CUTEK Wood Preservative

Chemisys Manufacturing Pty Ltd

Chemwatch Hazard Alert Code: **3**

Version No: **2.17**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **28/11/2022**
Print Date: **28/11/2022**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	CUTEK Wood Preservative
Synonyms	Not Available
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains copper 8-quinolinol)
Other means of identification	Not Available

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

Relevant identified uses	For pre-treatment and remedial treatment of exterior wood in industrial and domestic situations by commercial operators only. CUTEK Wood Preservative coated wood resists damage caused by fungal decay, mould and termites. CUTEK Wood Preservative also protects exterior woods from moisture, improving dimensional stability and minimising warping, cupping, and splitting.

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**Chemisys Manufacturing Pty Ltd**
Address	72 Chetwynd Street Queensland 4129 Australia
Telephone	+617 3188 5242
Fax	+617 3073 3919
Website	www.cutek.com.au
Email	admin@chemisys.com.au

Emergency telephone number

Association / Organisation	**Chemisys Manufacturing Pty Ltd**
Emergency telephone numbers	+617 3188 5246
Other emergency telephone numbers	131 126

SECTION 2 Hazards identification

Classification of the substance or mixture

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

Chemwatch Hazard Ratings

		Min	Max
Flammability	1		
Toxicity	2		
Body Contact	2		
Reactivity	0		
Chronic	3		

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

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Poisons Schedule	6
Classification [1]	Serious Eye Damage/Eye Irritation Category 2A, Sensitisation (Respiratory) Category 1, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Flammable Liquids Category 4, Hazardous to the Aquatic Environment Acute Hazard Category 3, Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Aspiration Hazard Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 3
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

Label elements

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

Hazard statement(s)

H319	Causes serious eye irritation.
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H336	May cause drowsiness or dizziness.
H227	Combustible liquid.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H304	May be fatal if swallowed and enters airways.
H412	Harmful to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P261	Avoid breathing mist/vapours/spray.
P271	Use only a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P284	[In case of inadequate ventilation] wear respiratory protection.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.
P331	Do NOT induce vomiting.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P342+P311	If experiencing respiratory symptoms: Call a POISON CENTER/doctor/physician/first aider.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam to extinguish.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

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

Continued...

Precautionary statement(s) Disposal

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

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
10380-28-6	<10	copper 8-quinolinol
64359-81-5	<10	4,5-dichloro-2-octyl-3(2H)-isothiazolone
1330-20-7	<10	xylene
Not Available	10-30	aromatic hydrocarbons
64742-47-8	10-30	distillates, petroleum, light, hydrotreated
64742-65-0.	30-60	paraffinic distillate, heavy, solvent-dewaxed (severe)
Not Available	10-30	phosphoric esters

Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available*

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Wash out immediately with fresh running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▸ Other measures are usually unnecessary.
Ingestion	▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Seek medical advice. ▸ Avoid giving milk or oils. ▸ Avoid giving alcohol. ▸ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

Indication of any immediate medical attention and special treatment needed

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.
Treat symptomatically.
For petroleum distillates
· In case of ingestion, gastric lavage with activated charcoal can be used promptly to prevent absorption - decontamination (induced emesis or lavage) is controversial and should be considered on the merits of each individual case; of course the usual precautions of an endotracheal tube should be considered prior to lavage, to prevent aspiration.
· Individuals intoxicated by petroleum distillates should be hospitalized immediately, with acute and continuing attention to neurologic and cardiopulmonary function.
· Positive pressure ventilation may be necessary.
· Acute central nervous system signs and symptoms may result from large ingestions of aspiration-induced hypoxia.

Continued...

· After the initial episode,individuals should be followed for changes in blood variables and the delayed appearance of pulmonary oedema and chemical pneumonitis. Such patients should be followed for several days or weeks for delayed effects, including bone marrow toxicity, hepatic and renal impairment Individuals with chronic pulmonary disease will be more seriously impaired, and recovery from inhalation exposure may be complicated.
· Gastrointestinal symptoms are usually minor and pathological changes of the liver and kidneys are reported to be uncommon in acute intoxications.
· Chlorinated and non-chlorinated hydrocarbons may sensitize the heart to epinephrine and other circulating catecholamines so that arrhythmias may occur.Careful consideration of this potential adverse effect should precede administration of epinephrine or other cardiac stimulants and the selection of bronchodilators.
BP America Product Safety & Toxicology Department
for copper intoxication:

- Unless extensive vomiting has occurred empty the stomach by lavage with water, milk, sodium bicarbonate solution or a 0.1% solution of potassium ferrocyanide (the resulting copper ferrocyanide is insoluble).
- Administer egg white and other demulcents.
- Maintain electrolyte and fluid balances.
- Morphine or meperidine (Demerol) may be necessary for control of pain.
- If symptoms persist or intensify (especially circulatory collapse or cerebral disturbances, try BAL intramuscularly or penicillamine in accordance with the supplier's recommendations.
- Treat shock vigorously with blood transfusions and perhaps vasopressor amines.
- If intravascular haemolysis becomes evident protect the kidneys by maintaining a diuresis with mannitol and perhaps by alkalinising the urine with sodium bicarbonate.
- It is unlikely that methylene blue would be effective against the occassional methaemoglobinemia and it might exacerbate the subsequent haemolytic episode.
- Institute measures for impending renal and hepatic failure.

[GOSSELIN, SMITH & HODGE: Commercial Toxicology of Commercial Products]

- A role for activated charcoals for emesis is, as yet, unproven.
- In severe poisoning CaNa2EDTA has been proposed.

[ELLENHORN & BARCELOUX: Medical Toxicology]

- Heavy and persistent skin contamination over many years may lead to dysplastic changes. Pre-existing skin disorders may be aggravated by exposure to this product.
- In general, emesis induction is unnecessary with high viscosity, low volatility products, i.e. most oils and greases.
- High pressure accidental injection through the skin should be assessed for possible incision, irrigation and/or debridement.

NOTE: Injuries may not seem serious at first, but within a few hours tissue may become swollen, discoloured and extremely painful with extensive subcutaneous necrosis. Product may be forced through considerable distances along tissue planes.

SECTION 5 Firefighting measures

Extinguishing media

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

Special hazards arising from the substrate or mixture

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

Advice for firefighters

Fire Fighting	‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear full body protective clothing with breathing apparatus. ‣ Prevent, by any means available, spillage from entering drains or water course. ‣ Use water delivered as a fine spray to control fire and cool adjacent area. ‣ Avoid spraying water onto liquid pools. ‣ **DO NOT** approach containers suspected to be hot. ‣ Cool fire exposed containers with water spray from a protected location. ‣ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	‣ Combustible. ‣ Slight fire hazard when exposed to heat or flame. ‣ Heating may cause expansion or decomposition leading to violent rupture of containers. ‣ On combustion, may emit toxic fumes of carbon monoxide (CO). ‣ May emit acrid smoke. ‣ Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO2) phosphorus oxides (POx) sulfur oxides (SOx) metal oxides other pyrolysis products typical of burning organic material.

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	CARE: Water in contact with hot liquid may cause foaming and a steam explosion with wide scattering of hot oil and possible severe burns. Foaming may cause overflow of containers and may result in possible fire.
HAZCHEM	•3Z

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

Environmental hazard - contain spillage.
Slippery when spilt.

- Clean up all spills immediately.
- Avoid breathing vapours and contact with skin and eyes.
- Control personal contact with the substance, by using protective equipment.
- Contain and absorb spill with sand, earth, inert material or vermiculite.
- Wipe up.
- Place in a suitable, labelled container for waste disposal.

Major Spills

Environmental hazard - contain spillage.
Chemical Class: aromatic hydrocarbons
For release onto land: recommended sorbents listed in order of priority.

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS

LAND SPILL - SMALL

Feathers - pillow	1	throw	pitchfork	DGC, RT
cross-linked polymer - particulate	2	shovel	shovel	R,W,SS
cross-linked polymer- pillow	2	throw	pitchfork	R, DGC, RT
sorbent clay - particulate	3	shovel	shovel	R, I, P,
treated clay/ treated natural organic - particulate	3	shovel	shovel	R, I
wood fibre - pillow	4	throw	pitchfork	R, P, DGC, RT

LAND SPILL - MEDIUM

cross-linked polymer -particulate	1	blower	skiploader	R, W, SS
treated clay/ treated natural organic - particulate	2	blower	skiploader	R, I
sorbent clay - particulate	3	blower	skiploader	R, I, P
polypropylene - particulate	3	blower	skiploader	W, SS, DGC
feathers - pillow	3	throw	skiploader	DGC, RT
expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC

Legend
DGC: Not effective where ground cover is dense
R; Not reusable
I: Not incinerable
P: Effectiveness reduced when rainy
RT:Not effective where terrain is rugged
SS: Not for use within environmentally sensitive sites
W: Effectiveness reduced when windy
Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;
R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988
Slippery when spilt.

- Absorb or contain isothiazolinone liquid spills with sand, earth, inert material or vermiculite.
- The absorbent (and surface soil to a depth sufficient to remove all of the biocide) should be shovelled into a drum and treated with an 11% solution of sodium metabisulfite ($Na_2S_2O_5$) or sodium bisulfite ($NaHSO_3$), or 12% sodium sulfite (Na_2SO_3) and 8% hydrochloric acid (HCl).
- Glutathione has also been used to inactivate the isothiazolinones.
- Use 20 volumes of decontaminating solution for each volume of biocide, and let containers stand for at least 30 minutes to deactivate microbicide before disposal.
- If contamination of drains or waterways occurs, advise emergency services.

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	‣ After clean up operations, decontaminate and launder all protective clothing ‣ and equipment before storing and re-using.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	The conductivity of this material may make it a static accumulator., A liquid is typically considered nonconductive if its conductivity is below 100 pS/m and is considered semi-conductive if its conductivity is below 10 000 pS/m., Whether a liquid is nonconductive or semi-conductive, the precautions are the same., A number of factors, for example liquid temperature, presence of contaminants, and anti-static additives can greatly influence the conductivity of a liquid. ‣ Containers, even those that have been emptied, may contain explosive vapours. ‣ Do NOT cut, drill, grind, weld or perform similar operations on or near containers. ‣ Avoid all personal contact, including inhalation. ‣ Wear protective clothing when risk of exposure occurs. ‣ Use in a well-ventilated area. ‣ Prevent concentration in hollows and sumps. ‣ **DO NOT** enter confined spaces until atmosphere has been checked. ‣ Avoid smoking, naked lights or ignition sources. ‣ Avoid contact with incompatible materials. ‣ When handling, **DO NOT** eat, drink or smoke. ‣ Keep containers securely sealed when not in use. ‣ Avoid physical damage to containers. ‣ Always wash hands with soap and water after handling. ‣ Work clothes should be laundered separately. ‣ Use good occupational work practice. ‣ Observe manufacturer's storage and handling recommendations contained within this SDS. ‣ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions. ‣ **DO NOT** allow clothing wet with material to stay in contact with skin
Other information	‣ Store in original containers. ‣ Keep containers securely sealed. ‣ Store in a cool, dry, well-ventilated area. ‣ Store away from incompatible materials and foodstuff containers. ‣ Protect containers against physical damage and check regularly for leaks. ‣ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	‣ Metal can or drum ‣ Packaging as recommended by manufacturer. ‣ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	Inorganic derivative of Group 11 metal. Xylenes: ‣ may ignite or explode in contact with strong oxidisers, 1,3-dichloro-5,5-dimethylhydantoin, uranium fluoride ‣ attack some plastics, rubber and coatings ‣ may generate electrostatic charges on flow or agitation due to low conductivity. ‣ Vigorous reactions, sometimes amounting to explosions, can result from the contact between aromatic rings and strong oxidising agents. ‣ Aromatics can react exothermically with bases and with diazo compounds. For alkyl aromatics: The alkyl side chain of aromatic rings can undergo oxidation by several mechanisms. The most common and dominant one is the attack by oxidation at benzylic carbon as the intermediate formed is stabilised by resonance structure of the ring. ‣ Following reaction with oxygen and under the influence of sunlight, a hydroperoxide at the alpha-position to the aromatic ring, is the primary oxidation product formed (provided a hydrogen atom is initially available at this position) - this product is often short-lived but may be stable dependent on the nature of the aromatic substitution; a secondary C-H bond is more easily attacked than a primary C-H bond whilst a tertiary C-H bond is even more susceptible to attack by oxygen ‣ Monoalkylbenzenes may subsequently form monocarboxylic acids; alkyl naphthalenes mainly produce the corresponding naphthalene carboxylic acids. ‣ Oxidation in the presence of transition metal salts not only accelerates but also selectively decomposes the hydroperoxides. ‣ Hock-rearrangement by the influence of strong acids converts the hydroperoxides to hemiacetals. Peresters formed from the hydroperoxides undergo Criegee rearrangement easily. ‣ Alkali metals accelerate the oxidation while CO2 as co-oxidant enhances the selectivity. ‣ Microwave conditions give improved yields of the oxidation products. ‣ Photo-oxidation products may occur following reaction with hydroxyl radicals and NOx - these may be components of photochemical smogs. Oxidation of Alkylaromatics: T.S.S Rao and Shubhra Awasthi: E-Journal of Chemistry Vol 4, No. 1, pp 1-13 January 2007

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	· **CARE**: Water in contact with heated material may cause foaming or a steam explosion with possible severe burns from wide scattering of hot material. Resultant overflow of containers may result in fire. · Oil leaks in a pressurized circuit may result in a fine flammable spray (the lower flammability limit for oil mist is reached for a concentration of about 45 g/m3 · Autoignition temperatures may be significantly lower under particular conditions (slow oxidation on finely divided materials..

+	x	+	o	+	+	+

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

Note: Depending on other risk factors, compatibility assessment based on the table above may not be relevant to storage situations, particularly where large volumes of dangerous goods are stored and handled. Reference should be made to the Safety Data Sheets for each substance or article and risks assessed accordingly.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	xylene	Xylene (o-, m-, p-isomers)	80 ppm / 350 mg/m3	655 mg/m3 / 150 ppm	Not Available	Not Available
Australia Exposure Standards	distillates, petroleum, light, hydrotreated	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	paraffinic distillate, heavy, solvent-dewaxed (severe)	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
xylene	Not Available	Not Available	Not Available
distillates, petroleum, light, hydrotreated	140 mg/m3	1,500 mg/m3	8,900 mg/m3
paraffinic distillate, heavy, solvent-dewaxed (severe)	140 mg/m3	1,500 mg/m3	8,900 mg/m3

Ingredient	Original IDLH	Revised IDLH
copper 8-quinolinol	Not Available	Not Available
4,5-dichloro-2-octyl-3(2H)-isothiazolone	Not Available	Not Available
xylene	900 ppm	Not Available
distillates, petroleum, light, hydrotreated	2,500 mg/m3	Not Available
paraffinic distillate, heavy, solvent-dewaxed (severe)	2,500 mg/m3	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
copper 8-quinolinol	E	≤ 0.01 mg/m³
4,5-dichloro-2-octyl-3(2H)-isothiazolone	E	≤ 0.1 ppm
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

IFRA Prohibited Fragrance Substance
The International Fragrance Association (IFRA) Standards form the basis for the globally accepted and recognized risk management system for the safe use of

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fragrance ingredients and are part of the IFRA Code of Practice. This is the self-regulating system of the industry, based on risk assessments carried out by an independent Expert Panel
Toxicity and Irritation data for petroleum-based mineral oils are related to chemical components and vary as does the composition and source of the original crude. A small but definite risk of occupational skin cancer occurs in workers exposed to persistent skin contamination by oils over a period of years. This risk has been attributed to the presence of certain polycyclic aromatic hydrocarbons (PAH) (typified by benz[a]pyrene).
Petroleum oils which are solvent refined/extracted or severely hydrotreated, contain very low concentrations of both.
for mineral oils (excluding metal working fluids), pure, highly and severely refined:
Human exposure to oil mist alone has not been demonstrated to cause health effects except at levels above 5 mg/m3 (this applies to particulates sampled by a method that does not collect vapour). It is not advisable to apply this standard to oils containing unknown concentrations and types of additive.
These exposure guidelines have been derived from a screening level of risk assessment and should not be construed as unequivocally safe limits. ORGS represent an 8-hour time-weighted average unless specified otherwise.
CR = Cancer Risk/10000; UF = Uncertainty factor:
TLV believed to be adequate to protect reproductive health:
LOD: Limit of detection
Toxic endpoints have also been identified as:
D = Developmental; R = Reproductive; TC = Transplacental carcinogen
Jankovic J., Drake F.: A Screening Method for Occupational Reproductive
American Industrial Hygiene Association Journal 57: 641-649 (1996)

Exposed individuals are **NOT** reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odour Safety Factor (OSF) is determined to fall into either Class C, D or E.

The Odour Safety Factor (OSF) is defined as:

OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm

Classification into classes follows:

Class	OSF	Description
A	550	Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by working activities
B	26-550	As "A" for 50-90% of persons being distracted
C	1-26	As "A" for less than 50% of persons being distracted
D	0.18-1	10-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached
E	<0.18	As "D" for less than 10% of persons aware of being tested

CEL TWA: 0.1 mg/m3; STEL 0.3 mg/m3 total isothiazolinones (Rohm and Haas)
(CEL = Chemwatch Exposure Limit)

for kerosene CAS 8008-20-6
TLV TWA: 100 mg/m3 as total hydrocarbon vapour Skin A3
OEL TWA: 14 ppm, 100 mg/m3 [NIOSH, 1985]
REL TWA: 150 ppm [Shell]
CEL TWA: 300 ppm, 900 mg/m3
(CEL = Chemwatch Exposure Limit)

for petroleum distillates:
CEL TWA: 500 ppm, 2000 mg/m3 (compare OSHA TWA)
(CEL = Chemwatch Exposure Limit)
for xylenes:
IDLH Level: 900 ppm
Odour Threshold Value: 20 ppm (detection), 40 ppm (recognition)
NOTE: Detector tubes for o-xylene, measuring in excess of 10 ppm, are available commercially. (m-xylene and p-xylene give almost the same response).
Xylene vapour is an irritant to the eyes, mucous membranes and skin and causes narcosis at high concentrations. Exposure to doses sufficiently high to produce intoxication and unconsciousness also produces transient liver and kidney toxicity. Neurologic impairment is NOT evident amongst volunteers inhaling up to 400 ppm though complaints of ocular and upper respiratory tract irritation occur at 200 ppm for 3 to 5 minutes.
Exposure to xylene at or below the recommended TLV-TWA and STEL is thought to minimise the risk of irritant effects and to produce neither significant narcosis or chronic injury. An earlier skin notation was deleted because percutaneous absorption is gradual and protracted and does not substantially contribute to the dose received by inhalation.
Odour Safety Factor(OSF)
OSF=4 (XYLENE)

NOTE L: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 3% DMSO extract as measured by IP 346.
European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP
NOTE P: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.01% w/w benzene (EINECS No 200-753-7). Note E shall also apply when the substance is classified as a carcinogen. This note applies only to certain complex oil-derived substances in Annex VI.
European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to

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provide this high level of protection.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
Employers may need to use multiple types of controls to prevent employee overexposure.

General exhaust is adequate under normal operating conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air)	0.25-0.5 m/s (50-100 f/min)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only	2: Contaminants of high toxicity
3: Intermittent, low production.	3: High production, heavy use
4: Large hood or large air mass in motion	4: Small hood - local control only

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

Personal protection

Eye and face protection

- Safety glasses with side shields.
- Chemical goggles.
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]

Skin protection

See Hand protection below

Hands/feet protection

- Wear chemical protective gloves, e.g. PVC.
- Wear safety footwear or safety gumboots, e.g. Rubber

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:
· frequency and duration of contact,
· chemical resistance of glove material,

Continued...

	· glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. ▸ Butyl rubber gloves · Nitrile rubber gloves (Note: Nitric acid penetrates nitrile gloves in a few minutes.)
Body protection	See Other protection below
Other protection	▸ Overalls. ▸ P.V.C apron. ▸ Barrier cream. ▸ Skin cleansing cream. ▸ Eye wash unit.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
"Forsberg Clothing Performance Index".
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
CUTEK Wood Preservative

Material	CPI
PE/EVAL/PE	A
PVA	A
TEFLON	A
VITON	A
BUTYL	C
BUTYL/NEOPRENE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PVC	C
PVDC/PE/PVDC	C

* CPI - Chemwatch Performance Index

Respiratory protection

Type AK-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	AK-AUS P2	-	AK-PAPR-AUS / Class 1 P2
up to 50 x ES	-	AK-AUS / Class 1 P2	-
up to 100 x ES	-	AK-2 P2	AK-PAPR-2 P2 ^

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

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

Continued...

A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	amber/green

Physical state	Liquid	**Relative density (Water = 1)**	0.88
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)**	17-32
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	80	**Taste**	Not Available
Evaporation rate	Not Available BuAC = 1	**Explosive properties**	Not Available
Flammability	Combustible.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Immiscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	300

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

Continued...

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. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Inhalation hazard is increased at higher temperatures. High inhaled concentrations of mixed hydrocarbons may produce narcosis characterised by nausea, vomiting and lightheadedness. Inhalation of aerosols may produce severe pulmonary oedema, pneumonitis and pulmonary haemorrhage. Inhalation of petroleum hydrocarbons consisting substantially of low molecular weight species (typically C2-C12) may produce irritation of mucous membranes, incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and anaesthetic stupor. Massive exposures may produce central nervous system depression with sudden collapse and deep coma; fatalities have been recorded. Irritation of the brain and/or apnoeic anoxia may produce convulsions. Although recovery following overexposure is generally complete, cerebral micro-haemorrhage of focal post-inflammatory scarring may produce epileptiform seizures some months after the exposure. Pulmonary episodes may include chemical pneumonitis with oedema and haemorrhage. The lighter hydrocarbons may produce kidney and neurotoxic effects. Pulmonary irritancy increases with carbon chain length for paraffins and olefins. Alkenes produce pulmonary oedema at high concentrations. Liquid paraffins may produce anaesthesia and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce polyneuropathy. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue and vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons are cardiac sensitisers and may cause ventricular fibrillations. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal. Inhalation of oil droplets/ aerosols may cause discomfort and may produce chemical pneumonitis. Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination Copper poisoning following exposure to copper dusts and fume may result in headache, cold sweat and weak pulse. Capillary, kidney, liver and brain damage are the longer term manifestations of such poisoning. Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in "metal fume fever". Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure. Headache, fatigue, lassitude, irritability and gastrointestinal disturbances (e.g., nausea, anorexia and flatulence) are the most common symptoms of xylene overexposure. Injury to the heart, liver, kidneys and nervous system has also been noted amongst workers. Transient memory loss, renal impairment, temporary confusion and some evidence of disturbance of liver function was reported in three workers overcome by gross exposure to xylene (10000 ppm). One worker died and autopsy revealed pulmonary congestion, oedema and focal alveolar haemorrhage. Volunteers inhaling xylene at 100 ppm for 5 to 6 hours showed changes in manual coordination reaction time and slight ataxia. Tolerance developed during the workweek but was lost over the weekend. Physical exercise may antagonise this effect. Xylene body burden in humans exposed to 100 or 200 ppm xylene in air depends on the amount of body fat with 4% to 8% of total absorbed xylene accumulating in adipose tissue. Xylene is a central nervous system depressant. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal. Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.
Ingestion	Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual. Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result. Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis). Isothiazolinones are moderately to highly toxic by oral administration. The major signs of toxicity were severe gastric irritation, lethargy, and ataxia Ingestion of petroleum hydrocarbons may produce irritation of the pharynx, oesophagus, stomach and small intestine with oedema and mucosal ulceration resulting; symptoms include a burning sensation in the mouth and throat. Large amounts may produce narcosis with nausea and vomiting, weakness or dizziness, slow and shallow respiration, swelling of the abdomen, unconsciousness and convulsions. Myocardial injury may produce arrhythmias, ventricular fibrillation and electrocardiographic changes. Central nervous system depression may also occur. Light aromatic hydrocarbons produce a warm, sharp, tingling sensation on contact with taste buds and may anaesthetise the tongue. Aspiration into the lungs may produce coughing, gagging and a chemical pneumonitis with pulmonary oedema and haemorrhage. Numerous cases of a single oral exposure to high levels of copper have been reported. Consumption of copper-contaminated drinking water has been associated with mainly gastrointestinal symptoms including nausea, abdominal pain, vomiting and diarrhoea. A metallic taste, nausea, vomiting and epigastric burning often occur after ingestion of copper and its derivatives. The

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	vomitus is usually green/blue and discolours contaminated skin. Acute poisonings from the ingestion of copper salts are rare due to their prompt removal by vomiting. Vomiting is due mainly to the local and astringent action of copper ion on the stomach and bowel. Emesis usually occurs within 5 to 10 minutes but may be delayed if food is present in the stomach. Should vomiting not occur, or is delayed, gradual absorption from the bowel may result in systemic poisoning with death, possibly, following within several days. Apparent recovery may be followed by lethal relapse. Systemic effects of copper resemble other heavy metal poisonings and produce wide-spread capillary damage, kidney and liver damage and central nervous system excitation followed by depression. Haemolytic anaemia (a result of red-blood cell damage) has been described in acute human poisoning. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products.] Other symptoms of copper poisoning include lethargy, neurotoxicity, and increased blood pressure and respiratory rates. Coma and death have followed attempted suicides using solutions of copper sulfate. Copper is an essential element and most animal tissues have measurable amounts of copper associated with them. Humans have evolved mechanisms which maintain is availability whilst limiting its toxicity (homeostasis). Copper is initially bound in the body to a blood-borne protein, serum albumin and thereafter is more firmly bound to another protein, alpha-ceruloplasmin. Such binding effectively "inactivates" the copper, thus reducing its potential to produce toxic damage. In healthy individuals, bound copper can reach relatively high levels without producing adverse health effects. Excretion in the bile represents the major pathway by which copper is removed from the body when it reaches potentially toxic levels. Copper may also be stored in the liver and bone marrow where it is bound to another protein, metallothionein. A combination of binding and excretion ensures that the body is able to tolerate relatively high loadings of copper.
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. The material may accentuate any pre-existing dermatitis condition Aqueous solutions of isothiazolinones may be irritating or even corrosive depending on concentration. Solutions containing more than 0.5% (5000 ppm active substance) may produce severe irritation of human skin whilst solutions containing more than 100 ppm may irritate the skin. Exposure to copper, by skin, has come from its use in pigments, ointments, ornaments, jewellery, dental amalgams and IUDs and as an antifungal agent and an algicide. Although copper algicides are used in the treatment of water in swimming pools and reservoirs, there are no reports of toxicity from these applications. Reports of allergic contact dermatitis following contact with copper and its salts have appeared in the literature, however the exposure concentrations leading to any effect have been poorly characterised. In one study, patch testing of 1190 eczema patients found that only 13 (1.1%) cross-reacted with 2% copper sulfate in petrolatum. The investigators warned, however, that the possibility of contamination with nickel (an established contact allergen) might have been the cause of the reaction. Copper salts often produce an itching eczema in contact with skin. This is, likely, of a non-allergic nature. The liquid may be miscible with fats or oils and may degrease the skin, producing a skin reaction described as non-allergic contact dermatitis. The material is unlikely to produce an irritant dermatitis as described in EC Directives . Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	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). Solutions containing isothiazolinones may produce corrosion of the mucous membranes and cornea. Instillation of 0.1 ml of an aqueous solution containing 560 ppm isothiazolinone into rabbit eye did not produce irritation whereas concentrations, typically around 3% and 5.5 %, were severely irritating or corrosive to the eye.. Symptoms included clouding of the cornea, chemosis and swelling of the eyelids. Petroleum hydrocarbons may produce pain after direct contact with the eyes. Slight, but transient disturbances of the corneal epithelium may also result. The aromatic fraction may produce irritation and lachrymation. Copper salts, in contact with the eye, may produce conjunctivitis or even ulceration and turbidity of the cornea.
Chronic	Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial number of individuals at a greater frequency than would be expected from the response of a normal population. Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in impaired fertility on the basis of: - clear evidence in animal studies of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects but which is not a secondary non-specific consequence of other toxic effects. Principal route of exposure is by skin contact; lesser exposures include inhalation of fumes from hot oils, oil mists or droplets. Prolonged contact with mineral oils carries with it the risk of skin conditions such as oil folliculitis, eczematous dermatitis, pigmentation of the face (melanosis) and warts on the sole of the foot (plantar warts). With highly refined mineral oils no

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appreciable systemic effects appear to result through skin absorption.
Exposure to oil mists frequently elicits respiratory conditions, such as asthma; the provoking agent is probably an additive. High oil mist concentrations may produce lipoid pneumonia although clinical evidence is equivocal. In animals exposed to concentrations of 100 mg/m3 oil mist, for periods of 12 to 26 months, the activity of lung and serum alkaline phosphatase enzyme was raised; 5 mg/m3 oil mist did not produce this response. These enzyme changes are sensitive early indicators of lung damage. Workers exposed to vapours of mineral oil and kerosene for 5 to 35 years showed an increased prevalence of slight basal lung fibrosis.
Many studies have linked cancers of the skin and scrotum with mineral oil exposure. Contaminants in the form of additives and the polycyclic aromatic hydrocarbons (PAHs - as in the crude base stock) are probably responsible. PAH levels are higher in aromatic process oils/used/reclaimed motor oils. Subchronic 90-day feeding studies conducted on male and female rats on highly refined white mineral oils and waxes found that higher molecular-weight hydrocarbons (microcrystalline waxes and the higher viscosity oils) were without biological effects. Paraffin waxes and low- to mid viscosity oils produced biological effects that were inversely proportional to molecular weight, viscosity and melting point: oil-type and processing did not appear to be determinants. Biological effects were more pronounced in females than in males. Effects occurred mainly in the liver and mesenteric lymph nodes and included increased organ weights, microscopic inflammatory changes, and evidence for the presence of saturated mineral hydrocarbons in affected tissues. Inflammation of the cardiac mitral valve was also observed at high doses in rats treated with paraffin waxes.
Smith J.H., et al: Toxicologic Pathology: 24, 2, 214-230, 1996
Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/or memory loss, tremor in the fingers and tongue, vertigo, olfactory disorders, constriction of visual field, paraesthesias of the extremities, weight loss and anaemia and degenerative changes in the liver and kidney. Chronic exposure by petroleum workers, to the lighter hydrocarbons, has been associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness and paraesthesias), psychological and neurophysiological deficits, bone marrow toxicities (including hypoplasia possibly due to benzene) and hepatic and renal involvement. Chronic dermal exposure to petroleum hydrocarbons may result in defatting which produces localised dermatoses. Surface cracking and erosion may also increase susceptibility to infection by microorganisms. One epidemiological study of petroleum refinery workers has reported elevations in standard mortality ratios for skin cancer along with a dose-response relationship indicating an association between routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have been unable to confirm this finding.
Hydrocarbon solvents are liquid hydrocarbon fractions derived from petroleum processing streams, containing only carbon and hydrogen atoms, with carbon numbers ranging from approximately C5-C20 and boiling between approximately 35-370 deg C. Many of the hydrocarbon solvents have complex and variable compositions with constituents of 4 types, alkanes (normal paraffins, isoparaffins, and cycloparaffins) and aromatics (primarily alkylated one- and two-ring species). Despite the compositional complexity, most hydrocarbon solvent constituents have similar toxicological properties, and the overall toxicological hazards can be characterized in generic terms. Hydrocarbon solvents can cause chemical pneumonitis if aspirated into the lung, and those that are volatile can cause acute CNS effects and/or ocular and respiratory irritation at exposure levels exceeding occupational recommendations. Otherwise, there are few toxicologically important effects. The exceptions, n-hexane and naphthalene, have unique toxicological properties

Animal studies:
No deaths or treatment related signs of toxicity were observed in rats exposed to light alkylate naphtha (paraffinic hydrocarbons) at concentrations of 668, 2220 and 6646 ppm for 6 hrs/day, 5 days/wk for 13 weeks. Increased liver weights and kidney toxicity (male rats) was observed in high dose animals. Exposure to pregnant rats at concentrations of 137, 3425 and 6850 ppm did not adversely affect reproduction or cause maternal or foetal toxicity. Lifetime skin painting studies in mice with similar naphthas have shown weak or no carcinogenic activity following prolonged and repeated exposure. Similar naphthas/distillates, when tested at nonirritating dose levels, did not show any significant carcinogenic activity indicating that this tumorigenic response is likely related to chronic irritation and not to dose. The mutagenic potential of naphthas has been reported to be largely negative in a variety of mutagenicity tests. The exact relationship between these results and human health is not known. Some components of this product have been shown to produce a species specific, sex hormonal dependent kidney lesion in male rats from repeated oral or inhalation exposure. Subsequent research has shown that the kidney damage develops via the formation of a alpha-2u-globulin, a mechanism unique to the male rat. Humans do not form alpha-2u-globulin, therefore, the kidney effects resulting from this mechanism are not relevant in human.
For copper and its compounds (typically copper chloride):
Acute toxicity: There are no reliable acute oral toxicity results available. Animal testing shows that skin in exposure to copper may lead to hardness of the skin, scar formation, exudation and reddish changes. Inflammation, irritation and injury of the skin were noted.
Repeat dose toxicity: Animal testing shows that very high levels of copper monochloride may cause anaemia.
Genetic toxicity: Copper monochloride does not appear to cause mutations in vivo, although chromosomal aberrations were seen at very high concentrations in vitro.
Cancer-causing potential: There was insufficient information to evaluate the cancer-causing activity of copper monochloride.
The isothiazolinones are known contact sensitisers. Data are presented which demonstrate that, in comparison with the chlorinated and dichlorinated compounds which share immunological cross-reactivity, the non-chlorinated isothiazolinones have a lower potential for sensitization and no documented immunological cross-reaction with the chlorinated isothiazolinones. The risk of sensitization depends on how contact with the product occurs. The risk is greater when the skin barrier has been damaged and smaller when the skin is healthy. Dermatological studies have demonstrated that mixed isothiazolinone concentrations below 20 ppm may cause sensitisation and that allergic reactions can be provoked in sensitized persons even with concentrations in the range of 7-15 ppm active isothiazolinones.
The isothiazolinones are a group of heterocyclic sulfur-containing compounds. In general all are electrophilic molecules containing an activated N-S bond that enables them with nucleophilic cell entities, thus exerting biocidal activity. A vinyl activated chlorine atom makes allows to molecule to exert greater antimicrobial efficiency but at the same time produces a greater potential for sensitisation.
Several conclusions relating to the sensitising characteristics of the isothiazolinones may therefore be drawn* :

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- The strongest sensitisers are the chlorinated isothiazolinones.
- There are known immunological cross-reactions between at least 2 different chlorinated isothiazolinones.
- There appears to be no immunological cross reaction between non-chlorinated isothiazolinones and chlorinated isothiazolinones.
- Although classified as sensitisers, the nonchlorinated isothiazolinones are considerably less potent sensitisers than are the chlorinated isothiazolinones.
- By avoiding the use of chlorinated isothiazolinones, the potential to induce sensitisation is greatly reduced.
- Despite a significant percentage of the population having been previously sensitised to chlorinated and non-chlorinated species, it is likely that careful and judicious use of non-chlorinated isothiazolinones will result in reduced risk of allergic reactions in those persons.
- Although presently available data promise that several non-chlorinated isothiazolinones will offer effective antimicrobial protection in industrial and personal care products, it is only with the passage of time that proof of their safety in use or otherwise will become available.

* B.R. Alexander: Contact Dermatitis 2002, 46, pp 191-196

Although there have been conflicting reports in the literature, it has been reported by several investigators that isothiazolinones are mutagenic in *Salmonella typhimurium* strains (Ames test). Negative results were obtained in studies of the DNA-damaging potential of mixed isothiazolinones (Kathon) in mammalian cells *in vitro* and of cytogenetic effects and DNA-binding *in vivo.* The addition of rat liver S-9 (metabolic activation) reduced toxicity but did not eliminate mutagenicity. These compounds bind to the proteins in the S-9. At higher concentrations of Kathon the increase in mutagenicity may be due to an excess of unbound active compounds.

A study of cutaneous application of Kathon CG in 30 months, three times per week at a concentration of 400 ppm (0.04%) a.i. had no local or systemic tumourigenic effect in male mice. No dermal or systemic carcinogenic potential was observed.

Reproduction and teratogenicity studies with rats, given isothiazolinone doses of 1.4-14 mg/kg/day orally from day 6 to day 15 of gestation, showed no treatment related effects in either the dams or in the foetuses

On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.

Prolonged or repeated contact with xylenes may cause defatting dermatitis with drying and cracking. Chronic inhalation of xylenes has been associated with central nervous system effects, loss of appetite, nausea, ringing in the ears, irritability, thirst anaemia, mucosal bleeding, enlarged liver and hyperplasia. Exposure may produce kidney and liver damage. In chronic occupational exposure, xylene (usually mix ed with other solvents) has produced irreversible damage to the central nervous system and ototoxicity (damages hearing and increases sensitivity to noise), probably due to neurotoxic mechanisms.

Industrial workers exposed to xylene with a maximum level of ethyl benzene of 0.06 mg/l (14 ppm) reported headaches and irritability and tired quickly. Functional nervous system disturbances were found in some workers employed for over 7 years whilst other workers had enlarged livers.

Xylene has been classed as a developmental toxin in some jurisdictions.

Small excess risks of spontaneous abortion and congenital malformation were reported amongst women exposed to xylene in the first trimester of pregnancy. In all cases, however, the women were also been exposed to other substances. Evaluation of workers chronically exposed to xylene has demonstrated lack of genotoxicity. Exposure to xylene has been associated with increased risks of haemopoietic malignancies but, again, simultaneous exposure to other substances (including benzene) complicates the picture. A long-term gavage study to mixed xylenes (containing 17% ethyl benzene) found no evidence of carcinogenic activity in rats and mice of either sex.

CUTEK Wood Preservative

TOXICITY	IRRITATION
Not Available	Not Available

copper 8-quinolinol

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >2000 mg/kg[2]	Not Available
Oral (Mouse) LD50; 3940 mg/kg[2]	

4,5-dichloro-2-octyl-3(2H)-isothiazolone

TOXICITY	IRRITATION
Inhalation(Rat) LC50: 0.758 mg/L4h[2]	Not Available

xylene

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >1700 mg/kg[2]	Eye (human): 200 ppm irritant
Inhalation(Rat) LC50: 5000 ppm4h[2]	Eye (rabbit): 5 mg/24h SEVERE
Oral (Mouse) LD50; 2119 mg/kg[2]	Eye (rabbit): 87 mg mild
	Eye: adverse effect observed (irritating)[1]
	Skin (rabbit):500 mg/24h moderate
	Skin: adverse effect observed (irritating)[1]

distillates, petroleum, light, hydrotreated

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >2000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
Inhalation(Rat) LC50: >4.3 mg/l4h[1]	Skin: adverse effect observed (irritating)[1]

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	Oral (Rat) LD50; >5000 mg/kg[2]	
paraffinic distillate, heavy, solvent-dewaxed (severe)	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50: 2.18 mg/l4h[2]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; >5000 mg/kg[2]	
Legend:	*1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*	

COPPER 8-QUINOLINOL	Equivocal tumourigen by RTECS criteria Animal tests record Flaccid paralysis, convulsions, dyspnae; with tumours at site of application In rabbits and dogs, quinoline and its metabolites are excreted in the urine. Urinary excretion of quinoline and its metabolites was nearly complete 24 hours after i.v. dosing of dogs with 20 or 25 mg/kg . Less than 0.5% of the administered quinoline was excreted unchanged. Approximately 29%-32% of the administered quinoline was recovered from the urine as 3-hydroxyquinoline (free and conjugated forms). Approximately 0.4%-0.8% of free quinoline was detected in rabbit urine collected 24 hours after administration of an oral dose of 250 mg/kg . Approximately 6.7%-11.0 % of the quinoline was determined to be excreted as a labile compound that yields quinoline on heating with acid. About 3%-4% of quinoline was excreted as the metabolite 5,6-dihydroxyquinoline. **Repeat dose toxicity**: Groups of 20 male Sprague-Dawley rats were fed a diet containing 0.05% (low-dose), 0.10% (mid-dose), or 0.25% (high-dose) quinoline for approximately 16-40 weeks. Absolute and relative liver weights were significantly increased in all treatment groups, and the difference between initial and final mean body weights decreased with increasing dose. Histological examination of the liver revealed fatty change, bile duct proliferation, and oval cells in treated animals. Also, nodular hyperplasia was seen in the mid- and high-dose animals. **Carcinogenicity:** No reliable human epidemiological studies are available that address the potential carcinogenicity of quinoline. However, laboratory studies have shown that quinoline is mitogenic and mutagenic in vitro and in vivo , and that humans and rats share a common quinoline-metabolizing P450 enzyme. Liver tumors have been observed in rats and mice exposed to quinoline via oral and i.p. routes of exposure, but not in rats exposed subcutaneously, despite the fact that the s.c. injections resulted in maximally tolerated doses more than 40 times higher than i.p. doses given to mice . The observation of skin tumors on mice dermally exposed to quinoline and tumor promoter tetradecanoyl phorbol acetate suggests that quinoline can initiate skin tumors (no other tumor types were reported) without first-pass metabolism in the liver, but the question of whether inhaled quinoline would have such effects without promotion remains. Several animal studies report hepatocarcinogenicity (hepatocellular carcinomas and haemangioendotheliomas or haemangiosarcomas, a vascular tumor) in rats and mice following oral dosing with quinoline. Quinoline has also been reported to be a hepatocarcinogen in newborn mice following intraperitoneal exposure . Metastatic changes, arising from these tumors, were detected in the lungs of some of the rats. Hepatic tumours (carcinomas, adenomas, and basophilic altered foci) were observed in male newborn mice, but not male or female newborn rats. No tumors, but basophilic altered foci, were observed in female newborn mice. Quinoline initiated skin tumors in female SENCAR mice following dermal application Important aspects of the hepatocarcinogenicity of quinoline are the relatively short latency period (as low as 12 weeks) for tumor formation, and the fact that one of the tumor types observed, haemangioendotheliomas, is uncommon in rats and mice. Other studies indicate species differences in regard to liver tumorigenesis by quinoline; mice and rats are most susceptible and hamsters and guinea pigs appear to be resistant. Quinoline is considered *likely to be carcinogenic in humans* in accordance with proposed EPA carcinogen risk assessment guidelines (U.S. EPA, 1996) on the basis of observations of exposure-related increased incidence of an unusual malignant tumor in multiple strains of rats and mice, in multiple experiments using oral, dermal, i.p., and s.c. dosing, and at an early age. This determination is supported by studies that demonstrate that quinoline is genotoxic. Quinoline can apparently act as a promoter of liver carcinogenicity as well. Quinoline, 3-fluoroquinone (3-FQ), or 5-fluoroquinone (5-FQ) were fed to F344 male rats in their diet (0.1% and 0.05%) for a period of 6 weeks following a single, 200 mg/kg i.p. injection of the liver carcinogen diethylnitrosamine (DEN). The number and areas of GST-P (placental glutathione S10 transferase)-positive foci induced in the liver increased significantly as a result of treatment with 0.1% but not 0.05% quinoline **Genotoxicity:** Quinoline is a mutagen in *Salmonella typhimurium* in the presence of metabolic activation . Quinoline has also been shown to induce chromosome aberrations and sister chromatid exchanges in the rat liver and micronucleus formation in the bone marrow of CD1 male mice. Although a predominance of data suggest that quinoline is genotoxic, the results of at least one study indicate that a nongenotoxic (i.e., mitogenic) mechanism of action may play a role in its hepatocarcinogenicity (Quinoline was found to have significant activity in the *Salmonella typhimurium* strain TA100, but generally not in strains TA1537 and TA1538 , nor TA98 , suggesting that it may be acting via base-pair substitution). 3-Fluoro- and 2- and 3-chloroquinolines were less mutagenic than all other fluoro- and chloro-substituted derivatives of quinoline . The 3-fluoro derivative of quinoline completely blocks the mutagenic activity of quinoline. Substitutions at other locations do not reduce quinoline s mutagenicity, and in some cases enhance it (presumably by inhibiting detoxification pathways). Studies suggest that the 2,3-epoxide is the active metabolic mutagen based on the fact that the 4-chloro isomer is weakly mutagenic (presumably no mutagenicity would be observed if a 3,4-epoxide were necessary), the 4-methyl isomer is strongly mutagenic (suggested to be because of suppression of detoxification of the 2,3-epoxide), and the 2-methyl isomer is weakly mutagenic (the authors report that methyl substitution at the site of epoxide formation is known to partially reduce mutagenicity)
4,5-DICHLORO-2-OCTYL-3(2H)-ISOTHIAZOLONE	Guinea Pig Assay: causes sensitisation * Did not show teratogenic effects in animal experiments. * Not mutagenic * *Rohm and Haas MSDS Rozone 2000 Mildewcide The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic

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	skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.
XYLENE	Reproductive effector in rats The material may produce severe irritation to the eye causing pronounced 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.
CUTEK Wood Preservative & COPPER 8-QUINOLINOL	Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens). Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis. Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure.
CUTEK Wood Preservative & DISTILLATES, PETROLEUM, LIGHT, HYDROTREATED & PARAFFINIC DISTILLATE, HEAVY, SOLVENT-DEWAXED (SEVERE)	Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length,with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent that iso- or cyclo-paraffins. The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption,is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.
CUTEK Wood Preservative & PARAFFINIC DISTILLATE, HEAVY, SOLVENT-DEWAXED (SEVERE)	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 *residual base oils* 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)). 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

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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.
Highly and Severely Refined Distillate Base Oils
Acute toxicity: Multiple studies of the acute toxicity of highly & severely refined base oils have been reported. Irrespective of the crude source or the method or extent of processing, the oral LD50s have been observed to be >5 g/kg (bw) and the dermal LD50s have ranged from >2 to >5g/kg (bw). The LC50 for inhalation toxicity ranged from 2.18 mg/l to> 4 mg/l.
When tested for skin and eye irritation, the materials have been reported as “non-irritating” to “moderately irritating”
Testing in guinea pigs for sensitization has been negative
Repeat dose toxicity: . Several studies have been conducted with these oils**. T**he weight of evidence from all available data on highly & severely refined base oils support the presumption that a distillate base oil s toxicity is inversely related to the degree of processing it receives. Adverse effects have been reported with even the most severely refined white oils - these appear to depend on animal species and/ or the peculiarities of the study.

- The granulomatous lesions induced by the oral administration of white oils are essentially foreign body responses. The lesions occur only in rats, of which the Fischer 344 strain is particularly sensitive,
- The testicular effects seen in rabbits after dermal administration of a highly to severely refined base oil were unique to a single study and may have been related to stress induced by skin irritation, and
- The accumulation of foamy macrophages in the alveolar spaces of rats exposed repeatedly via inhalation to high levels of highly to severely refined base oils is not unique to these oils, but would be seen after exposure to many water insoluble materials.

Reproductive and developmental toxicity: A highly refined base oil was used as the vehicle control in a one-generation reproduction study. The study was conducted according to the OECD Test Guideline 421. There was no effect on fertility and mating indices in either males or females. At necropsy, there were no consistent findings and organ weights and histopathology were considered normal by the study s authors.
A single generation study in which a white mineral oil (a food/ drug grade severely refined base oil) was used as a vehicle control is reported. Two separate groups of pregnant rats were administered 5 ml/kg (bw)/day of the base oil via gavage, on days 6 through 19 of gestation. In one of the two base oil dose groups, three malformed foetuses were found among three litters The study authors considered these malformations to be minor and within the normal ranges for the strain of rat.
Genotoxicity:
In vitro (mutagenicity): Several studies have reported the results of testing different base oils for mutagenicity using a modified Ames assay Base oils with no or low concentrations of 3-7 ring PACs had low mutagenicity indices.
In vivo (chromosomal aberrations): A total of seven base stocks were tested in male and female Sprague-Dawley rats using a

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	bone marrow cytogenetics assay. The test materials were administered via gavage at dose levels ranging from 500 to 5000 mg/kg (bw). Dosing occurred for either a single day or for five consecutive days. None of the base oils produced a significant increase in aberrant cells. **Carcinogenicity:** Highly & severely refined base oils are not carcinogens, when given either orally or dermally.
CUTEK Wood Preservative & DISTILLATES, PETROLEUM, LIGHT, HYDROTREATED	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 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 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 stress-related 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-

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	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.
COPPER 8-QUINOLINOL & 4,5-DICHLORO-2-OCTYL-3(2H)-ISOTHIAZOLONE	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.
COPPER 8-QUINOLINOL & XYLENE & PARAFFINIC DISTILLATE, HEAVY, SOLVENT-DEWAXED (SEVERE)	The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.
DISTILLATES, PETROLEUM, LIGHT, HYDROTREATED & PARAFFINIC DISTILLATE, HEAVY, SOLVENT-DEWAXED (SEVERE)	No significant acute toxicological data identified in literature search.

Acute Toxicity	✗	**Carcinogenicity**	✗
Skin Irritation/Corrosion	✓	**Reproductivity**	✗
Serious Eye Damage/Irritation	✓	**STOT - Single Exposure**	✓
Respiratory or Skin sensitisation	✓	**STOT - Repeated Exposure**	✗
Mutagenicity	✗	**Aspiration Hazard**	✓

Legend: ✗ *– Data either not available or does not fill the criteria for classification*
✓ *– Data available to make classification*

SECTION 12 Ecological information

Toxicity

CUTEK Wood Preservative

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

copper 8-quinolinol

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	0.132-0.203mg/L	4
EC50(ECx)	120h	Algae or other aquatic plants	<0.002mg/L	4
LC50	96h	Fish	0.006-0.011mg/L	4

4,5-dichloro-2-octyl-3(2H)-isothiazolone

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	48h	Crustacea	0.005mg/l	Not Available

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	EC50	72h	Algae or other aquatic plants	0.003mg/l	4
	EC50	48h	Crustacea	0.005mg/l	Not Available
	LC50	96h	Fish	0.003mg/l	Not Available
	EC50	96h	Algae or other aquatic plants	0.002-0.01mg/L	4

xylene	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	4.6mg/l	2
	EC50	48h	Crustacea	1.8mg/l	2
	NOEC(ECx)	73h	Algae or other aquatic plants	0.44mg/l	2
	LC50	96h	Fish	2.6mg/l	2

distillates, petroleum, light, hydrotreated	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	NOEC(ECx)	3072h	Fish	1mg/l	1

paraffinic distillate, heavy, solvent-dewaxed (severe)	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	ErC50	72h	Algae or other aquatic plants	>1000mg/l	1
	NOEC(ECx)	504h	Crustacea	>1mg/l	1
	EC50	48h	Crustacea	>1000mg/l	1
	EC50	96h	Algae or other aquatic plants	>1000mg/l	1

Legend:	*Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*

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.
For Aromatic Substances Series:
Environmental Fate: Large, molecularly complex polycyclic aromatic hydrocarbons, or PAHs, are persistent in the environment longer than smaller PAHs.
Atmospheric Fate: PAHs are 'semi-volatile substances" which can move between the atmosphere and the Earth's surface in repeated, temperature-driven cycles of deposition and volatilization. Terrestrial Fate: BTEX compounds have the potential to move through soil and contaminate ground water, and their vapors are highly flammable and explosive.
Ecotoxicity - Within an aromatic series, acute toxicity increases with increasing alkyl substitution on the aromatic nucleus. The order of most toxic to least in a study using grass shrimp and brown shrimp was dimethylnaphthalenes > methylnaphthalenes >naphthalenes. Anthrcene is a phototoxic PAH. UV light greatly increases the toxicity of anthracene to bluegill sunfish. Biological resources in strong sunlight are at more risk than those that are not. PAHs in general are more frequently associated with chronic risks.
For copper:
Atmospheric Fate - Copper is unlikely to accumulate in the atmosphere due to a short residence time for airborne copper aerosols. Airborne coppers, however, may be transported over large distances. Air Quality Standards: no data available.
Aquatic Fate: Toxicity of copper is affected by pH and hardness of water. Total copper is rarely useful as a predictor of toxicity. In natural sea water, more than 98% of copper is organically bound and in river waters a high percentage is often organically bound, but the actual percentage depends on the river water and its pH.
Ecotoxicity: Copper accumulates significantly in the food chain. The toxic effect of copper in the aquatic biota depends on the bio-availability of copper in water which, in turn, depends on its physico-chemical form (i.e. speciation). Bioavailability is decreased by complexation and adsorption of copper by natural organic matter, iron and manganese hydrated oxides, and chelating agents excreted by algae and other aquatic organisms. Copper exhibits significant toxicity in some aquatic organisms. Some algal species are very sensitive to copper. Silicate, iron, manganese and EDTA may reduce bioavailability.
For petroleum distillates:
Environmental fate:
When petroleum substances are released into the environment, four major fate processes will take place: dissolution in water, volatilization, biodegradation and adsorption. These processes will cause changes in the composition of these UVCB substances. In the case of spills on land or water surfaces, photodegradation-another fate process-can also be significant.
As noted previously, the solubility and vapour pressure of components within a mixture will differ from those of the component alone. These interactions are complex for complex UVCBs such as petroleum hydrocarbons.
Each of the fate processes affects hydrocarbon families differently. Aromatics tend to be more water-soluble than aliphatics of the same carbon number, whereas aliphatics tend to be more volatile. Thus,when a petroleum mixture is released into the environment, the principal water contaminants are likely to be aromatics, whereas aliphatics will be the principal air contaminants . The trend in volatility by component class is as follows: alkenes = alkanes > aromatics = cycloalkanes. The most soluble and volatile components have the lowest molecular weight; thus there is a general shift to higher molecular weight components in residual materials.
Biodegradation:
Biodegradation is almost always operative when petroleum mixtures are released into the environment. It has been widely demonstrated that nearly all soils and sediments have populations of bacteria and other organisms capable of degrading petroleum hydrocarbons Degradation occurs both in the presence and absence of oxygen. Two key factors that determine degradation rates are oxygen supply and molecular structure. In general, degradation is more rapid under aerobic conditions. Decreasing trends in degradation rates according to structure are as follows:
(1) n-alkanes, especially in the C10–C25 range, which are degraded readily;
(2) isoalkanes;

Continued...

(3) alkenes;
(4) benzene, toluene, ethylbenzene, xylenes (BTEX) (when present in concentrations that are not toxic to microorganisms);
(5) monoaromatics;
(6) polynuclear (polycyclic) aromatic hydrocarbons (PAHs); and
(7) higher molecular weight cycloalkanes (which may degrade very slowly.
Three weathering processes-dissolution in water, volatilization and biodegradation-typically result in the depletion of the more readily soluble,volatile and degradable compounds and the accumulation of those most resistant to these processes in residues.
When large quantities of a hydrocarbon mixture enter the soil compartment, soil organic matter and other sorption sites in soil are fully saturated and the hydrocarbons will begin to form a separate phase (a non-aqueous phase liquid, or NAPL) in the soil. At concentrations below the retention capacity for the hydrocarbon in the soil, the NAPL will be immobile this is referred to as residual NAPL . Above the retention capacity, the NAPL becomes mobile and will move within the soil
Bioaccumulation:
Bioaccumulation potential was characterized based on empirical and/or modelled data for a suite of petroleum hydrocarbons expected to occur in petroleum substances. Bioaccumulation factors (BAFs) are the preferred metric for assessing the bioaccumulation potential of substances, as the bioconcentration factor (BCF) may not adequately account for the bioaccumulation potential of substances via the diet, which predominates for substances with log Kow > ~4.5
In addition to fish BCF and BAF data, bioaccumulation data for aquatic invertebrate species were also considered. Biota-sediment/soil accumulation factors (BSAFs), trophic magnification factors and biomagnification factors were also considered in characterizing bioaccumulation potential.
Overall, there is consistent empirical and predicted evidence to suggest that the following components have the potential for high bioaccumulation, with BAF/BCF values greater than 5000: C13–C15 isoalkanes, C12 alkenes, C12–C15 one-ring cycloalkanes, C12 and C15 two-ring cycloalkanes, C14 polycycloalkanes, C15 one-ring aromatics, C15 and C20 cycloalkane monoaromatics, C12–C13 diaromatics, C20 cycloalkane diaromatics, and C14 and C20 three-ring PAHs
These components are associated with a slow rate of metabolism and are highly lipophilic. Exposures from water and diet, when combined, suggest that the rate of uptake would exceed that of the total elimination rate. Most of these components are not expected to biomagnify in aquatic or terrestrial foodwebs, largely because a combination of metabolism, low dietary assimilation efficiency and growth dilution allows the elimination rate to exceed the uptake rate from the diet; however,
one study suggests that some alkyl-PAHs may biomagnify.While only BSAFs were found for some PAHs, it is possible that BSAFs will be > 1 for invertebrates, given that they do not have the same metabolic competency as fish.
In general, fish can efficiently metabolize aromatic compounds. There is some evidence that alkylation increases bioaccumulation of naphthalene but it is not known if this can be generalized to larger PAHs or if any potential increase in bioaccumulation due to alkylation will be sufficient to exceed a BAF/BCF of 5000.
Some lower trophic level organisms (i.e., invertebrates) appear to lack the capacity to efficiently metabolize aromatic compounds, resulting in high bioaccumulation potential for some aromatic components as compared to fish.
This is the case for the C14 three-ring PAH, which was bioconcentrated to a high level (BCF > 5000) by invertebrates but not by fish. There is potential for such bioaccumulative components to reach toxic levels in organisms if exposure is continuous and of sufficient magnitude, though this is unlikely in the water column following a spill scenario due to relatively rapid dispersal
Bioaccumulation of aromatic compounds might be lower in natural environments than what is observed in the laboratory. PAHs may sorb to organic material suspended in the water column (dissolved humic material), which decreases their overall bioavailability primarily due to an increase in size. This has been observed with fish
Ecotoxicity:
Diesel fuel studies in salt water are available. The values varied greatly for aquatic species such as rainbow trout and Daphnia magna, demonstrating the inherent variability of diesel fuel compositions and its effects on toxicity. Most experimental acute toxicity values are above 1 mg/L. The lowest 48-hour LC50 for salmonids was 2.4 mg/L. Daphnia magna had a 24-hour LC50 of 1.8 mg/.The values varied greatly for aquatic species such as rainbow trout and Daphnia magna, demonstrating the inherent variability of diesel fuel compositions and its effects on toxicity. Most experimental acute toxicity values are above 1 mg/L. The lowest 48-hour LC50 for salmonids was 2.4 mg/L. Daphnia magna had a 24-hour LC50 of 1.8 mg/L
The tropical mysid Metamysidopsis insularis was shown to be very sensitive to diesel fuel, with a 96-hour LC50 value of 0.22 mg/L this species has been shown to be as sensitive as temperate mysids to toxicants. However,However this study used nominal concentrations,and therefore was not considered acceptable. In another study involving diesel fuel, the effect on brown or common shrimp (Crangon crangon) a 96-hour LC50 of 22 mg/L was determined. A “gas oil”was also tested and a 96-hour LC50 of 12 mg/L.was determined
The steady state cell density of marine phytoplankton decreased with increasing concentrations of diesel fuel, with different sensitivities between species . The diatom Phaeodactylum tricornutum showed a 20% decrease in cell density in 24 hours following a 3 mg/L exposure with a 24-hour no-observed effect concentration (NOEC) of 2.5 mg/L. The microalga Isochrysis galbana was more tolerant to diesel fuel, with a 24-hour lowest-observed-effect concentration (LOEC) of 26 mg/L (14% decrease in cell density), and a NOEC of 25 mg/L.
Finally, the green algae Chlorella salina was relatively insensitive to diesel fuel contamination, with a 24-hour LOEC of 170 mg/L (27% decrease in cell density), and a NOEC of 160 mg/L . All populations of phytoplankton returned to a steady state within 5 days of exposure
In sandy soils, earthworm (Eisenia fetida) mortality only occurred at diesel fuel concentrations greater than 10 000 mg/kg, which was also the concentration at which sub-lethal weight loss was recorded
Nephrotoxic effects of diesel fuel have been documented in several animal and human studies.Some species of birds (mallard ducks in particular) are generally resistant to the toxic effects of petrochemical ingestion, and large amounts of petrochemicals are needed in order to cause direct mortality

For copper: Ecotoxicity - Significant effects are expected on various species of microalgae, some species of macroalgae, and a range of invertebrates, including crustaceans, gastropods and sea urchins. Copper is moderately toxic to crab and their larvae and is highly toxic to gastropods (mollusks, including oysters, mussels and clams). In fish, the acute lethal concentrations of copper depends both on test species and exposure conditions. Waters with high concentrations of copper can have significant effects on diatoms and sensitive invertebrates, notably cladocerans (water fleas). Most taxonomic groups of macroalgae and invertebrates will be severely affected.
For Copper: Typical foliar levels of copper are: Uncontaminated soils (0.3-250 mg/kg) ; Contaminated soils (150-450 mg/kg) ; Mining/smelting soils (6.1-25 mg/kg80 mg/kg300 mg/kg).
Terrestrial Fate: Plants - Generally, vegetation reflects soil copper levels in its foliage. This is dependent upon the bioavailability of copper and the physiological requirements of species concerned. Crops are often more sensitive to copper than the native flora. Soil: In soil, copper levels are raised by application of fertilizer, fungicides, from deposition of highway dusts and from urban, mining and industrial sources. Chronic and or acute effects on sensitive species occur as a result of human activities such as copper fertilizer addition and addition of sludge. When soil levels exceed 150 mg Cu/kg, native and agricultural species show chronic effects. Soils in the range 500-1000 mg Cu/kg act in a strongly selective fashion allowing the survival of only copper-tolerant species and strains. At 2000 Cu mg/kg, most species cannot survive. By 3500 mg Cu/kg, areas are largely devoid of vegetation cover. The organic content of the soil appears to be a key factor affecting the bioavailability of copper. On normal forest soils, non-rooted plants such as mosses and lichens show higher copper concentrations. The fruiting bodies and mycorrhizal sheaths of soil fungi associated with higher plants in forests often accumulate copper to much higher levels than plants at the same site.

Continued...

For Xylenes:
log Koc : 2.05-3.08; Koc : 25.4-204; Half-life (hr) air : 0.24-42; Half-life (hr) H2O surface water : 24-672; Half-life (hr) H2O ground : 336-8640; Half-life (hr) soil : 52-672; Henry's Pa m3 /mol : 637-879; Henry's atm m3 /mol - 7.68E-03; BOD 5 if unstated - 1.4,1%; COD - 2.56,13% ThOD - 3.125 : BCF : 23; log BCF : 1.17-2.41.
Environmental Fate: Most xylenes released to the environment will occur in the atmosphere and volatilisation is the dominant environmental fate process. Soil - Xylenes are expected to have moderate mobility in soil evaporating rapidly from soil surfaces. The extent of the degradation is expected to depend on its concentration, residence time in the soil, the nature of the soil, and whether resident microbial populations have been acclimated. Xylene can remain below the soil surface for several days and may travel through the soil profile and enter groundwater. Soil and water microbes may transform it into other, less harmful compounds, although this happens slowly. It is not clear how long xylene remains trapped deep underground in soil or groundwater, but it may be months or years.
Atmospheric Fate: Xylene evaporates quickly into the air from surface soil and water and can remain in the air for several days until it is broken down by sunlight into other less harmful chemicals. In the ambient atmosphere, xylenes are expected to exist solely in the vapour phase. Xylenes are degraded in the atmosphere with an estimated atmospheric lifetime of about 0.5 to 2 days. Xylene may contribute to photochemical smog formation. p-Xylene has a moderately high photochemical reactivity under smog conditions, higher than the other xylene isomers. The photooxidation of p-xylene results in the production of carbon monoxide, formaldehyde, glyoxal, methylglyoxal, 3-methylbenzylnitrate, m-tolualdehyde, 4-nitro-3-xylene, 5-nitro-3-xylene, 2,6-dimethyl-p-benzoquinone, 2,4-dimethylphenol, 6-nitro-2,4-dimethylphenol, 2,6-dimethylphenol, and 4-nitro-2,6-dimethylphenol.
Aquatic Fate: p-xylene may adsorb to suspended solids and sediment in water and is expected to volatilise from water surfaces. Estimated volatilisation half-lives for a model river and model lake are 3 hours and 4 days, respectively. Measurements taken from goldfish, eels and clams indicate that bioconcentration in aquatic organisms is low. Photo-oxidation in the presence of humic acids may play an important role in the abiotic degradation of p-xylene. p-Xylene is biodegradable and has been observed to degrade in pond water however; it is unclear if it degrades in surface waters. p-Xylene has been observed to degrade in anaerobic and aerobic groundwater; however, it is known to persist for many years in groundwater, at least at sites where the concentration might have been quite high.
Ecotoxicity: Xylenes are slightly toxic to fathead minnow, rainbow trout and bluegill and not acutely toxic to water fleas. For Photobacterium phosphoreum EC50 (24 h): 0.0084 mg/L. and Gammarus lacustris LC50 (48 h): 0.6 mg/L.
The isothiazolinones are very toxic to marine organisms (fish, Daphnia magna and algae)
The high water solubility and low log Kow values of several chlorinated and non-chlorinated indicate a low potential for bioaccumulation.
Studies of 5-chloro-2-methyl-4-isothiazolin-3-one (CMI) in bluegill sunfish (Lepornis machrochirus) show BCF values of 102, 114 and 67 at nominal concentrations of 0.02, 0.12 and 0.8 mg/l. The BCF for 2-methyl-4-isothiazolin-3-one (MI) was determined at 2.3 at a nominal concentration of 0.12 mg/l
Primary biodegradation of MI and CMI occurred with half-lives of less than 24 hours in aerobic and anoxic sediments, and within a period of less than one week the parent compounds were depleted to very low levels that could not be clearly distinguished from analytical artifacts. The ultimate aerobic biodegradability of both MI and CMI attained levels of > 55% within 29 days. Furthermore, the proposed metabolites of MI and CMI are considered to have a low aquatic toxicity on the basis of QSAR estimates and the measured toxicity of the structurally related N-(n-octyl) malonamic acid.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
copper 8-quinolinol	HIGH	HIGH
4,5-dichloro-2-octyl-3(2H)-isothiazolone	HIGH	HIGH
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
copper 8-quinolinol	LOW (LogKOW = 0.5382)
4,5-dichloro-2-octyl-3(2H)-isothiazolone	HIGH (LogKOW = 4.7295)
xylene	MEDIUM (BCF = 740)
distillates, petroleum, light, hydrotreated	LOW (BCF = 159)

Mobility in soil

Ingredient	Mobility
copper 8-quinolinol	LOW (KOC = 4649000)
4,5-dichloro-2-octyl-3(2H)-isothiazolone	LOW (KOC = 5796)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ 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.

Continued...

	▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. 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. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) 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 applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	NO
HAZCHEM	•3Z

Land transport (ADG)

UN number	3082
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains copper 8-quinolinol)
Transport hazard class(es)	Class: 9 Subrisk: Not Applicable
Packing group	III
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: 274 331 335 375 AU01 Limited quantity: 5 L

Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082
are not subject to this Code when transported by road or rail in;
(a) packagings;
(b) IBCs; or
(c) any other receptacle not exceeding 500 kg(L).
- Australian Special Provisions (SP AU01) - ADG Code 7th Ed.

Air transport (ICAO-IATA / DGR)

UN number	3082
UN proper shipping name	Environmentally hazardous substance, liquid, n.o.s. (contains copper 8-quinolinol)
Transport hazard class(es)	ICAO/IATA Class: 9 ICAO / IATA Subrisk: Not Applicable ERG Code: 9L
Packing group	III
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: A97 A158 A197 A215

Continued...

Cargo Only Packing Instructions	964
Cargo Only Maximum Qty / Pack	450 L
Passenger and Cargo Packing Instructions	964
Passenger and Cargo Maximum Qty / Pack	450 L
Passenger and Cargo Limited Quantity Packing Instructions	Y964
Passenger and Cargo Limited Maximum Qty / Pack	30 kg G

Sea transport (IMDG-Code / GGVSee)

UN number	3082
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains copper 8-quinolinol)
Transport hazard class(es)	IMDG Class: 9 IMDG Subrisk: Not Applicable
Packing group	III
Environmental hazard	Not Applicable
Special precautions for user	EMS Number: F-A, S-F Special provisions: 274 335 969 Limited Quantities: 5 L

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
copper 8-quinolinol	Not Available
4,5-dichloro-2-octyl-3(2H)-isothiazolone	Not Available
xylene	Not Available
aromatic hydrocarbons	Not Available
distillates, petroleum, light, hydrotreated	Not Available
paraffinic distillate, heavy, solvent-dewaxed (severe)	Not Available
phosphoric esters	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
copper 8-quinolinol	Not Available
4,5-dichloro-2-octyl-3(2H)-isothiazolone	Not Available
xylene	Not Available
aromatic hydrocarbons	Not Available
distillates, petroleum, light, hydrotreated	Not Available
paraffinic distillate, heavy, solvent-dewaxed (severe)	Not Available
phosphoric esters	Not Available

SECTION 15 Regulatory information

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

copper 8-quinolinol is found on the following regulatory lists

Continued...

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

4,5-dichloro-2-octyl-3(2H)-isothiazolone is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)

xylene is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

distillates, petroleum, light, hydrotreated is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
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 1: Carcinogenic to humans

paraffinic distillate, heavy, solvent-dewaxed (severe) is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	No (copper 8-quinolinol; 4,5-dichloro-2-octyl-3(2H)-isothiazolone)
Canada - NDSL	No (xylene; aromatic hydrocarbons; distillates, petroleum, light, hydrotreated; paraffinic distillate, heavy, solvent-dewaxed (severe); phosphoric esters)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (phosphoric esters)
Vietnam - NCI	Yes
Russia - FBEPH	No (copper 8-quinolinol; 4,5-dichloro-2-octyl-3(2H)-isothiazolone)
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	28/11/2022
Initial Date	04/12/2017

Continued...

SDS Version Summary

Version	Date of Update	Sections Updated
1.17	28/11/2022	Physical Properties, Use

Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.
The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

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

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end of SDS