

Callington Haven Pty Ltd

Chemwatch: 5225-93 Version No: 3.1.1.1 Safety Data Sheet according to WHS and ADG requirements Chemwatch Hazard Alert Code: 0

Issue Date: **10/10/2016** Print Date: **15/01/2019** L.GHS.AUS.EN

SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	Fresh & Clean Deodorant Disc
Synonyms	Not Available
Other means of identification	Not Available

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

Relevant identified uses	Air freshener and odour neutraliser.
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Details of the supplier of the safety data sheet

Registered company name	Callington Haven Pty Ltd
Address	30 South Street Rydalmere NSW 2116 Australia
Telephone	+61 2 9898 2700
Fax	+61 2 9475 0449
Website	www.callingtonhaven.com
Email	customerservice@callington.com

Emergency telephone number

Association / Organisation	Chemwatch
Emergency telephone numbers	Not Available
Other emergency telephone numbers	Not Available

CHEMWATCH EMERGENCY RESPONSE

Primary Number	Alternative Number 1	Alternative Number 2
+61 1800 951 288	+61 2 9186 1132	Not Available

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SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification	Not Applicable
Label elements	
Hazard pictogram(s)	Not Applicable
SIGNAL WORD	NOT APPLICABLE

Hazard statement(s)

Not Applicable

Precautionary statement(s) Prevention

Not Applicable

Precautionary statement(s) Response

Not Applicable

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

Not Applicable

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
Not Available		solid white disc impregnated with liquid comprising
25498-49-1	50	tripropylene glycol monomethyl ether
64742-47-8	30	distillates, petroleum, light, hydrotreated
64742-57-0	20	residual oils, petroleum, hydrotreated

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	 If this product comes in contact with eyes: Wash out immediately with water. If irritation continues, seek medical attention. Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	Wash affected areas with warm water and soap. Seek medical attention if swelling/redness/blistering or irritation occurs.
Inhalation	 If fumes, aerosols or combustion products are inhaled remove from contaminated area. Other measures are usually unnecessary.
Ingestion	 Not considered a normal route of entry. Immediately give a glass of water. First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

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

Special hazards arising from the substrate or mixture

Fire Incompatibility	None known
Advice for firefighters	
	Alert Fire Brigade and tell them location and nature of hazard.

- Fire Fighting
 Prevent, by any means available, spillage from e
 - 9 Prevent, by any means available, spillage from entering drains or water courses.
 Use water delivered as a fine spray to control fire and cool adjacent area.

	 DO NOT approach containers suspected to be hot. Cool fire exposed containers with water spray from a protected location. If safe to do so, remove containers from path of fire. Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	Combustible. Will burn if ignited. Decomposes on heating and produces toxic fumes of: carbon monoxide (CO) carbon dioxide (CO2)
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	Sweep up.
Major Spills	 Clean up all spills immediately. Secure load if safe to do so. Bundle/collect recoverable product. Collect remaining material in containers with covers for disposal.

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	No special handling procedures required.	
Other information	 Keep dry. Store under cover. Protect containers against physical damage. Observe manufacturer's storage and handling recommendations contained within this SDS. 	

Conditions for safe storage, including any incompatibilities

Suitable container	Store in original containers.
Storage incompatibility	None known

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure	distillates, petroleum, light,	Oil mist, refined mineral	5	Not	Not	Not
Standards	hydrotreated		mg/m3	Available	Available	Available
Australia Exposure	residual oils, petroleum,	Oil mist, refined	5	Not	Not	Not
Standards	hydrotreated	mineral	mg/m3	Available	Available	Available

EMERGENCY LIMITS

Ingredient	Material name	Material name		TEEL-2	TEEL-3
tripropylene glycol monomethyl ether	Tripropylene glycol monomethyl ether; (1-(2-(2-Methoxy-1-methylethoxy)-1-methylethoxy)-2-propanol)		2 ppm	22 ppm	75 ppm
tripropylene glycol monomethyl ether	Tripropylene glycol methyl ether		9.6 mg/m3	110 mg/m3	630 mg/m3
Ingredient	Original IDLH	Revised IDLH			

tripropylene glycol monomethyl ether	Not Available	Not Available
distillates, petroleum, light, hydrotreated	2,500 mg/m3	Not Available
residual oils, petroleum, hydrotreated	2,500 mg/m3	Not Available

MATERIAL DATA

Exposure controls

Appropriate engineering controls	None under normal operating conditions.
Personal protection	
Eye and face protection	None under normal operating conditions.
Skin protection	See Hand protection below
Hands/feet protection	None under normal operating conditions.
Body protection	See Other protection below
Other protection	None under normal operating conditions.

Respiratory protection

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

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

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

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

^ - Full-face

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

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Solid white disc with lemon fragrance. Contained in a protective plastic case.		
Physical state	Manufactured	Relative density (Water = 1)	Not Applicable
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Applicable	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Applicable	Viscosity (cSt)	Not Applicable
Initial boiling point and boiling range (°C)	Not Applicable	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available
Evaporation rate	Not Applicable	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available

Upper Explosive Limit (%)	Not Applicable	Surface Tension (dyn/cm or mN/m)	Not Applicable
Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Applicable	Gas group	Not Available
Solubility in water	Not Applicable	pH as a solution (1%)	Not Applicable
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

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

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	Not normally a hazard due to physical form of product.
Ingestion	Not normally a hazard due to physical form of product.
Skin Contact	Not normally a hazard due to physical form of product. Irritation and skin reactions are possible with sensitive skin
Eye	Not normally a hazard due to physical form of product.
Chronic	► Generally not applicable.

Fresh & Clean Deodorant	TOXICITY	IRRITATION
Disc	Not Available	Not Available
	TOXICITY	IRRITATION
tripropylene glycol monomethyl ether	Dermal (rabbit) LD50: =15440 mg/kg ^[2]	Not Available
monometryrether	Oral (rat) LD50: 3200 mg/kg ^[2]	
	тохісіту	IRRITATION
distillates, petroleum, light, hydrotreated	dermal (rat) LD50: >2000 mg/kg ^[1]	Not Available
ngnt, nyurotreateu	Oral (rat) LD50: >5000 mg/kg ^[2]	
	тохісіту	IRRITATION
residual oils, petroleum,	Dermal (rabbit) LD50: >2000 mg/kg ^[1]	Not Available
hydrotreated	Inhalation (rat) LC50: >5.3 mg/l4 h ^[1]	
	Oral (rat) LD50: >5000 mg/kg ^[1]	
Legend:		bstances - Acute toxicity 2.* Value obtained from manufacturer's SDS. ECS - Register of Toxic Effect of chemical Substances

TRIPROPYLENE GLYCOL
MONOMETHYL ETHERfor propylene glycol ethers (PGEs):
Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB);
dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM).
Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that
propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated
with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the
developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene
glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The

	reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids.
	Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an
	alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects).
	This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product.
	Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of
	low toxicity and completely metabolised in the body. As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for
	PGEs is via the urine and expired air. A small portion is excreted in the faeces.
	As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m3 for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m3. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m3), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB
	is moderately irritating to skin while the remaining category members are slightly to non-irritating None are skin sensitisers.
	In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying
	histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested).
	Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no
	effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m3 (600 ppm) for PnB and 2,010 mg/m3 (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m3 (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m3 (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave
	similarly to other category members. One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m3) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m3). For
	offspring toxicity the NOAEL is 1000 ppm (3686 mg/m3), with decreased body weights occurring at 3000 ppm (11058 mg/m3). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. in a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health.
	In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs,
	have been reported. Commercially available PGEs showed no teratogenicity. The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. <i>In vitro</i> , negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic <i>in vivo</i> . In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice.
	For "kerosenes"
	Acute toxicity: Oral LD50s for three kerosenes (Jet A, CAS No. 8008-20-6 and CAS No. 64742-81-0) ranged from > 2 to >20 g/kg The dermal LD50s of the same three kerosenes were all >2.0 g//kg. Inhalation LC50 values in Sprague-Dawley rats for straight run kerosene (CAS No. 8008-20-6) and hydrodesulfurised kerosene (CAS No. 64742-81-0) were reported to be > 5 and > 5.2 mg/l, respectively. No mortalities in rats were reported in rats when exposed for eight hours to saturated vapor of deodorised kerosene (probably a desulfurised kerosene). Six hour exposures of cats to the same
DISTILLATES, PETROLEUM, LIGHT, HYDROTREATED	material produced an LC50 of >6.4 mg/l When tested in rabbits for skin irritation, straight run kerosene (CAS No. 8008-20-6) produced "moderate" to "severe" irritation. Six additional skin irritation studies on a range of kerosenes produced "mild" to "severe" irritation. An eye irritation in rabbits of straight run kerosene (CAS No. 8008-20-6) produced Draize scores of 0.7 and 2.0 (unwashed
	and washed eyes) at 1 hour. By 24 hours, the Draize scores had returned to zero. Eye irritation studies have also been reported for hydrodesulfurized kerosene and jet fuel. These materials produced more irritation in the unwashed eyes at 1 hour than had the straight run kerosene. The eye irritation persisted longer than that seen with straight run kerosene, but

hour than had the straight run kerosene. The eye irritation persisted longer than that seen with straight run kerosene, but

by day 7 had resolved.

Straight run kerosene (CAS No. 8008-20-6), Jet A, and hydrodesulfurized kerosene (CAS No. 64742-81-0) have not produced sensitisation when tested in guinea pigs

Repeat-Dose toxicity: Multiple repeat-dose toxicity studies have been reported on a variety of kerosenes or jet fuels. When applied dermally, kerosenes and jet fuels have been shown to produce dermal and systemic effects Dose levels of 200, 1000 and 2000 mg/kg of a straight run kerosene (CAS No. 8008-20-6) were applied undiluted to the skin of male and female New Zealand white rabbits The test material was applied 3x/week for 28 days. One male and one female in the 2000 mg/kg dose group found dead on days 10 and 24 respectively were thought to be treatment-related. Clinical signs that were considered to be treatment-related included: thinness, nasal discharge, lethargy, soiled anal area, anal discharge, wheezing. The high dose group appeared to have a treatment related mean body weight loss when compared to controls. Dose-related skin irritation was observed, ranging from "slight" to "moderate" in the low and high dose groups, respectively. Other treatment-related dermal findings included cracked, flaky and/or leathery skin, crusts and/or hair loss. Reductions in RBC, haemoglobin and haematocrit were seen in the male dose groups. There were no treatment related effects on a variety of clinical chemistry values. Absolute and relative weights for a number of organs were normal, with the following exceptions that were judged to be treatment-related:

• increased relative heart weights for the mid- and high- dose males and females,

· increased absolute and relative spleen weights in treated females, and

• differences in absolute and relative adrenal weights in both male and female treated animals (considered to be stressrelated and therefore, indirectly related to treatment).

Gross necropsy findings were confined largely to the skin. Enlarged spleens were seen in the female groups. Microscopic examination of tissues taken at necropsy found proliferative inflammatory changes in the treated skin of all male and female animals in the high dose group. These changes were, in the majority of animals, accompanied by an increase in granulopoiesis of the bone marrow. Four of six high dose males had testicular changes (multifocal or diffuse tubular hypoplasia) that were considered by the study authors to be secondary to the skin and/or weight changes.

In a different study, hydrodesulfurised kerosene was tested in a thirteen-week dermal study using Sprague-Dawley rats. Test material was applied 5x/week to the skin of male and female rats at dose levels of 165, 330 and 495 mg/kg. Aside from skin irritation at the site of application, there were no treatment-related clinical signs during the study. Screening of all animals using a functional observation battery (FOB) did not find any substance-related effects. Opthalomological examination of all animals also found no treatment-related effects. There were no treatment-related effects on growth rates, hematological or clinical chemical values, or absolute or relative organ weights. Microscopic examination of tissues from animals surviving to termination found no treatment-related changes, with the exception of a minimal degree of a proliferative and inflammatory changes in the skin.

A hydrodesulfurised middle distillate (CAS no. 64742-80-9) has also been tested in a four week inhalation study. In the study, Sprague-Dawley rats were exposed to a nominal concentration of 25mg/m3 kerosene. Exposures were for approximately 6 hr/day, five days each week for four consecutive weeks. There were no treatment-related effects on clinical condition, growth rate, absolute or relative organ weights, or any of the hematological or clinical chemistry determinations. Microscopic examination found no treatment-related changes observed in any tissues.

Carcinogenicity: In addition to the repeat-dose studies discussed above, a number of dermal carcinogenicity studies have been performed on kerosenes or jet fuels. .Following the discovery that hydrodesulfurised (HDS) kerosene caused skin tumors in lifetime mouse skin painting studies, the role of dermal irritation in tumor formation was extensively studied. HDS kerosene proved to be a mouse skin tumor promoter rather than initiator, and this promotion required prolonged dermal irritation . If the equivalent dose of kerosene was applied to the skin in manner that did not cause significant skin irritation (eg, dilution with a mineral oil) no skin tumors occurred . Dermal bioavailability studies in mice confirmed that the reduced irritation seen with samples in mineral oil was not due to decreased skin penetration . The effect of chronic acanthosis on the dermal tumorigenicity of a hydrodesulfurised kerosene was studied and the author concluded that hyperplasia was essential for tumor promotion. However, the author also concluded that subacute inflammation did not appear to be a significant factor

A sample of a hydrodesulfurised kerosene has been tested in an initiation-promotion assay in male CD-1 mice . Animal survivals were not effected by exposure to the kerosene. The study's authors concluded that the kerosene was not an initiator but it did show tumor promoting activity.

In-Vitro (Genotoxicity): The potential *in vitro* genotoxicities of kerosene and jet fuel have been evaluated in a variety of studies. Standard Ames assays on two kerosene samples and a sample of Jet A produced negative results with/without activation. Modified Ames assays on four kerosenes also produced negative results (with/without activation) except for one positive assay that occurred with activation . The testing of five kerosene and jet fuel samples in mouse lymphoma assays produced a mixture of negative and positive results . Hydrodesulfurized kerosene tested in a sister chromatid exchange assay produced negative results (with/without activation)

In-Vivo Genotoxicity: Multiple *in vivo* genotoxicity studies have been done on a variety of kerosene-based materials. Four samples of kerosene were negative and a sample of Jet A was positive in *in vivo* bone marrow cytogenetic tests in Sprague-Dawley rats. One of the kerosene samples produced a positive response in male mice and negative results in females when tested in a sister chromatid exchange assay. Both deodorised kerosene and Jet A samples produced negative results in dominant lethal assays. The kerosene was administered to both mice and rats intraperitoneally, while the jet fuel was administered only to mice via inhalation.

Reproductive/Developmental Toxicity Either 0, 20, 40 or 60% (v/v) kerosene in mineral oil was applied to the skin of the rats. The dose per body weight equivalents were 0, 165, 330 and 494 mg/kg. Test material was applied daily, 7 days/week from 14 days premating through 20 days of gestation. There were no treatment-related effects on mortality and no clinical signs of toxicity were observed. There were no compound-related effects on any of the reproductive/developmental parameters. The authors concluded that the no observable effect level (NOEL) for reproductive/developmental toxicity of HDS kerosene under the treatment conditions of the study was 494 mg/kg/day. Developmental toxicity screening studies on a kerosene and a sample of Jet A have been reported . There were no compound-related deaths in either study. While kerosene produced no clinical signs, the jet fuel produced a dose-related eye irritation (or infection). The signs of irritation lasted from 2 to 8 days with most animals showing signs for 3 days. Neither of the test materials had an effect on body weights or food consumption. Examination of offspring at delivery did not reveal any treatment-related abnormalities, soft tissue changes or skeletal abnormalities. The sex ratio of the fetuses

	was also unaffected by treatment with either of the compounds.
	The materials included in the Lubricating Base Oils category are related from both process and physical-chemical
	perspectives; The potential toxicity of a specific distillate base oil is inversely related to the severity or extent of processing the oil has
	undergone, since: The adverse effects of these materials are associated with undesirable components, and The levels of the undesirable components are inversely related to the degree of processing; Distillate base oils receiving the same degree or extent of processing will have similar toxicities; The potential toxicity of <i>residual base oils</i> is independent of the degree of processing the oil receives. The reproductive and developmental toxicity of the distillate base oils is inversely related to the degree of
	processing. The degree of refining influences the carcinogenic potential of the oils. Whereas mild acid / earth refining processes are inadequate to substantially reduce the carcinogenic potential of lubricant base oils, hydrotreatment and / or solvent
	extraction methods can yield oils with no carcinogenic potential. Unrefined and mildly refined distillate base oils contain the highest levels of undesirable components, have the largest variation of hydrocarbon molecules and have shown the highest potential carcinogenic and mutagenic activities. Highly and severely refined distillate base oils are produced from unrefined and mildly refined oils by removing or transforming
	undesirable components. In comparison to unrefined and mildly refined base oils, the highly and severely refined distillate base oils have a smaller range of hydrocarbon molecules and have demonstrated very low mammalian toxicity. Mutagenicity and carcinogenicity testing of residual oils has been negative, supporting the belief that these materials lack biologically active components or the components are largely non-bioavailable due to their molecular size.
	Toxicity testing has consistently shown that lubricating base oils have low acute toxicities. Numerous tests have shown that a lubricating base oil's mutagenic and carcinogenic potential correlates with its 3-7 ring polycyclic aromatic compound (PAC) content, and the level of DMSO extractables (e.g. IP346 assay), both characteristics that are directly related to the degree/conditions of processing
	Skin irritating is not significant (CONCAWE) based on 14 tests on 10 CASs from the OLBO class (Other Lubricant Base Oils). Each study lasted for 24 hours, a period of time 6 times longer than the duration recommended by the OECD method).
	Eye irritation is not significant according to experimental data (CONCAWE studies) based on 9 "in vivo" tests on 7 CASs from the OLBO class(Other Lubricant Base Oils). Sensitisation: The substance does not cause the sensitization of the respiratory tract or of the skin. (CONCAWE studies
	based on 14 tests on 11 CASs from the OLBO class(Other Lubricant Base Oils)) Germ cell mutagenicity: The tests performed within the 'in vivo" studies regarding gene mutation at mice micronuclei indicated negative results (CONCAWE studies. AMES tests had negative results in 7 studies performed on 4 CASs from the OLBO class(Other Lubricant Base Oils)).
RESIDUAL OILS, PETROLEUM, HYDROTREATED	Reproduction toxicity: Reproduction / development toxicity monitoring according to OECD 421 or 422 methods. CONCAWE tests gave negative results in oral gavage studies. Pre-birth studies regarding toxicity in the unborn foetus development process showed a maternal LOAEL (Lowest Observed Adverse Effect Level) of 125 mg/kg body/day, based on dermal irritation and a NOAEL (No Observable Adverse Effect Level) of 2000 mg/kg body/day, which shows that the
	substance is not toxic for reproduction. STOT (toxicity on specific target organs) – repeated exposure: Studies with short term repeated doses (28-day test) on rabbit skin indicated the NOAEL value of 1000 mg/kg. NOAEL for inhalation, local effects > 280 mg/m3 and for systemic effects NOAEL > 980 mg/m3.
	Sub-chronic toxicity 90-day study Dermal: NOAEL > 2000 mg/kg (CONCAWE studies). Repeat dose toxicity:
	Oral NOAEL for heavy paraffinic distillate aromatic extract could not be identified and is less than 125 mg/kg/day when administered orally. Inhalation
	The NOAEL for lung changes associated with oil deposition in the lungs was 220 mg/m3. As no systemic toxicity was observed, the overall NOAEL for systemic effects was > 980 mg/m3. Dermal
	In a 90 day subchronic dermal study, the administration of Light paraffinic distillate solvent extract had an adverse effect on survivability, body weights, organ weights (particularly the liver and thymus), and variety of haematology and serum chemistry parameters in exposed animals. Histopathological changes which were treatment-related were most prominent in the adrenals, bone marrow, kidneys, liver, lymph nodes, skin, stomach, and thymus. Based on the results of this study, the NOAEL for the test material is less than 30 mg/kg/day. Toxicity to reproduction:
	Mineral oil (a white mineral oil) caused no reproductive or developmental toxicity with 1 mL/kg/day (i.e., 1000 mg/kg/day) in an OECD 421 guideline study, but did cause mild to moderate skin irritation. Therefore, the reproductive/developmental NOAEL for this study is =1000 mg/kg/day and no LOAEL was determined. Developmental toxicity, teratogenicity:
	Heavy paraffinic distillate furfural extract produced maternal, reproductive and foetal toxicity. Maternal toxicity was exhibited as vaginal discharge (dose-related), body weight decrease, reduction in thymus weight and increase in liver weight (125 mg/kg/day and higher) and aberrant haematology and serum chemistry (125 and/or 500 mg/kg/day). Evidence of potential reproductive effects was shown by an increased number of dams with resorptions and intrauterine death. Distillate aromatic extract (DAE) was developmentally toxic regardless of exposure duration as indicated by increased resorptions and decreased foetal body weights. Furthermore, when exposures were increased to 1000 mg/kg/day and
	given only during gestation days 10 through 12, cleft palate and ossification delays were observed. Cleft palate was

considered to indicate a potential teratogenic effect of DAE.

The following Oil Industry Note (OIN) has been applied: OIN 8 - The classifications as a reproductive toxicant category 2; H361d (Suspected of damaging the unborn child) and specific target organ toxicant category 1; H372 (Causes damage to organs through prolonged or repeated exposure) need not apply if the substance is not classified as carcinogenic Toxicokinetics of lubricant base oils has been examined in rodents. Absorption of other lubricant base oils across the small intestine is related to carbon chain length; hydrocarbons with smaller chain length are more readily absorbed than hydrocarbons with a longer chain length. The majority of an oral dose of mineral hydrocarbon is not absorbed and is excreted unchanged in the faeces. Distribution of mineral hydrocarbons following absorption has been observed in liver, fat, kidney, brain and spleen. Excretion of absorbed mineral hydrocarbons occurs via the faeces and urine. Based on the pharmacokinetic parameters and disposition profiles, the data indicate inherent strain differences in the total systemic exposure (~4 fold greater systemic dose in F344 vs SD rats), rate of metabolism, and hepatic and lymph node retention of C26H52, which may be associated with the different strain sensitivities to the formation of liver granulomas and MLN histiocytosis.

for Unrefined and Mildly Refined Distillate Base Oils

Acute toxicity: LD50s of >5000 mg/kg (bw) and >2g/kg (bw) for the oral and dermal routes of exposure, respectively, have been observed in rats dosed with an unrefined light paraffinic distillate The same material was also reported to be "moderately irritating" to the skin of rabbits. When tested for eye irritation in rabbits, the material produced Draize scores of 3.0 and 4.0 (unwashed/washed eyes) at 24 hours, with the scores returning to zero by 48 hours. The material was reported to be "not sensitising" when tested in guinea pigs

Repeat dose toxicity: 200, 1000 and 2000 mg/kg (bw)/day of an unrefined base oil has been applied undiluted to the skin of male and female rabbit. The test material was applied to the rabbits' skins 3 times/week for 4 weeks. To ensure maximum exposure, the applied material was covered with an occlusive dressing for 6 hours. In the high dose group, body weight gains were affected by treatment. These effects were largely due to effects on growth rate during the first week of the study. There were no significant differences between treated and control groups for any of the recorded haematological and clinical chemistry values. Gross and microscopic pathology findings relating to the treated skin were seen in all rabbits in the highest dose group. The findings consisted of "slight" to "moderate" proliferative changes in the treated skin.

Reproductive/ developmental toxicity No reproductive or developmental toxicity studies have been reported for unrefined & mildly refined distillate base oils. However, a developmental toxicity screening study has been reported for heavy vacuum gas oil, a material with a process history similar to the unrefined distillate base oils. As an unrefined vacuum distillate material, heavy vacuum gas oil contains the broadest spectrum of chemical components and highest concentration of bioavailable and/or biologically active components Because of their lack of or low level of processing, in comparison to other refined base oils. the unrefined lubricating base oils will also have higher concentrations of bioavailable and/or biologically active components.

Heavy vacuum gas oil was applied daily to the skin of pregnant rats on days 0-19 of gestation. Dose levels administered included: 30, 125, 500 and 1000 mg/kg (bw)/day. All animals were euthanised on day 20. In the dams, the only dose-related finding at gross necropsy was pale colored lungs in four animals in the highest dose group and in one animal in the 500 mg/kg (bw)/day group. Mean thymus weights of the dams in the highest dose group were approximately half those of the control groups. Although absolute liver weights were unaffected by exposure to the gas oil, mean relative liver weights were increased (approximately 15%) in groups exposed to doses greater than 125 mg/kg (bw)/day. Maternal and foetal body weights were reduced at 500 and 1000 mg/kg (bw)/day. Significant increases in resorptions were also seen in these two dose groups. Soft tissue variations and malformations, and skeletal malformations were also increased at 500 and 1000 mg/kg

Genotoxicity: Modified Ames assays have been carried out on a number of base oils that were either unrefined or poorly refined. The oils were found to be mutagenic, with a strong correlation between mutagenicity and 3-7 ring PAC content. **Carcinogenicity:** The general conclusions that can drawn from the animal carcinogenicity studies are potential skin carcinogens. When applied repeatedly to the skin, carcinogenic base oils are associated only with skin tumours and not with an increase in systemic tumours

Residual Base Oils

Residual oils have substantial polycyclic aromatic compound (PAC) levels when assayed by traditional methods. On this basis, they would be expected to have mutagenic and/or carcinogenic activity. However, no adverse effects have been seen in either in vitro mutagenicity or dermal carcinogenicity testing of residual base oils, irrespective of the degree of processing they have undergone. Ultraviolet, HPLC/UV, GC/MS, and infrared analyses of these oils indicate that the aromatics they contain are predominantly 1-3 rings that are highly alkylated (paraffinic and naphthenic). Because they are found in such a high boiling material (> 550 C), it is estimated that the alkyl side-chains of these 1-3 ring aromatics would be approximately 13 to 25 carbons in length. These highly alkylated aromatic ring materials are either devoid of the biological activity necessary to cause mutagenesis and carcinogenesis, or are largely non-bioavailable to the organisms **Acute toxicity**: There are no acute toxicity data available for the residual base oils. It is thought that the high molecular weight of these materials and associated low bioavailability preclude the systemic doses necessary to produce acute toxicity. Furthermore, tests of a variety of distillate base oils, including unrefined materials that contain high levels of biologically active materials, have consistently shown low acute toxicity.

Repeat dose toxicity: No subchronic repeat-dose studies have been reported on residual base oils. However, two dermal carcinogenicity studies have been performed

Reproductive and developmental toxicity: There are no reproductive or developmental toxicity data available for the residual base oils

Carcinogenicity: A dermal carcinogenicity study of a residual base oil in mice has been reported. The test substance was described as "a non-solvent refined, deasphalted, dewaxed residual paraffinic lubricant base oil". For eighteen months, three times/week, undiluted test material was applied to the skin of female CF1 mice. Two other groups of mice underwent similar treatments, but for only 22 or 52 weeks. The base oil produced minimal clinical evidence of skin irritation. No tumours of epidermal origin were observed in animals dosed with the base oil. Furthermore, no treatment-related effects were observed with regard to clinical condition, body weight gain, mortality or post mortem findings.

A second dermal carcinogenicity study of a residual base oil has been conducted in male C3H/HeJ mice. The test

 substance was described as "deasphalted, dewaxed, residual oil". The test material was ap backs, three times/week for 24 months. None of the animals treated with the test material other tumours considered treatment-related. The absence of systemic toxicity in these two dermal carcinogenicity studies supports th weight of the residual base oils and the resulting low bio- availability preclude the internal systemic toxicity. Genotoxicity: In vitro (mutagenicity): Samples of a vacuum residuum and four residual base oils tested frame shift mutations in modified Ames assays In vivo (chromosomal aberrations): There is no in vivo genotoxicity data available for the vitro mutagenicity studies on these materials have also been negative. Given these consists bioavailability of these materials, it is expected that in vivo mutagenicity tests would also 			test material developed skin tumours, or an supports the belief that the high molecular the internal doses necessary to elicit e oils tested negative for the induction of lable for the residual base oils. However, in produced negative results. Dermal tese consistent results, and the low
DISTILLATES, PETROLEUM, LIGHT, HYDROTREATED & RESIDUAL OILS, PETROLEUM, HYDROTREATED	No significant acute toxicological data identified in literature search.		
Acute Toxicity	×	Carcinogenicity	×
Skin Irritation/Corrosion	×	Reproductivity	×
Serious Eye Damage/Irritation	×	STOT - Single Exposure	×
Respiratory or Skin sensitisation	×	STOT - Repeated Exposure	×
Mutagenicity	×	Aspiration Hazard	×

✓ – Data available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Fresh & Clean Deodorant Disc	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCI
	LC50	96	Fish	11-619mg/L	2
tripropylene glycol monomethyl ether	EC50	48	Crustacea	>10mg/L	1
monometnyr etner	EC50	96	Algae or other aquatic plants	9-69mg/L	2
	NOEC	240	Algae or other aquatic plants	482.5mg/L	2
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURC
	LC50	96	Fish	>1-mg/L	2
distillates, petroleum, light, hydrotreated	EC50	48	Crustacea	>1-mg/L	2
ngin, nyuroneateu	EC50	72	Algae or other aquatic plants	>1-mg/L	2
	NOEC	3072	Fish	=1mg/L	1
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURC
esidual oils, petroleum, hydrotreated	LC50	96	Fish	>100mg/L	2
nyaronculcu	EC50	48	Crustacea	>10-mg/L	2
Legend:	Toxicity 3. EP Data 5. ECE1	WWIN Suite V3.12 (QSAR) - Aqua	ope ECHA Registered Substances - Ecotoxico atic Toxicity Data (Estimated) 4. US EPA, Eco Data 6. NITE (Japan) - Bioconcentration Data	tox database - Aqua	

Prevent, by any means available, spillage from entering drains or water courses. **DO NOT** discharge into sewer or waterways.

Ingredient	Persistence: Water/Soil	Persistence: Air
tripropylene glycol monomethyl ether	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation	
tripropylene glycol monomethyl ether	LOW (LogKOW = -0.2027)	
distillates, petroleum, light, hydrotreated	LOW (BCF = 159)	

Mobility in soil

Ingredient	Mobility
tripropylene glycol monomethyl ether	LOW (KOC = 10)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods Product / Packaging disposal • Recycle wherever possible or consult manufacturer for recycling options. • Consult State Land Waste Management Authority for disposal. • Bury residue in an authorised landfill. • Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 TRANSPORT INFORMATION

Labels Required

Marine Pollutant	NO Not Applicable
HAZCHEM	Not Applicable

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

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

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

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

TRIPROPYLENE GLYCOL MONOMETHYL ETHER(25498-49-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

DISTILLATES, PETROLEUM, LIGHT, HYDROTREATED(64742-47-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards	Australia Standard for the Uniform Scheduling of Medicines and Poisons
Australia Hazardous Chemical Information System (HCIS) - Hazardous	(SUSMP) - Appendix E (Part 2)
Chemicals	Australia Standard for the Uniform Scheduling of Medicines and Poisons
Australia Inventory of Chemical Substances (AICS)	(SUSMP) - Schedule 5
	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

RESIDUAL OILS, PETROLEUM, HYDROTREATED(64742-57-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure StandardsAustralia Inventory of Chemical Substances (AICS)Australia Hazardous Chemical Information System (HCIS) - Hazardous
ChemicalsInternational Agency for Research on Cancer (IARC) - Agents Classified
by the IARC Monographs

National Inventory Status

National Inventory	Status
Australia - AICS	Yes
Canada - DSL	Yes
Canada - NDSL	No (tripropylene glycol monomethyl ether; distillates, petroleum, light, hydrotreated; residual oils, petroleum, hydrotreated)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Legend:	Yes = All ingredients are on the inventory No = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION

Revision Date	10/10/2016
Initial Date	Not Available

SDS Version Summary

Version	lssue Date	Sections Updated
2.1.1.1	09/10/2016	Acute Health (eye), Acute Health (inhaled), Acute Health (skin), Acute Health (swallowed), Advice to Doctor, Chronic Health, Classification, Disposal, Fire Fighter (fire/explosion hazard), Fire Fighter (fire fighting), First Aid (swallowed), Handling Procedure, Ingredients, Physical Properties, Spills (major), Storage (storage incompatibility), Name
3.1.1.1	10/10/2016	Acute Health (eye), Acute Health (inhaled), Acute Health (skin), Acute Health (swallowed), Advice to Doctor, Appearance, Chronic Health, Classification, Disposal, Engineering Control, Environmental, Exposure Standard, Fire Fighter (extinguishing media), Fire Fighter (fire/explosion hazard), Fire Fighter (fire fighting), Fire Fighter (fire incompatibility), First Aid (eye), First Aid (inhaled), First Aid (skin), First Aid (swallowed), Handling Procedure, Personal Protection (other), Personal Protection (Respirator), Personal Protection (eye), Personal Protection (hands/feet), Physical Properties, Spills (major), Spills (minor), Storage (storage incompatibility), Storage (storage requirement), Storage (suitable container), Toxicity and Irritation (Other), Use

Other information

Ingredients with multiple cas numbers

Name	CAS No
tripropylene glycol monomethyl ether	25498-49-1, 20324-33-8

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

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

Definitions and abbreviations

PC-TWA: Permissible Concentration-Time Weighted Average

PC-STEL: Permissible Concentration-Short Term Exposure Limit

IARC: International Agency for Research on Cancer

ACGIH: American Conference of Governmental Industrial Hygienists

STEL: Short Term Exposure Limit

TEEL: Temporary Emergency Exposure Limit。

IDLH: Immediately Dangerous to Life or Health Concentrations

OSF: Odour Safety Factor

NOAEL :No Observed Adverse Effect Level LOAEL: Lowest Observed Adverse Effect Level TLV: Threshold Limit Value LOD: Limit Of Detection OTV: Odour Threshold Value BCF: BioConcentration Factors BEI: Biological Exposure Index

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