger when empty.</li> <li>Return to supplier for reuse/ recycling if possible.</li> <li>Otherwise:</li> <li>If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.</li> <li>Where possible retain label warnings and SDS and observe all notices pertaining to the product.</li> <li>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</li> <li>A Hierarchy of Controls seems to be common - the user should investigate:</li> <li>Reduction</li> <li>Reuse</li> <li>Recycling</li> <li>Disposal (if all else fails)</li> <li>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be appropriate.</li> <li>DO NOT allow wash water from cleaning or process equipment to enter drains.</li> <li>It may be necessary to collect all wash water for treatment before disposal.</li> <li>In all cases disposal to sever may be subject to local laws and regulations and these should be considered first.</li> <li>Where in doubt contact the responsible authority.</li> <li>Recycle wherever possible or consult manufacturer for recycling options.</li> <li>Consult State Land Waste Authority of disposal.</li> <li>Bury or incinerate residue at an approved site.</li> </ul>
	r Recycle containers it possible, of dispose of in an autoorised jandtill

### **SECTION 14 Transport information**

### Labels Required

Marine Pollutant	
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

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

Product name	Group
peanut oil	Not Available
nandrolone decanoate	Not Available
methandriol dipropionate	Not Available

### Transport in bulk in accordance with the ICG Code

Product name	Ship Type
peanut oil	Not Available
nandrolone decanoate	Not Available
methandriol dipropionate	Not Available

### **SECTION 15 Regulatory information**

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

#### peanut oil is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

### nandrolone decanoate is found on the following regulatory lists

Australia Chemicals with non-industrial uses removed from the Australian Inventory of Chemical Substances (old Inventory)

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

Chemical Footprint Project - Chemicals of High Concern List

methandriol dipropionate is found on the following regulatory lists

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2A: Probably carcinogenic to humans

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

Continued...

Ranvet's Filybol

Australia Chemicals with non-industrial uses removed from the Australian Inventory of Chemical Substances (old Inventory) Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) -

Schedule 4

Chemical Footprint Project - Chemicals of High Concern List

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2A: Probably carcinogenic to humans

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

#### **National Inventory Status**

National Inventory	Status		
Australia - AIIC / Australia Non-Industrial Use	Yes		
Canada - DSL	No (methandriol dipropionate)		
Canada - NDSL	No (peanut oil; nandrolone decanoate; methandriol dipropionate)		
China - IECSC	No (nandrolone decanoate; methandriol dipropionate)		
Europe - EINEC / ELINCS / NLP	Yes		
Japan - ENCS	No (peanut oil; nandrolone decanoate; methandriol dipropionate)		
Korea - KECI	No (nandrolone decanoate; methandriol dipropionate)		
New Zealand - NZIoC	Yes		
Philippines - PICCS	No (nandrolone decanoate; methandriol dipropionate)		
USA - TSCA	No (nandrolone decanoate; methandriol dipropionate)		
Taiwan - TCSI	No (nandrolone decanoate; methandriol dipropionate)		
Mexico - INSQ	No (peanut oil; methandriol dipropionate)		
Vietnam - NCI	No (nandrolone decanoate; methandriol dipropionate)		
Russia - FBEPH	No (nandrolone decanoate; methandriol dipropionate)		
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	12/10/2021
Initial Date	11/01/2009

#### **SDS Version Summary**

Version	Date of Update	Sections Updated
7.1	11/01/2019	One-off system update. NOTE: This may or may not change the GHS classification
8.1	12/10/2021	Classification change due to full database hazard calculation/update.

#### 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 Cancel 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 AIIC: Australian Inventory of Industrial Chemicals DSL: Domestic Substances List NDSL: Non-Domestic Substances List IECSC: Inventory of Existing Chemical Substance in China EINECS: European INventory of Existing Commercial chemical Substances ELINCS: European List of Notified Chemical Substances NLP: No-Longer Polymers ENCS: Existing and New Chemical Substances Inventory KECI: Korea Existing Chemicals Inventory NZIoC: New Zealand Inventory of Chemicals PICCS: Philippine Inventory of Chemicals and Chemical Substances TSCA: Toxic Substances Control Act TCSI: Taiwan Chemical Substance Inventory

INSQ: Inventario Nacional de Sustancias Químicas

NCI: National Chemical Inventory FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

This document is copyright.

Apart from any fair dealing for the purposes of private study, research, review or criticism, as permitted under the Copyright Act, no part may be reproduced by any process without written permission from CHEMWATCH. TEL (+61 3) 9572 4700.