EMPIRE Markers

Techtronic Industries Australia Pty Ltd

Chemwatch Hazard Alert Code: **3**

Chemwatch: **5412-35**
Version No: **2.1.1.1**
Safety Data Sheet according to WHS and ADG requirements

Issue Date: **22/07/2020**
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SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	EMPIRE Markers
Synonyms	EMFINEB EMPIRE Worksite Marker – Fine Black 1PC; EMFINEB-4PK EMPIRE Worksite Marker – Fine Black 4PK; EMFINECR EMPIRE Worksite Marker – Fine Red 1PC; EMFINECB EMPIRE Worksite Marker – Fine Blue 1PC; EMFINEC-4PK EMPIRE Worksite Marker – Fine Coloured 4PK; EMMEDB EMPIRE Worksite Marker – Medium Black 1PC; EMMEDB-CT EMPIRE Worksite Marker – Chisel Tip Black 1PC
Other means of identification	Not Available

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

Relevant identified uses	Worksite Markers. Use according to manufacturer's directions.

Details of the supplier of the safety data sheet

Registered company name	**Techtronic Industries Australia Pty Ltd**	**Techtronic Industries N.Z. Limited**
Address	31 Gilby Road Mount Waverley VIC 3149 Australia	Unit C, 70 Business Parade South Highbrook Auckland 2013 New Zealand
Telephone	1300 361 505	0800 279 624
Fax	Not Available	+64 800 479 624
Website	http://www.empiretools.com.au/	http://www.empiretools.co.nz/
Email	customerservice@ttibrands.com.au	customerservice@ttibrands.com.au

Emergency telephone number

Association / Organisation	**Poison Information Centre**	**Poison Information Centre (New Zealand)**
Emergency telephone numbers	13 11 26 (24 hours a day, seven days a week, Australia)	0800 764 766 (New Zealand)
Other emergency telephone numbers	0800 764 766 (New Zealand)	Not Available

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Skin Corrosion/Irritation Category 1B, Serious Eye Damage Category 1, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Aspiration Hazard Category 1, Acute Aquatic Hazard Category 3, Chronic Aquatic 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**

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Hazard statement(s)

H314	Causes severe skin burns and eye damage.
H336	May cause drowsiness or dizziness.
H304	May be fatal if swallowed and enters airways.
H412	Harmful to aquatic life with long lasting effects.
AUH018	In use, may form flammable/explosive vapour/air mixture.

Precautionary statement(s) Prevention

P260	Do not breathe mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P273	Avoid release to the environment.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER or doctor/physician.
P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.
P303+P361+P353	IF ON SKIN (or hair): Remove/Take off immediately all contaminated clothing. Rinse skin with water/shower.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P321	Specific treatment (see advice on this label).
P363	Wash contaminated clothing before reuse.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.

Precautionary statement(s) Storage

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

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
9003-07-0	40-60	polypropylene
9003-56-9	20-40	styrene/ butadiene/ acrylonitrile copolymer
71-23-8	1-20	n-propanol
9004-34-6	1-10	cellulose
8032-32-4.	1-10	petroleum ether
107-98-2	1-10	propylene glycol monomethyl ether - alpha isomer
12645-31-7	0.1-10	octyl acid phosphate
Not Available	0.1-1	acrylic (nib)
84281-86-7	<1	C.I. Solvent Violet 8.
495-54-5	<1	C.I. Solvent Orange 3, base

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Immediately hold eyelids apart and flush the eye continuously with running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally

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	lifting the upper and lower lids. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin or hair contact occurs: ▸ Immediately flush body and clothes with large amounts of water, using safety shower if available. ▸ Quickly remove all contaminated clothing, including footwear. ▸ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▸ Transport to hospital, or doctor. For thermal burns: ▸ Decontaminate area around burn. ▸ Consider the use of cold packs and topical antibiotics. For first-degree burns (affecting top layer of skin) ▸ Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides. ▸ Use compresses if running water is not available. ▸ Cover with sterile non-adhesive bandage or clean cloth. ▸ Do NOT apply butter or ointments; this may cause infection. ▸ Give over-the counter pain relievers if pain increases or swelling, redness, fever occur. For second-degree burns (affecting top two layers of skin) ▸ Cool the burn by immerse in cold running water for 10-15 minutes. ▸ Use compresses if running water is not available. ▸ Do NOT apply ice as this may lower body temperature and cause further damage. ▸ Do NOT break blisters or apply butter or ointments; this may cause infection. ▸ Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape. To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort): ▸ Lay the person flat. ▸ Elevate feet about 12 inches. ▸ Elevate burn area above heart level, if possible. ▸ Cover the person with coat or blanket. ▸ Seek medical assistance. For third-degree burns Seek immediate medical or emergency assistance. In the mean time: ▸ Protect burn area cover loosely with sterile, nonstick bandage or, for large areas, a sheet or other material that will not leave lint in wound. ▸ Separate burned toes and fingers with dry, sterile dressings. ▸ Do not soak burn in water or apply ointments or butter; this may cause infection. ▸ To prevent shock see above. ▸ For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway. ▸ Have a person with a facial burn sit up. ▸ Check pulse and breathing to monitor for shock until emergency help arrives. In case of burns: ▸ Immediately apply cold water to burn either by immersion or wrapping with saturated clean cloth. ▸ **DO NOT** remove or cut away clothing over burnt areas. **DO NOT** pull away clothing which has adhered to the skin as this can cause further injury. ▸ **DO NOT** break blister or remove solidified material. ▸ Quickly cover wound with dressing or clean cloth to help prevent infection and to ease pain. ▸ For large burns, sheets, towels or pillow slips are ideal; leave holes for eyes, nose and mouth. ▸ **DO NOT** apply ointments, oils, butter, etc. to a burn under any circumstances. ▸ Water may be given in small quantities if the person is conscious. ▸ Alcohol is not to be given under any circumstances. ▸ Reassure. ▸ Treat for shock by keeping the person warm and in a lying position. ▸ Seek medical aid and advise medical personnel in advance of the cause and extent of the injury and the estimated time of arrival of the patient.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor.
Ingestion	▸ For advice, contact a Poisons Information Centre or a doctor at once. ▸ Urgent hospital treatment is likely to be needed. ▸ 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.

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	▸ Transport to hospital or doctor without delay. ▸ 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

Treat symptomatically.

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

- Do NOT direct a solid stream of water or foam into burning molten material; this may cause spattering and spread the fire.
- Alcohol stable 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	**WARNING:** In use may form flammable/ explosive vapour-air mixtures. Once acrylic fibre is ignited, an exothermic reaction can occur in the absence of oxygen with evolution of hazardous materials. ▸ Combustible. ▸ Slight fire hazard when exposed to heat or flame. ▸ Acids may react with metals to produce hydrogen, a highly flammable and explosive gas. ▸ Heating may cause expansion or decomposition leading to violent rupture of containers. ▸ May emit acrid smoke and corrosive fumes. Combustion products include: carbon dioxide (CO_2) nitrogen oxides (NO_x) phosphorus oxides (PO_x) other pyrolysis products typical of burning organic material. **NOTE:** Burns with intense heat. Produces melting, flowing, burning liquid and dense acrid black smoke. May emit poisonous fumes. May emit corrosive fumes. Fines were retained in a filter trap upstream of a centrifugal fan after polypropylene powder was conveyed by suction through a duct system as an air dispersion. A relatively course filter however very fine powder to pass ad it was eventually retained over a long period in a silencer at the fan outlet. The thickening deposit eventually self-heated and ignited, with the fire spreading very rapidly in the air stream. A second fire, also apparently with polypropylene powder, occurred in a flash-dryer and cyclone system. Washing the dryer case with water had led to a build-up of aggregated powder which had degraded, melted and ignited. Nitrogen purging was introduced as a preventative measure. **CARE**: Contamination of heated / molten liquid with water may cause violent steam explosion, with scattering of hot contents.
HAZCHEM	Not Applicable

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

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Methods and material for containment and cleaning up

Minor Spills	Environmental hazard - contain spillage. ▸ Remove all ignition sources. ▸ 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. Moderate hazard. ▸ Clear area of personnel and move upwind. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear breathing apparatus plus protective gloves. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ No smoking, naked lights or ignition sources. ▸ Increase ventilation. ▸ Stop leak if safe to do so. ▸ Contain spill with sand, earth or vermiculite. ▸ Collect recoverable product into labelled containers for recycling. ▸ Absorb remaining product with sand, earth or vermiculite. ▸ Collect solid residues and seal in labelled drums for disposal. ▸ Wash area and prevent runoff into drains. ▸ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	▸ **DO NOT** allow clothing wet with material to stay in contact with skin ▸ 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.
Other information	▸ Store in original containers. ▸ Keep containers securely sealed. ▸ No smoking, naked lights or ignition sources. ▸ 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. ▸ Polyethylene or polypropylene container. ▸ Packing as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▸ Avoid reaction with oxidising agents, bases and strong reducing agents. ▸ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

Continued...

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	n-propanol	Propyl alcohol	200 ppm / 492 mg/m3	614 mg/m3 / 250 ppm	Not Available	Not Available
Australia Exposure Standards	cellulose	Cellulose (paper fibre)	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	petroleum ether	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	propylene glycol monomethyl ether - alpha isomer	Propylene glycol monomethyl ether	100 ppm / 369 mg/m3	553 mg/m3 / 150 ppm	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
polypropylene	Polypropylene	5.2 mg/m3	58 mg/m3	350 mg/m3
n-propanol	n-Propanol (Propyl alcohol, n-)	250 ppm	670 ppm	4000* ppm
petroleum ether	Petroleum distillates; petroleum ether; includes clay-treated light naphthenic [64742-45-6]; low boiling [68477-31-6]; petroleum extracts [64742-06-9]; petroleum base oil [64742-46-7]; petroleum 50 thinner, petroleum spirits [64475-85-0], Soltrol, VM&P naphtha [8032-32-4]; Ligroine, and paint solvent; petroleum paraffins C5-C20 [64771-72-8]; hydrotreated light naphthenic [64742-53-6]; solvent refined light naphthenic [64741-97-5]; and machine coolant 1	1,100 mg/m3	1,800 mg/m3	40,000 mg/m3
propylene glycol monomethyl ether - alpha isomer	Propylene glycol monomethyl ether; (Ucar Triol HG-170)	100 ppm	160 ppm	660 ppm

Ingredient	Original IDLH	Revised IDLH
polypropylene	Not Available	Not Available
styrene/ butadiene/ acrylonitrile copolymer	Not Available	Not Available
n-propanol	800 ppm	Not Available
cellulose	Not Available	Not Available
petroleum ether	2,500 mg/m3	Not Available
propylene glycol monomethyl ether - alpha isomer	Not Available	Not Available
octyl acid phosphate	Not Available	Not Available
C.I. Solvent Violet 8.	Not Available	Not Available
C.I. Solvent Orange 3, base	Not Available	Not Available

OCCUPATIONAL EXPOSURE BANDING

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
octyl acid phosphate	C	> 1 to ≤ 10 parts per million (ppm)
C.I. Solvent Violet 8.	E	≤ 0.01 mg/m³
C.I. Solvent Orange 3, base	E	≤ 0.01 mg/m³
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

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
NOTE H: Special requirements exist in relation to classification and labelling of this substance. This note applies to certain coal- and oil -derived substances and to certain entries for groups of 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

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Appropriate engineering controls	General exhaust is adequate under normal operating conditions.
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 ▸ When handling corrosive liquids, wear trousers or overalls outside of boots, to avoid spills entering boots. **NOTE:** ▸ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▸ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. 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, · 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. ▸ When handling hot materials wear heat resistant, elbow length gloves. ▸ Rubber gloves are not recommended when handling hot objects, materials ▸ Protective gloves eg. Leather gloves or gloves with Leather facing
Body protection	See Other protection below
Other protection	▸ When handling hot or molten liquids, wear trousers or overalls outside of boots, to avoid spills entering boots. ▸ Overalls.

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	▸ 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:
EMPIRE Markers

Material	CPI
NEOPRENE	A
BUTYL	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PVC	C
TEFLON	C
VITON	C

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

Respiratory protection

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

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

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

For molten materials:

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Black liquid; partly mixes with water.

Physical state	Liquid	**Relative density (Water = 1)**	Not Available
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available

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Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Partly miscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 STABILITY AND REACTIVITY

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

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	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 of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual. Cellulose, after a single intratracheal dose (15 mg per animal) brought about fibrosing granulomatous bronchioloalveolitis and an increase of IgA production in the bronchioalveolar lavage. Fibrosing alveolitis showed moderate progression as a function of time. Injury of Type I pneumocytes and incomplete repair of Type II pneumocytes were detected. The damage of alveolar epithelium initiated and activated a series of processes that led to definite pulmonary alterations and pulmonary fibrosis leading to disintegration of the alveolo-capillary morphological functional unit. Tatrai, E. et al: Journal of Applied Toxicology; 16(2) 129-135 (1996) Some health effects associated with wood, cotton, flax, jute and hemp particles or fibres are not attributable to cellulose content but to other substances and/or impurities. Subjects unaccustomed to n-propanol exposure experienced mild irritation of the eyes, nose and throat at 400 ppm. Processing for an overly long time or processing at overly high temperatures may cause generation and release of highly irritating vapours, which irritate eyes, nose, throat, causing red itching eyes, coughing, sore throat. Exposure to aliphatic alcohols with more than 3 carbons may produce central nervous system effects such as headache, dizziness, drowsiness, muscle weakness, delirium, CNS depression, coma, seizure, and neurobehavioural changes. Symptoms are more acute with higher alcohols. Respiratory tract involvement may produce irritation of the mucosa, respiratory insufficiency, respiratory depression secondary to CNS depression, pulmonary oedema, chemical pneumonitis and bronchitis. Cardiovascular involvement may result in arrhythmias and hypotension. Gastrointestinal effects may include nausea and vomiting. Kidney and liver damage may result following massive exposures. The alcohols are potential irritants being, generally, stronger irritants than similar organic structures that lack functional groups (e.g. alkanes) but are much less irritating than the corresponding amines, aldehydes or ketones. Alcohols and glycols (diols) rarely represent serious hazards in the workplace, because their vapour concentrations are usually less than the levels which produce significant irritation which, in turn, produce significant central nervous system effects as well. Inhalation hazard is increased at higher temperatures. Exposure to toxic levels of butadiene has also produced chromosome damage. Human volunteers exposed at 2000-8000 ppm 1,3-butadiene for 6-8 hours showed slight smarting of the eyes, difficulty in focusing on instrument scales and a transient objection to butadiene odour. Characteristics of exposure include dry nose/mouth/throat, fatigue, headache, vertigo, nausea, narcosis, respiratory paralysis, and central nervous system depression. Very high concentrations may cause loss of consciousness or death. Repeated and prolonged exposure to 1,3-butadiene vapour may cause kidney and liver damage. Deep anaesthesia was induced in rabbits in 8 to 10 minutes at 200000 to 250000 ppm. Recovery from brief periods of anaesthesia occurred within two minutes of terminating the exposure.
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.
Skin Contact	The material can produce chemical burns following direct contact with the skin. Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. The calculated human skin permeability coefficient for n-propanol is 1.3 x 10-3 cm/hr (U.S. Environment Protection Agency) The diepoxide of butadiene (1,2:3,4-diepoxybutane), a probable metabolite, has been reported to be a mild skin tumourigen when applied topically to the skin of mice Most liquid alcohols appear to act as primary skin irritants in humans. Significant percutaneous absorption occurs in rabbits but not apparently in man. Toxic amounts of for propylene glycol monomethyl ether (PGME) may be absorbed through the skin following extensive prolonged contact ; this may result in drowsiness. Constant contact with the beta-isomer, on the skin of rabbits, for several weeks

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	caused very mild, simple irritation. Dose rates of 10 mg/kg produced incomplete anaesthesia, depression, and slight increase in kidney weights in test animals. 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	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
Chronic	Repeated or prolonged exposure to corrosives may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant 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 exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals. The celluose derivatives pass essentially unchanged through the gastrointestinal tract following oral administration to rats, dogs and man. Acute, subchronic, chronic toxicity, reproductive and developmental toxicity, genotoxicity and carcinogenicity studies of cellulose derivatives indicated that they are practically non-toxic when administered by oral, intraperitoneal, subcutaneous or dermal routes. While no clinical inhalation studies have been conducted, long term exposure to the dusts of cellulose ethers in manufacturing operations has not lead to any significant adverse effects. Ocular and dermal irritation studies indicate that the cellulose derivatives are, at most, minimally irritating and are not dermal sensitisers. Clinical studies confirm these results. Amended Safety Assessment of Cellulose and Related Polymers as used in Cosmetics: Final Report of the Cosmetic Ingredient Review (CIR) Expert Panel: March 2009 Inhalation studies indicate that cellulose fibres may be fibrogenic; this finding continues to be the subject of extensive research. Cellulose is not considered an inert substance because : · in rats, it causes granulomatous fibrosing alveolitis at the end of the third month after exposure, · in rats there was an increase in the secretion of plasminogen activator and interleukin 1 as well as the release of lactate dehydrogenase from macrophages, in a manner similar to asbestos, · there were increases in the incidence of obstructive lung diseases and bronchial asthma in humans at work and in the residential environment where exposure to cellulose was common, · the substance may induce free radical production in human leucocytes. Byssinosis is an occupational disease of the lungs caused by inhalation of cotton dust or dusts from other vegetable fibres such as flax, hemp, or sisal. Byssinosis is a chronic, asthma-like narrowing of the airways. Also called brown lung disease, byssinosis occurs almost exclusively in people who work with unprocessed cotton. Cotton dust disease, "byssinosis", is well known among cotton mill workers. Cotton dust consists largely of cellulose fibre. Exposure to two components of the total dust, the "respirable" and "medium" fraction correlated significantly with the prevalence of respiratory symptoms. Inhalation exposure to a concentration of 0.3 to 0.4 mg/m3 of "fly-free" dust results in a 20% occurrence of byssinosis. "Fly-free" dust is the sum of respirable and medium-length fibres. At 0.46 mg/m3, Grade II byssinosis occurs. A byssinosis (all grades) prevalence of 20%, at 0.3 mg/m3 occurs when the fibre length is less than 15 um (aerodynamic equivalent diameter). Byssinosis is not caused by mechanical irritation but by reactions caused by pharmacologically active substances producing oedema or contraction of the smooth musculature of the airways. The causative agent is suspected to be an endotoxin, in turn, thought to be a cell wall component of bacteria found in cotton. Symptoms of byssinosis include chest tightness, wheezing and dyspnoea. Symptoms usually appear after an absence from work and may subside after 2-days of exposure. As the disease progresses, symptoms may persist for longer periods until they are constant. The individual may eventually exhibit chronic bronchitis and emphysema. Increased physical exertion may produce shortness of breath. Studies with some glycol ethers (principally the monoethylene glycols) and their esters indicate reproductive changes, testicular atrophy, infertility and kidney function changes. The metabolic acetic acid derivatives of glycol ethers (alkoxyacetic acids), not the ether itself, have been found to be the proximal reproductive toxin in animals. The potency of these metabolites decreases significantly as the chain length of the ether increases. Consequently glycol ethers with longer substituents (e.g diethylene glycols, triethylene glycols) have not generally been associated with reproductive effects. One of the most sensitive indicators of toxic effects observed from many of the glycol ethers is an increase in the erythrocytic osmotic fragility in rats Which produces haemolytic anaemia). This appears to be related to the development of haemoglobinuria (blood in the urine) at higher exposure levels or as a result of chronic exposure. Glycol ethers based on propylene oxides, propylene glycol ethers, dipropylene glycol ethers and tripropylene glycol ethers are mainly available, commercially, as alpha-isomers (because of thermodynamic considerations); these are incapable of forming alkoxyacetic or alkoxypropionic acids as metabolites and therefore do not produce erythrocyte fragility unless contaminated by ethylene glycol ethers or to a significant degree by the beta-isomer . beta-Isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects). Acrylonitrile is a skin and respiratory sensitiser. Chronic exposures may produce severe liver inflammation. Chronic effects of occupational exposure include skin and eye irritation, nausea, vomiting, weakness, fatigue, jaundice, anaemia, leukocytosis, bilirubinaemia, increased serum thiocyanate concentrations, and hepatic and renal irritation. When administered orally (by gavage or in drinking water), acrylonitrile induced increased incidences of fore-stomach squamous cell papillomas, central nervous system microgliomas, mammary gland carcinomas and Zymbal gland carcinomas, forestomach papillomas and acanthomas, and central nervous system neoplasms in rats of both sexes. An epidemiological study of textile-plant workers potentially exposed to acrylonitrile and observed for 20 years, showed an increased incidence of cancers of the lung; further follow-up of this cohort revealed a continued excess of lung cancer, although during the actual 5-year follow-up period, there was no excess. This follow-up showed a significant excess of cancer of the prostate. *NTP*

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Studies indicate that diets containing large amounts of non-absorbable polysaccharides, such as cellulose, might decrease absorption of calcium, magnesium, zinc and phosphorus.
The material contains a substantial proportion of a polymer considered to be of low concern (PLC). The trend towards production of lower molecular weight polymers (thus reducing the required level of solvent use and creating a more "environmentally-friendly" material) has brought with it the need to define PLCs as those
having molecular weights of between 1000 and 10000 and containing less than 10% of the molecules with molecular weight below 500 and less than 25% of the molecules with a molecular weight below 1000. These may contain unlimited low concern functional groups or moderate concern reactive functional groups with a combined functional group equivalent weight (FGEW, a concept developed by the US EPA describing whether the reactive functional group is sufficiently diluted by polymeric material) of a 1000 or more (provided no high concern groups are present) or high concern reactive functional groups with a FGEW of 5000 or more (FGEW includes moderate concern groups if present).
having molecular weights exceeding 10000 (without restriction on reactive groups).
inhalation of polymers with molecular weights > 70,000 Da has been linked with irreversible lung damage due to lung overloading and impaired clearance of particles from the lung, particularly following repeated exposure. If the polymer is inhaled at low levels and/or infrequently, it is assumed that it will be cleared from the lungs.
Reactive functional groups are in turn classified as being of low, moderate or high concern Classification of the polymer as a PLC, in accordance with established criteria, does not mean that hazards will not be associated with the polymer (during its import, manufacture, use, storage, handling or disposal). The polymer may, for example, contain a large number of particles in the respirable range, a hazard which may need to assessed in the health and safety risk assessment. Similarly a polymer with low concern reactive may be released into the environment in large quantities and produce an environmental hazard.
Whilst it is generally accepted that polymers with a molecular weight exceeding 1000 are unlikely to pass through biological membranes, oligomers with lower molecular weight and specifically, those with a molecular weight below 500, may. Estimations based on a "highly" dispersed polymer population (polydispersity = 10) suggests that the molecular weight of the polymer carrying a reactive group of high concern must be 5000 to be considered a PLC; similarly a polymer of approximate molecular weight 1000 could contain no more than one reactive group of moderate concern (for two moderate concern groups, the molecular weight would be about 2500).
When administered to rats by intubation (0.3 ml/kg twice each week for a total dosage of 50 ml) n-propanol produced severe liver injury and hyperplasia, malignant tumours (myeloid leukaemia, liver sarcomas, liver cell carcinoma) and benign tumours. When administered by subcutaneous injection an increased incidence of malignant and benign tumours was recorded
Amongst humans occupationally exposed to 1,3-butadiene several cancer sites with high statistically significant mortality ratios were identified.
These included cancer of the testes, cancers of the digestive system (oesophagus, stomach, large intestine), larynx and Hodgkin's disease.
Exposure by rats to 1,3-butadiene gas at 1000 ppm/6hrs/day, 5 days /week (105 weeks for females and 111 weeks for males) caused significant increases in the incidence of tumours at various sites; mammary gland adenomas and sarcomas; uterine sarcomas; Zymbal gland carcinomas; thyroid adenomas and pancreatic adenomas. A high incidence of malignant lymphoma was found amongst a group of exposed rats in a second study

EMPIRE Markers	TOXICITY	IRRITATION
	Not Available	Not Available

polypropylene	TOXICITY	IRRITATION
	Oral (rat) LD50: >8000 mg/kg[2]	Not Available

styrene/ butadiene/ acrylonitrile copolymer	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 5010 mg/kg[2]	Not Available
	Oral (rat) LD50: 5010 mg/kg[2]	

n-propanol	TOXICITY	IRRITATION
	dermal (rat) LD50: 4055 mg/kg[2]	Eye (rabbit): 20 mg/24h moderate
	Oral (rat) LD50: 1870 mg/kg[2]	Eye (rabbit): 4 mg open SEVERE
		Skin (rabbit): 20 mg/24h moderate
		Skin (rabbit): 500 mg open mild

cellulose	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg[2]	Not Available
	Inhalation (rat) LC50: >5.8 mg/l/4H[2]	
	Oral (rat) LD50: >5000 mg/kg[2]	

petroleum ether	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1900 mg/kg[1]	Eye (human): 880 ppm/15m
	Inhalation (rat) LC50: 3396.1206 mg/l/4H[2]	Eye: no adverse effect observed (not irritating)[1]
	Oral (rat) LD50: >4500 mg/kg[1]	Skin: adverse effect observed (irritating)[1]

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propylene glycol monomethyl ether - alpha isomer	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (rabbit) 230 mg mild
	Inhalation (rat) LC50: 12485.7375 mg/l/5h.d[2]	Eye (rabbit) 500 mg/24 h. - mild
	Oral (rat) LD50: 3739 mg/kg[2]	Eye (rabbit): 100 mg SEVERE
		Skin (rabbit) 500 mg open - mild
octyl acid phosphate	**TOXICITY**	**IRRITATION**
	Oral (rat) LD50: 2500 mg/kg[1]	Eye: adverse effect observed (irritating)[1]
		Skin: adverse effect observed (corrosive)[1]
C.I. Solvent Violet 8.	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
C.I. Solvent Orange 3, base	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (rabbit) LD50: 20 mg/24h-mod
	Oral (rat) LD50: 1000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
Legend:	*1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*	

POLYPROPYLENE

* For pyrolyzate
for poly-alpha-olefins (PAOs):
PAOs are highly branched isoparaffinic chemicals produced by oligomerisation of 1-octene, 1-decene, and/or 1-dodecene. The crude polyalphaolefin mixture is then distilled into appropriate product fractions to meet specific viscosity specifications and hydrogenated.
Read across data exist for health effects endpoints from the following similar *hydrogenated* long chain branched alkanes derived from a C8, C10, and/or C12 alpha olefins:

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

The data for these structural analogs demonstrated no evidence of health effects. In addition, there is evidence in the literature that alkanes with 30 or more carbon atoms are unlikely to be absorbed when administered orally. The physicochemical data suggest that it is unlikely that significant absorption will occur. If a substance of the size and structure of a typical PAO is absorbed, then the principal mechanisms of absorption after oral administration are likely to be passive diffusion and absorption by way of the lymphatic system. The former requires both good lipid solubility and good water solubility as the substance has to partition from an aqueous environment through a lipophilic membrane into another aqueous environment during absorption. Absorption by way of the lymphatics occurs by mechanisms analogous to those that absorb fatty acids and is limited by the size of the molecule. Lipophilicity generally enhances the ability of chemicals to cross biological membranes. Biotransformation by mixed function oxidases often increases the water solubility of a substance; however, existing data suggest that these substances will not undergo oxidation to more hydrophilic metabolites. Finally, a chemical must have an active functional group that can interact chemically or physically with the target cell or receptor upon reaching it; there are no moieties in PAOs that represent a functional group that may have biological activity. The water solubilities of a C10 dimer PAO and a C12 trimer PAO were determined to be <1 ppb and < 1 ppt respectively. The partition coefficient for a C12 trimer PAO was determined to be log Kow of >7 . Given the very low water solubility it is extremely unlikely that PAOs will be absorbed by passive diffusion following oral administration, and the size of the molecules suggest that the extent of lymphatic absorption is likely to be very low. Although PAOs are relatively large lipophilic compounds, and molecular size may be a critical limiting determinant for absorption, there is some evidence that these substances are absorbed. However, the lack of observed toxicity in the studies with PAOs suggests that these products are absorbed poorly, if at all. Furthermore, a review of the literature regarding the absorption and metabolism of long chain alkanes indicates that alkanes with 30+ carbon atoms are unlikely to be absorbed. For example the absorption of squalane, an analogous C30 product, administered orally to male CD rats was examined - essentially all of the squalane was recovered unchanged in the faeces. At the same time, the hydrophobic properties of PAOs suggest that, should they be absorbed, they would undergo limited distribution in the aqueous systemic circulation and reach potential target organs in limited concentrations.
In addition to the general considerations discussed above, the low volatility of PAOs indicates that, under normal conditions of use or transportation, exposure by the inhalation route is unlikely. In particular, the high viscosity of these substances suggests that it would be difficult to generate a high concentration of respirable particles in the air.
Acute toxicity: PAOs (decene/dodecene copolymer, octene/decene/dodecene homo-polymer, and dodecene trimer) have been adequately tested for acute oral toxicity. There were no deaths when the test materials were administered at doses of 5,000 mg/kg (decene/dodecene copolymer and dodecene trimer) and at 2,000 mg/kg (octene/decene/dodecene copolymer) in rats. Overall, the acute oral LD50 for these substances was greater than the 2000 mg/kg limit dose, indicating a relatively low order of toxicity.
PAOs (decene/dodecene copolymer, octene/decene/dodecene copolymer, and dodecene trimer) have been tested for acute dermal toxicity. No mortality was observed for any substance when administered at the limit dose of 2000 or 5000 mg/kg. Overall,

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	the acute dermal LD50 for these substances was greater than the 2000 mg/kg limit dose, indicating a relatively low order of toxicity. 1-Decene, homopolymer, is absorbed (unexpectedly for a high molecular weight polymer) to a moderate degree in rat skin and is eliminated slowly PAOs (decene homopolymer, decene/dodecene copolymer, and decene trimer) have been tested for acute inhalation toxicity. Rats were exposed to aerosols of the substances at nominal atmospheric concentrations of 2.5, 5.0, and 5.06 mg/L, respectively, for four hours. These levels were the maximum attainable concentrations under the conditions of the tests, due to the low volatility and high viscosity of the test material. No mortality was noted, and all animals fully recovered following depuration. The lack of mortality at concentrations at or above the limit dose of 2.0 mg/L indicates a relatively low order of toxicity for these substances. **Repeat dose toxicity:** Eight repeated-dose toxicity studies using two different animal species, rats and mice, and oral and dermal routes of administration have been conducted with three structural analogs. These data suggest that the structural analogs exhibit a low order of toxicity following repeated applications, due to their similarity in chemical structures and physicochemical properties. One 28-day oral toxicity study in rats, one 90-day dermal and two 90-day dietary studies in rats, and a dermal carcinogenicity study in mice exist for decene homopolymer. A rat oral combined reproductive toxicity and 91-day systemic toxicity study was also conducted with decene homopolymer. In addition, 28-day rat oral toxicity studies exist for two structurally analogous substances (dodecene trimer and octene/decene/dodecene copolymer); and a 90-day rat dermal toxicity study exists for octene/decene/dodecene copolymer. Results from these studies show a low order of repeated dose toxicity. The dermal NOAEL for systemic toxicity studies was equal to or greater than 2000 mg/kg/day. The oral NOAEL for 1-decene homopolymer is between 5,000 and 20,000 mg/kg/day in Sprague-Dawley rats. Rats exposed repeatedly by dermal exposure at doses of 2000 mg/kg decene/dodecene copolymer showed increased incidences of hyperplasia of the sebaceous glands, hyperplasia/hyperkeratosis of the epidermis and dermal inflammation. These symptoms generally subsided within 2 weeks. Males showed decreased body weight gain and altered serum chemistry. In a 90-day feeding study rats receiving 20000 ppm of 1-decene, homopolymer, hydrogenated did not exhibit any clinical signs of systemic toxicity. Marginal effects on clinical chemistry (glucose and ALT in males; sodium, phosphorus and calcium in females) were seen. **Reproductive toxicity:** Data are available for decene homopolymer. Results from these studies show a low order of reproductive/ developmental toxicity. The NOAEL for reproductive toxicity was 1000 mg/kg/day, the highest concentration tested. The lack of effects on fertility in this study or effects on reproductive organs in this or other subchronic studies with closely related chemicals indicates that PAOs are unlikely to exert effects on reproduction. **Developmental toxicity:** Decene homopolymer (with 10 ppm of an antioxidant) was administered once daily on gestation days 0-19 via dermal application to presumed-pregnant rats at doses of 0, 800, and 2000 mg/kg/day. Dermal administration of the test material did not adversely affect parameters of reproductive performance during gestation, nor did it adversely affect *in utero* survival and development of the offspring. The NOAEL in this study for developmental parameters was 2000 mg/kg/day. **Genotoxicity:** Information for the following PAOs (decene homopolymer, octene/decene/dodecene copolymer, dodecene trimer; and decene/dodecene copolymer [*prepared from 10% C12 and 90% C10 alpha olefins; approx. 33% trimer and 51% tetramer, 16% pentamer and higher*]) is available. Either bacterial or mammalian gene mutation assays, *in vitro* chromosomal aberration assays, or *in vivo* chromosomal aberration assays have been conducted for these substances. Neither mutagenicity nor clastogenicity were exhibited by any of these substances in the referenced *in vivo* or *in vitro* tests, with or without metabolic activation. **Carcinogenicity:** While alpha-olefin polymers have similar properties to mineral oils, they do not contain polycyclic aromatic hydrocarbons, or other known possible carcinogens. Decene homopolymer produced no treatment-related tumors in C3H mice treated with a 50 ul/application twice weekly for 104 weeks. In addition, survival (56%) was greater than in any other group, including the untreated control.
STYRENE/ BUTADIENE/ ACRYLONITRILE COPOLYMER	Ultrafine particles (UFPs) may be produced at lower temperatures during the 3D printing process Concerns have been raised regarding airborne UFP concentrations generated while printing with ABS, as UFPs have been linked with adverse health effects
N-PROPANOL	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.
PETROLEUM ETHER	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

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	liver. for petroleum: Altered mental state, drowsiness, peripheral motor neuropathy, irreversible brain damage (so-called Petrol Sniffer's Encephalopathy), delirium, seizures, and sudden death have been reported from repeated overexposure to some hydrocarbon solvents, naphthas, and gasoline This product may contain benzene which is known to cause acute myeloid leukaemia and n-hexane which has been shown to metabolize to compounds which are neuropathic. This product contains toluene. There are indications from animal studies that prolonged exposure to high concentrations of toluene may lead to hearing loss. This product contains ethyl benzene and naphthalene from which there is evidence of tumours in rodents **Carcinogenicity:** Inhalation exposure to mice causes liver tumours, which are not considered relevant to humans. Inhalation exposure to rats causes kidney tumours which are not considered relevant to humans. **Mutagenicity:** There is a large database of mutagenicity studies on gasoline and gasoline blending streams, which use a wide variety of endpoints and give predominantly negative results. All in vivo studies in animals and recent studies in exposed humans (e.g. petrol service station attendants) have shown negative results in mutagenicity assays. **Reproductive Toxicity:** Repeated exposure of pregnant rats to high concentrations of toluene (around or exceeding 1000 ppm) can cause developmental effects, such as lower birth weight and developmental neurotoxicity, on the foetus. However, in a two-generation reproductive study in rats exposed to gasoline vapour condensate, no adverse effects on the foetus were observed. **Human Effects:** Prolonged/ repeated contact may cause defatting of the skin which can lead to dermatitis and may make the skin more susceptible to irritation and penetration by other materials. Lifetime exposure of rodents to gasoline produces carcinogenicity although the relevance to humans has been questioned. Gasoline induces kidney cancer in male rats as a consequence of accumulation of the alpha2-microglobulin protein in hyaline droplets in the male (but not female) rat kidney. Such abnormal accumulation represents lysosomal overload and leads to chronic renal tubular cell degeneration, accumulation of cell debris, mineralisation of renal medullary tubules and necrosis. A sustained regenerative proliferation occurs in epithelial cells with subsequent neoplastic transformation with continued exposure. The alpha2-microglobulin is produced under the influence of hormonal controls in male rats but not in females and, more importantly, not in humans.
PROPYLENE GLYCOL MONOMETHYL ETHER - ALPHA ISOMER	for propylene glycol ethers (PGEs): Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM). Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids. Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects). This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product. Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body. As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces. As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m3 for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m3. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m3), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating None are skin sensitisers. In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested). Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m3 (600 ppm) for PnB and 2,010 mg/m3 (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m3 (43 ppm).

Continued...

	In this study, the highest tested TPM concentration, 1010 mg/m3 (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members. One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m3) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m3). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m3), with decreased body weights occurring at 3000 ppm (11058 mg/m3). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. in a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health. In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity. The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic *in vivo*. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice. NOTE: For PGE - mixed isomers: Exposure of pregnant rats and rabbits to the substance did not give rise to teratogenic effects at concentrations up to 3000 ppm. Foetotoxic effects were seen in rats but not in rabbits at this concentration; maternal toxicity was noted in both species.
OCTYL ACID PHOSPHATE	for alkyl esters of phosphoric acid: The chemicals in this category exhibit a low to moderate order of acute toxicity. The rat oral LD50 values ranged from 500-1000 mg/kg with 2-ethylhexyl phosphate to >36,800 mg/kg for tris(2-ethylhexyl) phosphate. The dermal LD50 values ranged from 1200 to > 2000 mg/kg (rat) with bis(2-ethylhexyl) hydrogen phosphate to > 20,000 mg/kg (rabbit) with tris(2- ethylhexyl) phosphate. The inhalation LC50 values ranged from > 0.447 mg/l (4 hr. rat) with tris(2-ethylhexyl) phosphate to > 5.14 mg/l **(**4 hr. rat) with triisobutyl phosphate. **Metabolism:** Phosphoric acid esters are metabolized via dealkylation. Metabolism studies conducted on the tributyl phosphate indicate that dealkylation to form the alkyl alcohol is the primary route of metabolism Phosphoric acid tri-esters are rapidly metabolised to di-esters with mono-diesters also being produced. Studies of tributyl phosphate show that 40-64% of the parent compound is metabolised to dibutyl dihydrogen phosphate and that 1.1-2.1 % is metabolised to the monobutyl species. Therefore, tris(2-ethylhexyl) phosphate is expected to be metabolised to bis(2-ethylhexyl) phosphate (CAS RN: 298-07-7) and mono(2-ethylhexyl) phosphate (CAS RN 1070-03-7). Based on the evidence for dealkylation as the primary metabolic pathway, 2-ethylhexanol is the expected metabolite of tris(2-ethylhexyl) phosphate (CAS RN: 78-42-2) and 2-ethylhexyl phosphate (CAS RN: 12645-31-7). Triisobutyl phosphate is expected to be metabolised similarly as tributyl phosphate, with methoxypropanol as the alcohol metabolite Oral repeat dose NOAEL's in rats for dibutyl hydrogen phosphate, tributyl phosphate, ethylhexanol, 2- ethylhexanoic acid, bis(2-ethylhexyl) hydrogen phosphate, tris(2-ethylhexyl) phosphate, and triisobutyl phosphate were 30 mg/kg/day (44 days), 75 mg/kg/day (90 days), 125 mg/kg/day (90 days), 100 mg/kg/day (90 days), 250 mg/kg/day (5 days), and 1000 mg/kg/day (90 days), and 68.4-84.3 mg/kg (90 days), respectively. The weight of the evidence indicates that the members of this category are not genotoxic. Tris(2-ethylhexyl) phosphate, bis(2-ethylhexyl) hydrogen phosphate, 2-ethylhexyl phosphate, dibutyl hydrogen phosphate, tributyl phosphate, triisobutyl phosphate, 2-ethylhexanol, 2- ethylhexanoic acid, and phosphoric acid were negative in the Ames assay. Tris(2-ethylhexyl) phosphate, bis(2-ethylhexyl) phosphate, 2-ethylhexyl phosphate, and 2-ethylhexanol also were negative in the mouse lymphoma assay. Furthermore, tris(2-ethylhexyl) phosphate, dibutyl hydrogen phosphate, tributyl phosphate, and 2-ethylhexanol were negative in the chromosomal aberration assays (in vitro and/ or in vivo). Tris(2-ethylhexyl) phosphate was negative in a sister chromatid exchange assay while 2-ethylhexanoic acid was positive. Triisobutyl phosphate was negative in the in vivo mouse micronucleus assay. **Reproductive toxicity** was evaluated with a number of the members of this category. No effects on reproductive organs were observed in repeat dose studies with tris(2-ethylhexyl) phosphate, dibutyl hydrogen phosphate, tributyl phosphate, 2-ethylhexanol, or 2-ethylhexanoic acid. A two generation reproduction study with tributyl phosphate did not find any reproductive effects in rats at the highest dose tested (225 mg/kg/day). No significant effects on reproduction were seen in rats with an oral OECD 422 combined repeat dose toxicity and reproductive/developmental toxicity screen with dibutyl hydrogen phosphate (NOAEL = 1000 mg/kg). Reproductive effects were reported in rats at 300 mg/kg/day and 600 mg/kg/day in a one generation study with 2-ethylhexanoic acid. **Developmental toxicity**: The developmental toxicity of tributyl phosphate was evaluated in both rats and rabbits. Tributyl phosphate and triisobutyl phosphate were determined not to be teratogenic. 2-Ethylhexanol was found to cause developmental toxicity only at doses that were maternally toxic. Drinking water and gavage developmental toxicity studies have also been conducted with 2-ethylhexanoic acid in rats and rabbits. Developmental effects in rats at concentrations as low as 100 mg/kg administered in drinking water have been reported. Developmental studies with rats and rabbits concluded that 2-ethylhexanoic acid did not produce developmental effects in rats or rabbits under the conditions of these tests. The authors noted that the rat NOAEL was 100 mg/kg/day based on slight foetotoxicity at 250 mg/kg/day and that the rabbit NOAEL was 250 mg/kg/day (highest dose). The maternal NOAEL's for rats and rabbits were 250 mg/kg/day and 25 mg/kg/day, respectively.
C.I. SOLVENT ORANGE 3, BASE	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 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

Continued...

	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. The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. p-Phenylenediamines are oxidised by the liver microsomal enzymes (S9). Pure p-phenylenediamine is non-mutagenic in but becomes mutagenic after it is oxidized. Azo dyes containing phenylenediamine are mutagenic in certain assay most likely due to the formation of oxidized p-phenylenediamine. Modification of the moieties that can be metabolized to p-phenylenediamine by sulfonation, carboxylation or copper complexation eliminated the mutagenic responses. **NOTE:** Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA. Chrysoidin is mutagenic in bacteria when liver enzyme activators are present in the nutrient media; a liver metabolite, 1,2,4-triaminobenzene, one of the reduction products of chrysoidin may be responsible. [Garner & Nutman, Mutation Research, 44, pp 9-19, 1977] A report of bladder cancer in three amateur anglers with exposure to chrysoidine-dyed maggots stimulated reports of four further cases and two case-control studies A study in Yorkshire, UK, used an existing large-scale bladder cancer case-control study (over 900 pairs) and made further enquiries regarding fishing, maggots and dyes used on or in the maggots. The relative risks were 0.7 (95% confidence interval, 0.2-2.3) based on five exposed cases for the use of bronze (surface-coloured) maggots, and 2.0 (0.6-6.2) based on nine exposed cases for yellow maggots (ready or self-coloured) . A study in the West Midlands, UK, was smaller (202 pairs) but showed a higher percentage of use of dyed maggots (14% of cases, 8% of controls). A three-fold excess risk was noted for the use of bronze maggots for more than five years. This study almost certainly included five cases from the previous case reports that stimulated the case-control studies, but this factor is unlikely to remove the statistically significant excess risk
POLYPROPYLENE & STYRENE/ BUTADIENE/ ACRYLONITRILE COPOLYMER	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.
CELLULOSE & OCTYL ACID PHOSPHATE	Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. 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. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.
OCTYL ACID PHOSPHATE & C.I. SOLVENT VIOLET 8.	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

EMPIRE Markers

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
Not Available	Not Available	Not Available	Not Available	Not Available

polypropylene

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	12.237mg/L	3
EC50	96	Algae or other aquatic plants	40.113mg/L	3

styrene/ butadiene/ acrylonitrile copolymer

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	11.5mg/L	4

Continued...

n-propanol	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	3-800mg/L	2
	EC50	48	Crustacea	3-644mg/L	2
	EC50	96	Algae or other aquatic plants	861.193mg/L	3
	NOEC	48	Algae or other aquatic plants	1-150mg/L	2
cellulose	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	9160000mg/L	3
	EC50	96	Algae or other aquatic plants	340000000mg/L	3
petroleum ether	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	4.1mg/L	2
	EC50	48	Crustacea	4.5mg/L	2
	EC50	72	Algae or other aquatic plants	>1-mg/L	2
	LC50	96	Fish	4.1mg/L	2
	EC50	48	Crustacea	4.5mg/L	2
	EC50	72	Algae or other aquatic plants	>1-mg/L	2
	NOEC	72	Algae or other aquatic plants	<0.1mg/L	1
propylene glycol monomethyl ether - alpha isomer	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>=1-mg/L	2
	EC50	48	Crustacea	>=1-mg/L	2
	EC50	96	Algae or other aquatic plants	>1-mg/L	2
	EC0	48	Crustacea	>=1-mg/L	2
	NOEC	48	Crustacea	>=1-mg/L	2
octyl acid phosphate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>100mg/L	2
	EC50	72	Algae or other aquatic plants	15mg/L	2
	NOEC	72	Algae or other aquatic plants	10mg/L	2
C.I. Solvent Violet 8.	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
C.I. Solvent Orange 3, base	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>1mg/L	2
	EC50	48	Crustacea	0.42mg/L	2
	EC50	72	Algae or other aquatic plants	0.17mg/L	2
Legend:	*Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*				

Do NOT 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.
DO NOT discharge into sewer or waterways.
Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
polypropylene	LOW	LOW
n-propanol	LOW	LOW
cellulose	LOW	LOW
propylene glycol monomethyl ether - alpha isomer	LOW (Half-life = 56 days)	LOW (Half-life = 1.7 days)

Continued...

C.I. Solvent Orange 3, base	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
polypropylene	LOW (LogKOW = 1.6783)
n-propanol	LOW (LogKOW = 0.25)
cellulose	LOW (LogKOW = -5.1249)
propylene glycol monomethyl ether - alpha isomer	LOW (BCF = 2)
C.I. Solvent Orange 3, base	LOW (LogKOW = 2.1271)

Mobility in soil

Ingredient	Mobility
polypropylene	LOW (KOC = 23.74)
n-propanol	HIGH (KOC = 1.325)
cellulose	LOW (KOC = 10)
propylene glycol monomethyl ether - alpha isomer	HIGH (KOC = 1)
C.I. Solvent Orange 3, base	LOW (KOC = 874.4)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	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	Not Applicable

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

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

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

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

Continued...

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

POLYPROPYLENE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

STYRENE/ BUTADIENE/ ACRYLONITRILE COPOLYMER IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

N-PROPANOL IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)
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

CELLULOSE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

PETROLEUM ETHER IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

PROPYLENE GLYCOL MONOMETHYL ETHER - ALPHA ISOMER IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)

OCTYL ACID PHOSPHATE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

C.I. SOLVENT VIOLET 8. IS FOUND ON THE FOLLOWING REGULATORY LISTS

Not Applicable

C.I. SOLVENT ORANGE 3, BASE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Chemical Footprint Project - Chemicals of High Concern List

National Inventory Status

National Inventory	Status
Australia - AICS	No (C.I. Solvent Violet 8.)
Canada - DSL	No (C.I. Solvent Violet 8.)
Canada - NDSL	No (polypropylene; styrene/ butadiene/ acrylonitrile copolymer; n-propanol; petroleum ether; propylene glycol monomethyl ether - alpha isomer; octyl acid phosphate; C.I. Solvent Violet 8.; C.I. Solvent Orange 3, base)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (polypropylene; styrene/ butadiene/ acrylonitrile copolymer)
Japan - ENCS	No (cellulose; petroleum ether; C.I. Solvent Violet 8.)
Korea - KECI	No (C.I. Solvent Violet 8.)
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	No (C.I. Solvent Violet 8.)
Taiwan - TCSI	Yes
Mexico - INSQ	No (octyl acid phosphate; C.I. Solvent Violet 8.; C.I. Solvent Orange 3, base)
Vietnam - NCI	Yes
Russia - ARIPS	No (C.I. Solvent Violet 8.; C.I. Solvent Orange 3, base)

Continued...

Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 OTHER INFORMATION

Revision Date	22/07/2020
Initial Date	22/07/2020

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
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