



838AR (Aerosol) Total Ground Carbon Conductive Paint

MG Chemicals UK Limited

Version No: A-2.00

Safety data sheet according to REACH Regulation (EC) No 1907/2006, as amended by UK REACH Regulations SI 2019/758

Issue Date: 26/01/2022

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L.REACH.GB.EN

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

1.1. Product Identifier

Product name	838AR
Synonyms	SDS Code: 838AR-Aerosol; 838AR-340G UFI:SVJ0-506Q-Y001-04NU
Other means of identification	Total Ground Carbon Conductive Paint (Aerosol)

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

Relevant identified uses	Electrically conductive coating and EMI/RFI shield
Uses advised against	Not Applicable

1.3. Details of the supplier of the safety data sheet

Registered company name	MG Chemicals UK Limited	MG Chemicals (Head office)
Address	Heame House, 23 Bilston Street, Sedgely Dudley DY3 1JA United Kingdom	9347 - 193 Street Surrey V4N 4E7 British Columbia Canada
Telephone	+(44) 1663 362888	+(1) 800-201-8822
Fax	Not Available	+(1) 800-708-9888
Website	Not Available	www.mgchemicals.com
Email	sales@mgchemicals.com	Info@mgchemicals.com

1.4. Emergency telephone number

Association / Organisation	Verisk 3E (Access code: 335388)
Emergency telephone numbers	+(44) 20 35147487
Other emergency telephone numbers	+(0) 800 680 0425

SECTION 2 Hazards identification

2.1. Classification of the substance or mixture

Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567 [1]	H336 - Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, H223+H229 - Aerosols Category 2, H319 - Serious Eye Damage/Eye Irritation Category 2, H361 - Reproductive Toxicity Category 2, H317 - Sensitisation (Skin) Category 1, H351 - Carcinogenicity Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567

2.2. Label elements

Hazard pictogram(s)	  
Signal word	Warning

Hazard statement(s)

H336	May cause drowsiness or dizziness.
H223+H229	Flammable aerosol; Pressurized container: may burst if heated.
H319	Causes serious eye irritation.
H361	Suspected of damaging fertility or the unborn child.
H317	May cause an allergic skin reaction.
H351	Suspected of causing cancer.

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Supplementary statement(s)

EUH066	Repeated exposure may cause skin dryness or cracking.
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Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P211	Do not spray on an open flame or other ignition source.
P251	Do not pierce or burn, even after use.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing gas
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

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P302+P352	IF ON SKIN: Wash with plenty of water.
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.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.
P410+P412	Protect from sunlight. Do not expose to temperatures exceeding 50 °C/122 °F.
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.
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2.3. Other hazards

Inhalation may produce health damage*.

Cumulative effects may result following exposure*.

SECTION 3 Composition / information on ingredients

3.1.Substances

See 'Composition on ingredients' in Section 3.2

3.2.Mixtures

1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567	SCL / M-Factor	Nanoform Particle Characteristics
1.67-64-1 2.200-662-2 3.606-001-00-8 4.Not Available	21	<u>acetone</u> * -	Flammable Liquids Category 2, Serious Eye Damage/Eye Irritation Category 2, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3; H225, H319, H336 [2]	Not Available	Not Available
1.78-93-3 2.201-159-0 3.606-002-00-3 4.Not Available	20	<u>methyl ethyl ketone</u> * -	Flammable Liquids Category 2, Serious Eye Damage/Eye Irritation Category 2, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3; H225, H319, H336 [2]	Not Available	Not Available
1.108-10-1 2.203-550-1 3.606-004-00-4 4.Not Available	12	<u>methyl isobutyl ketone</u> * -	Flammable Liquids Category 2, Acute Toxicity (Inhalation) Category 4, Serious Eye Damage/Eye Irritation Category 2, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3; H225, H332, H319, H335 [2]	Not Available	Not Available
1.74-98-6 2.200-827-9 3.601-003-00-5 4.Not Available	12	<u>propane</u>	Flammable Gases Category 1, Gases Under Pressure; H220, H280 [2]	Not Available	Not Available
1.67-63-0 2.200-661-7 3.603-117-00-0 4.Not Available	7	<u>isopropanol</u>	Flammable Liquids Category 2, Serious Eye Damage/Eye Irritation Category 2, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3; H225, H319, H336 [2]	Not Available	Not Available

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1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567	SCL / M-Factor	Nanoform Particle Characteristics
1.75-28-5. 2.200-857-2 3.601-004-00-0 601-004-01-8 4.Not Available	6	<u>iso-butane</u>	Flammable Gases Category 1A, Gases Under Pressure (Liquefied Gas); H220, H280 [1]	Not Available	Not Available
1.123-86-4 2.204-658-1 3.607-025-00-1 4.Not Available	6	<u>n-butyl acetate</u> * -	Flammable Liquids Category 3, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3; H226, H336 [2]	Not Available	Not Available
1.141-78-6 2.205-500-4 3.607-022-00-5 4.Not Available	4	<u>ethyl acetate</u> * -	Flammable Liquids Category 2, Serious Eye Damage/Eye Irritation Category 2, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3; H225, H319, H336 [2]	Not Available	Not Available
1.1333-86-4 2.215-609-9 422-130-0 435-640-3 3.Not Available 4.Not Available	3	<u>carbon black</u>	Carcinogenicity Category 2; H351 [1]	Not Available	Not Available
1.108-65-6 2.203-603-9 3.607-195-00-7 4.Not Available	2	<u>propylene glycol monomethyl ether acetate, alpha-isomer</u> * -	Flammable Liquids Category 3; H226 [2]	Not Available	Not Available
1.25619-56-1 2.247-132-7 3.Not Available 4.Not Available	0.2	<u>barium dinonyl naphthalenesulfonate</u>	Acute Toxicity (Oral and Inhalation) Category 4, Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 2; H302+H332, H315, H318, H411 [1]	Not Available	Not Available
Legend:	1. Classified by Chemwatch; 2. Classification drawn from GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567; 3. Classification drawn from C&L; * EU IOELVs available; [e] Substance identified as having endocrine disrupting properties				

SECTION 4 First aid measures

4.1. Description of first aid measures

Eye Contact	<p>If aerosols come in contact with the eyes:</p> <ul style="list-style-type: none"> Immediately hold the eyelids apart and flush the eye continuously for at least 15 minutes 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. Transport to hospital or doctor without delay. Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>In case of cold burns (frost-bite):</p> <ul style="list-style-type: none"> Move casualty into warmth before thawing the affected part; if feet are affected carry if possible Bathe the affected area immediately in luke-warm water (not more than 35 deg C) for 10 to 15 minutes, immersing if possible and without rubbing DO NOT apply hot water or radiant heat. Apply a clean, dry, light dressing of 'fluffed-up' dry gauze bandage If a limb is involved, raise and support this to reduce swelling If an adult is involved and where intense pain occurs provide pain killers such as paracetamol Transport to hospital, or doctor Subsequent blackening of the exposed tissue indicates potential of necrosis, which may require amputation. <p>If solids or aerosol mists are deposited upon the skin:</p> <ul style="list-style-type: none"> Flush skin and hair with running water (and soap if available). Remove any adhering solids with industrial skin cleansing cream. DO NOT use solvents. Seek medical attention in the event of irritation.
Inhalation	<p>If aerosols, fumes or combustion products are inhaled:</p> <ul style="list-style-type: none"> Remove to fresh air. 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. If breathing is shallow or has stopped, ensure clear airway and apply resuscitation, 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	<ul style="list-style-type: none"> Not considered a normal route of entry. If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

4.2 Most important symptoms and effects, both acute and delayed

See Section 11

4.3. Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

To treat poisoning by the higher aliphatic alcohols (up to C7):

- Gastric lavage with copious amounts of water.
- It may be beneficial to instill 60 ml of mineral oil into the stomach.
- Oxygen and artificial respiration as needed.
- Electrolyte balance: it may be useful to start 500 ml. M/6 sodium bicarbonate intravenously but maintain a cautious and conservative attitude toward electrolyte replacement

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unless shock or severe acidosis threatens.

- ▶ To protect the liver, maintain carbohydrate intake by intravenous infusions of glucose.
- ▶ Haemodialysis if coma is deep and persistent. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products, Ed 5]

BASIC TREATMENT

- ▶ Establish a patent airway with suction where necessary.
- ▶ Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- ▶ Administer oxygen by non-rebreather mask at 10 to 15 l/min.
- ▶ Monitor and treat, where necessary, for shock.
- ▶ Monitor and treat, where necessary, for pulmonary oedema.
- ▶ Anticipate and treat, where necessary, for seizures.
- ▶ **DO NOT use emetics.** Where ingestion is suspected rinse mouth and give up to 200 ml water (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.
- ▶ Give activated charcoal.

ADVANCED TREATMENT

- ▶ Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- ▶ Positive-pressure ventilation using a bag-valve mask might be of use.
- ▶ Monitor and treat, where necessary, for arrhythmias.
- ▶ Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- ▶ If the patient is hypoglycaemic (decreased or loss of consciousness, tachycardia, pallor, dilated pupils, diaphoresis and/or dextrose strip or glucometer readings below 50 mg), give 50% dextrose.
- ▶ Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- ▶ Drug therapy should be considered for pulmonary oedema.
- ▶ Treat seizures with diazepam.
- ▶ Proparacaine hydrochloride should be used to assist eye irrigation.

EMERGENCY DEPARTMENT

- ▶ Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.
- ▶ Positive end-expiratory pressure (PEEP)-assisted ventilation may be required for acute parenchymal injury or adult respiratory distress syndrome.
- ▶ Acidosis may respond to hyperventilation and bicarbonate therapy.
- ▶ Haemodialysis might be considered in patients with severe intoxication.
- ▶ Consult a toxicologist as necessary. BRONSTEIN, A.C. and CURRANCE, P.L. EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

For C8 alcohols and above.

Symptomatic and supportive therapy is advised in managing patients.

for simple ketones:

BASIC TREATMENT

- ▶ Establish a patent airway with suction where necessary.
- ▶ Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- ▶ Administer oxygen by non-rebreather mask at 10 to 15 l/min.
- ▶ Monitor and treat, where necessary, for pulmonary oedema.
- ▶ Monitor and treat, where necessary, for shock.
- ▶ **DO NOT use emetics.** Where ingestion is suspected rinse mouth and give up to 200 ml water (5mL/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.
- ▶ Give activated charcoal.

ADVANCED TREATMENT

- ▶ Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- ▶ Consider intubation at first sign of upper airway obstruction resulting from oedema.
- ▶ Positive-pressure ventilation using a bag-valve mask might be of use.
- ▶ Monitor and treat, where necessary, for arrhythmias.
- ▶ Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- ▶ Drug therapy should be considered for pulmonary oedema.
- ▶ Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- ▶ Treat seizures with diazepam.
- ▶ Proparacaine hydrochloride should be used to assist eye irrigation.

EMERGENCY DEPARTMENT

- ▶ Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.
- ▶ Positive end-expiratory pressure (PEEP)-assisted ventilation may be required for acute parenchymal injury or adult respiratory distress syndrome.
- ▶ Consult a toxicologist as necessary.

BRONSTEIN, A.C. and CURRANCE, P.L.

EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

SECTION 5 Firefighting measures

5.1. Extinguishing media

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

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SMALL FIRE:

- ▶ Water spray, dry chemical or CO2

LARGE FIRE:

- ▶ Water spray or fog.

5.2. 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
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5.3. Advice for firefighters

Fire Fighting	<p>-----</p> <p>GENERAL</p> <p>-----</p> <ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear full body protective clothing with breathing apparatus. ▶ Fight fire from a safe distance, with adequate cover. ▶ If safe, switch off electrical equipment until vapour fire hazard removed. ▶ Use water delivered as a fine spray to control fire and cool adjacent area. ▶ DO NOT approach cylinders suspected to be hot. ▶ Cool fire exposed cylinders with water spray from a protected location. ▶ If safe to do so, remove cylinders from path of fire. ▶ Equipment should be thoroughly decontaminated after use. <p>-----</p> <p>FIRE FIGHTING PROCEDURES:</p> <p>-----</p> <ul style="list-style-type: none"> ▶ Excessive pressures may develop in a gas cylinder exposed in a fire; this may result in explosion. ▶ Cylinders with pressure relief devices may release their contents as a result of fire and the released gas may constitute a further source of hazard for the fire-fighter. ▶ Cylinders without pressure-relief valves have no provision for controlled release and are therefore more likely to explode if exposed to fire. <p>-----</p> <p>FIRE FIGHTING REQUIREMENTS:</p> <p>-----</p> <ul style="list-style-type: none"> ▶ Positive pressure, self-contained breathing apparatus is required for fire-fighting of hazardous materials. ▶ Full structural fire-fighting (bunker) gear is the minimum acceptable attire. ▶ The need for proximity, entry and special protective clothing should be determined for each incident, by a competent fire-fighting safety professional.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ Containers may explode when heated - Ruptured cylinders may rocket ▶ May burn but does not ignite easily. ▶ Fire exposed cylinders may vent contents through pressure relief devices thereby increasing vapour concentration.. ▶ Fire may produce irritating, poisonous or corrosive gases. ▶ Runoff may create fire or explosion hazard. ▶ May decompose explosively when heated or involved in fire. ▶ Contact with gas may cause burns, severe injury and/ or frostbite. ▶ POISONOUS: MAY BE FATAL IF INHALED, SWALLOWED OR ABSORBED THROUGH SKIN <p>Decomposition may produce toxic fumes of:</p> <p>carbon monoxide (CO)</p> <p>carbon dioxide (CO2)</p> <p>formaldehyde</p> <p>metal oxides</p> <p>other pyrolysis products typical of burning organic material.</p> <p>Contains low boiling substance: Closed containers may rupture due to pressure buildup under fire conditions.</p> <p>WARNING: Long standing in contact with air and light may result in the formation of potentially explosive peroxides.</p> <p>WARNING: Aerosol containers may present pressure related hazards.</p>

SECTION 6 Accidental release measures**6.1. Personal precautions, protective equipment and emergency procedures**

See section 8

6.2. Environmental precautions

See section 12

6.3. Methods and material for containment and cleaning up

Minor Spills	<ul style="list-style-type: none"> ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Wear protective clothing, impervious gloves and safety glasses. ▶ Shut off all possible sources of ignition and increase ventilation. ▶ Wipe up. ▶ If safe, damaged cans should be placed in a container outdoors, away from all ignition sources, until pressure has dissipated. ▶ Undamaged cans should be gathered and stowed safely.
Major Spills	<ul style="list-style-type: none"> ▶ Clear area of all unprotected personnel and move upwind. ▶ Alert Emergency Authority and advise them of the location and nature of hazard. ▶ Wear full body clothing with breathing apparatus. ▶ Prevent by any means available, spillage from entering drains and water-courses. ▶ Consider evacuation. ▶ Increase ventilation. ▶ No smoking or naked lights within area. ▶ Stop leak only if safe to do so. ▶ Water spray or fog may be used to disperse vapour.

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- ▶ **DO NOT** enter confined space where gas may have collected.
- ▶ Keep area clear until gas has dispersed.
- ▶ Remove leaking cylinders to a safe place.
- ▶ Fit vent pipes. Release pressure under safe, controlled conditions
- ▶ Burn issuing gas at vent pipes.
- ▶ **DO NOT** exert excessive pressure on valve; **DO NOT** attempt to operate damaged valve.
- ▶ Clear area of personnel and move upwind.
- ▶ Alert Fire Brigade and tell them location and nature of hazard.
- ▶ May be violently or explosively reactive.
- ▶ Wear breathing apparatus plus protective gloves.
- ▶ Prevent, by any means available, spillage from entering drains or water courses
- ▶ No smoking, naked lights or ignition sources.
- ▶ Increase ventilation.
- ▶ Stop leak if safe to do so.
- ▶ Water spray or fog may be used to disperse / absorb vapour.
- ▶ Absorb or cover spill with sand, earth, inert materials or vermiculite.
- ▶ If safe, damaged cans should be placed in a container outdoors, away from ignition sources, until pressure has dissipated.
- ▶ Undamaged cans should be gathered and stowed safely.
- ▶ Collect residues and seal in labelled drums for disposal.

6.4. Reference to other sections

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

SECTION 7 Handling and storage

7.1. Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ 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. ▶ DO NOT incinerate or puncture aerosol cans. ▶ DO NOT spray directly on humans, exposed food or food utensils. ▶ 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 are maintained.
Fire and explosion protection	See section 5
Other information	<ul style="list-style-type: none"> ▶ Cylinders should be stored in a purpose-built compound with good ventilation, preferably in the open. ▶ Such compounds should be sited and built in accordance with statutory requirements. ▶ The storage compound should be kept clear and access restricted to authorised personnel only. ▶ Cylinders stored in the open should be protected against rust and extremes of weather. ▶ Cylinders in storage should be properly secured to prevent toppling or rolling. ▶ Cylinder valves should be closed when not in use. ▶ Where cylinders are fitted with valve protection this should be in place and properly secured. ▶ Gas cylinders should be segregated according to the requirements of the Dangerous Goods Act. ▶ Preferably store full and empty cylinders separately. ▶ Check storage areas for hazardous concentrations of gases prior to entry. ▶ Full cylinders should be arranged so that the oldest stock is used first. ▶ Cylinders in storage should be checked periodically for general condition and leakage. ▶ Protect cylinders against physical damage. Move and store cylinders correctly as instructed for their manual handling. <p>NOTE: A 'G' size cylinder is usually too heavy for an inexperienced operator to raise or lower.</p>

7.2. Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Aerosol dispenser. ▶ Check that containers are clearly labelled.
Storage incompatibility	<p>n-Butyl acetate:</p> <ul style="list-style-type: none"> ▶ reacts with water on standing to form acetic acid and n-butyl alcohol ▶ reacts violently with strong oxidisers and potassium tert-butoxide ▶ is incompatible with caustics, strong acids and nitrates ▶ dissolves rubber, many plastics, resins and some coatings <p>Isopropanol (syn: isopropyl alcohol, IPA):</p> <ul style="list-style-type: none"> ▶ forms ketones and unstable peroxides on contact with air or oxygen; the presence of ketones especially methyl ethyl ketone (MEK, 2-butanone) will accelerate the rate of peroxidation ▶ reacts violently with strong oxidisers, powdered aluminium (exothermic), crotonaldehyde, diethyl aluminium bromide (ignition), dioxygenyl tetrafluoroborate (ignition/ ambient temperature), chromium trioxide (ignition), potassium-tert-butoxide (ignition), nitroform (possible explosion), oleum (pressure increased in closed container), cobalt chloride, aluminium triisopropoxide, hydrogen plus palladium dust (ignition), oxygen gas, phosgene, phosgene plus iron salts (possible explosion), sodium dichromate plus sulfuric acid (exothermic/ incandescence), triisobutyl aluminium ▶ reacts with phosphorus trichloride forming hydrogen chloride gas ▶ reacts, possibly violently, with alkaline earth and alkali metals, strong acids, strong caustics, acid anhydrides, halogens, aliphatic amines, aluminium isopropoxide, isocyanates, acetaldehyde, barium perchlorate (forms highly explosive perchloric ester compound), benzoyl peroxide, chromic acid, dialkylzincs, dichlorine oxide, ethylene oxide (possible explosion), hexamethylene diisocyanate (possible explosion), hydrogen peroxide (forms explosive compound), hypochlorous acid, isopropyl chlorocarbonate, lithium aluminium hydride, lithium tetrahydroaluminate, nitric acid, nitrogen dioxide, nitrogen tetraoxide (possible explosion), pentafluoroguanidine, perchloric acid (especially

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- hot), permonosulfuric acid, phosphorus pentasulfide, tangerine oil, triethylaluminium, triisobutylaluminium, trinitromethane
- ▶ attacks some plastics, rubber and coatings
 - ▶ reacts with metallic aluminium at high temperature
 - ▶ may generate electrostatic charges
- Methyl ethyl ketone:
- ▶ reacts violently with strong oxidisers, aldehydes, nitric acid, perchloric acid, potassium tert-butoxide, oleum
 - ▶ is incompatible with inorganic acids, aliphatic amines, ammonia, caustics, isocyanates, pyridines, chlorosulfonic acid
 - ▶ forms unstable peroxides in storage, or on contact with propanol or hydrogen peroxide
 - ▶ attacks some plastics
 - ▶ may generate electrostatic charges, due to low conductivity, on flow or agitation
- Methyl isobutyl ketone (MIBK)
- ▶ forms unstable and explosive peroxides on contact with air and/ or when in contact with hydrogen peroxide
 - ▶ reacts violently with strong oxidisers, aldehydes, aliphatic amines, nitric acid, perchloric acid, potassium tert-butoxide, strong acids, reducing agents
 - ▶ dissolves some plastics, resins and rubber
- Acetone:
- ▶ may react violently with chloroform, activated charcoal, aliphatic amines, bromine, bromine trifluoride, chlorotriazine, chromic(IV) acid, chromic(VI) acid, chromium trioxide, chromyl chloride, hexachloromelamine, iodine heptafluoride, iodoform, liquid oxygen, nitrosyl chloride, nitrosyl perchlorate, nitryl perchlorate, perchloromelamine, peroxomonosulfuric acid, platinum, potassium tert-butoxide, strong acids, sulfur dichloride, trichloromelamine, xenon tetrafluoride
 - ▶ reacts violently with bromoform and chloroform in the presence of alkalis or in contact with alkaline surfaces.
 - ▶ may form unstable and explosive peroxides in contact with strong oxidisers, fluorine, hydrogen peroxide (90%), sodium perchlorate, 2-methyl-1,3-butadiene
 - ▶ can increase the explosive sensitivity of nitromethane on contact flow or agitation may generate electrostatic charges due to low conductivity
 - ▶ dissolves or attacks most rubber, resins, and plastics (polyethylenes, polyester, vinyl ester, PVC, Neoprene, Viton)
- Alcohols
- ▶ are incompatible with strong acids, acid chlorides, acid anhydrides, oxidising and reducing agents.
 - ▶ reacts, possibly violently, with alkaline metals and alkaline earth metals to produce hydrogen
 - ▶ react with strong acids, strong caustics, aliphatic amines, isocyanates, acetaldehyde, benzoyl peroxide, chromic acid, chromium oxide, dialkylzincs, dichlorine oxide, ethylene oxide, hypochlorous acid, isopropyl chlorocarbonate, lithium tetrahydroaluminate, nitrogen dioxide, pentafluoroguanidine, phosphorus halides, phosphorus pentasulfide, tangerine oil, triethylaluminium, triisobutylaluminium
 - ▶ should not be heated above 49 deg. C. when in contact with aluminium equipment
- Butane/ isobutane
- ▶ reacts violently with strong oxidisers
 - ▶ reacts with acetylene, halogens and nitrous oxides
 - ▶ is incompatible with chlorine dioxide, conc. nitric acid and some plastics
 - ▶ may generate electrostatic charges, due to low conductivity, in flow or when agitated - these may ignite the vapour.
- Segregate from nickel carbonyl in the presence of oxygen, heat (20-40 C)
- ▶ Esters react with acids to liberate heat along with alcohols and acids.
 - ▶ Strong oxidising acids may cause a vigorous reaction with esters that is sufficiently exothermic to ignite the reaction products.
 - ▶ Heat is also generated by the interaction of esters with caustic solutions.
 - ▶ Flammable hydrogen is generated by mixing esters with alkali metals and hydrides.
 - ▶ Esters may be incompatible with aliphatic amines and nitrates.
- Ketones in this group:
- ▶ are reactive with many acids and bases liberating heat and flammable gases (e.g., H₂).
 - ▶ react with reducing agents such as hydrides, alkali metals, and nitrides to produce flammable gas (H₂) and heat.
 - ▶ are incompatible with isocyanates, aldehydes, cyanides, peroxides, and anhydrides.
 - ▶ react violently with aldehydes, HNO₃ (nitric acid), HNO₃ + H₂O₂ (mixture of nitric acid and hydrogen peroxide), and HClO₄ (perchloric acid).
 - ▶ may react with hydrogen peroxide to form unstable peroxides; many are heat- and shock-sensitive explosives.
- A significant property of most ketones is that the hydrogen atoms on the carbons next to the carbonyl group are relatively acidic when compared to hydrogen atoms in typical hydrocarbons. Under strongly basic conditions these hydrogen atoms may be abstracted to form an enolate anion. This property allows ketones, especially methyl ketones, to participate in condensation reactions with other ketones and aldehydes. This type of condensation reaction is favoured by high substrate concentrations and high pH (greater than 1 wt% NaOH).
- Propane:
- ▶ reacts violently with strong oxidisers, barium peroxide, chlorine dioxide, dichlorine oxide, fluorine etc.
 - ▶ liquid attacks some plastics, rubber and coatings
 - ▶ may accumulate static charges which may ignite its vapours
 - ▶ Compressed gases may contain a large amount of kinetic energy over and above that potentially available from the energy of reaction produced by the gas in chemical reaction with other substances

7.3. Specific end use(s)

See section 1.2

SECTION 8 Exposure controls / personal protection

8.1. Control parameters

Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
acetone	Dermal 186 mg/kg bw/day (Systemic, Chronic) Inhalation 1 210 mg/m ³ (Systemic, Chronic) Inhalation 2 420 mg/m ³ (Local, Acute) <i>Dermal 62 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 200 mg/m³ (Systemic, Chronic) *</i> <i>Oral 62 mg/kg bw/day (Systemic, Chronic) *</i>	10.6 mg/L (Water (Fresh)) 1.06 mg/L (Water - Intermittent release) 21 mg/L (Water (Marine)) 30.4 mg/kg sediment dw (Sediment (Fresh Water)) 3.04 mg/kg sediment dw (Sediment (Marine)) 29.5 mg/kg soil dw (Soil) 100 mg/L (STP)
methyl ethyl ketone	Dermal 1 161 mg/kg bw/day (Systemic, Chronic) Inhalation 600 mg/m ³ (Systemic, Chronic) <i>Dermal 412 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 106 mg/m³ (Systemic, Chronic) *</i> <i>Oral 31 mg/kg bw/day (Systemic, Chronic) *</i>	55.8 mg/L (Water (Fresh)) 55.8 mg/L (Water - Intermittent release) 55.8 mg/L (Water (Marine)) 284.74 mg/kg sediment dw (Sediment (Fresh Water)) 284.7 mg/kg sediment dw (Sediment (Marine)) 22.5 mg/kg soil dw (Soil) 709 mg/L (STP) 1000 mg/kg food (Oral)

Continued...

838AR (Aerosol) Total Ground Carbon Conductive Paint

Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
methyl isobutyl ketone	Dermal 11.8 mg/kg bw/day (Systemic, Chronic) Inhalation 83 mg/m ³ (Systemic, Chronic) Inhalation 83 mg/m ³ (Local, Chronic) Inhalation 208 mg/m ³ (Systemic, Acute) Inhalation 208 mg/m ³ (Local, Acute) <i>Dermal 4.2 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 14.7 mg/m³ (Systemic, Chronic) *</i> <i>Oral 4.2 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 14.7 mg/m³ (Local, Chronic) *</i> <i>Inhalation 155.2 mg/m³ (Systemic, Acute) *</i> <i>Inhalation 155.2 mg/m³ (Local, Acute) *</i>	0.6 mg/L (Water (Fresh)) 0.06 mg/L (Water - Intermittent release) 1.5 mg/L (Water (Marine)) 8.27 mg/kg sediment dw (Sediment (Fresh Water)) 0.83 mg/kg sediment dw (Sediment (Marine)) 1.3 mg/kg soil dw (Soil) 27.5 mg/L (STP)
isopropanol	Dermal 888 mg/kg bw/day (Systemic, Chronic) Inhalation 500 mg/m ³ (Systemic, Chronic) <i>Dermal 319 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 89 mg/m³ (Systemic, Chronic) *</i> <i>Oral 26 mg/kg bw/day (Systemic, Chronic) *</i>	140.9 mg/L (Water (Fresh)) 140.9 mg/L (Water - Intermittent release) 140.9 mg/L (Water (Marine)) 552 mg/kg sediment dw (Sediment (Fresh Water)) 552 mg/kg sediment dw (Sediment (Marine)) 28 mg/kg soil dw (Soil) 2251 mg/L (STP) 160 mg/kg food (Oral)
n-butyl acetate	Dermal 7 mg/kg bw/day (Systemic, Chronic) Inhalation 48 mg/m ³ (Systemic, Chronic) Inhalation 300 mg/m ³ (Local, Chronic) Dermal 11 mg/kg bw/day (Systemic, Acute) Inhalation 600 mg/m ³ (Systemic, Acute) Inhalation 600 mg/m ³ (Local, Acute) <i>Dermal 3.4 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 12 mg/m³ (Systemic, Chronic) *</i> <i>Oral 2 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 35.7 mg/m³ (Local, Chronic) *</i> <i>Dermal 6 mg/kg bw/day (Systemic, Acute) *</i> <i>Inhalation 300 mg/m³ (Systemic, Acute) *</i> <i>Oral 2 mg/kg bw/day (Systemic, Acute) *</i> <i>Inhalation 300 mg/m³ (Local, Acute) *</i>	0.18 mg/L (Water (Fresh)) 0.018 mg/L (Water - Intermittent release) 0.36 mg/L (Water (Marine)) 0.981 mg/kg sediment dw (Sediment (Fresh Water)) 0.098 mg/kg sediment dw (Sediment (Marine)) 0.09 mg/kg soil dw (Soil) 35.6 mg/L (STP)
ethyl acetate	Dermal 63 mg/kg bw/day (Systemic, Chronic) Inhalation 734 mg/m ³ (Systemic, Chronic) Inhalation 734 mg/m ³ (Local, Chronic) Inhalation 1 468 mg/m ³ (Systemic, Acute) Inhalation 1 468 mg/m ³ (Local, Acute) <i>Dermal 37 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 367 mg/m³ (Systemic, Chronic) *</i> <i>Oral 4.5 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 367 mg/m³ (Local, Chronic) *</i> <i>Inhalation 734 mg/m³ (Systemic, Acute) *</i> <i>Inhalation 734 mg/m³ (Local, Acute) *</i>	0.24 mg/L (Water (Fresh)) 0.024 mg/L (Water - Intermittent release) 1.65 mg/L (Water (Marine)) 1.15 mg/kg sediment dw (Sediment (Fresh Water)) 0.115 mg/kg sediment dw (Sediment (Marine)) 0.148 mg/kg soil dw (Soil) 650 mg/L (STP) 0.2 g/kg food (Oral)
carbon black	Inhalation 1 mg/m ³ (Systemic, Chronic) Inhalation 0.5 mg/m ³ (Local, Chronic) <i>Inhalation 0.06 mg/m³ (Systemic, Chronic) *</i>	1 mg/L (Water (Fresh)) 0.1 mg/L (Water - Intermittent release) 10 mg/L (Water (Marine))
propylene glycol monomethyl ether acetate, alpha-isomer	Dermal 796 mg/kg bw/day (Systemic, Chronic) Inhalation 275 mg/m ³ (Systemic, Chronic) Inhalation 550 mg/m ³ (Local, Acute) <i>Dermal 320 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 33 mg/m³ (Systemic, Chronic) *</i> <i>Oral 36 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 33 mg/m³ (Local, Chronic) *</i>	0.635 mg/L (Water (Fresh)) 0.064 mg/L (Water - Intermittent release) 6.35 mg/L (Water (Marine)) 3.29 mg/kg sediment dw (Sediment (Fresh Water)) 0.329 mg/kg sediment dw (Sediment (Marine)) 0.29 mg/kg soil dw (Soil) 100 mg/L (STP)

* Values for General Population

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	acetone	Acetone	500 ppm / 1210 mg/m ³	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	acetone	Acetone	500 ppm / 1210 mg/m ³	3620 mg/m ³ / 1500 ppm	Not Available	Not Available
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	methyl ethyl ketone	Butanone	200 ppm / 600 mg/m ³	900 mg/m ³ / 300 ppm	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	methyl ethyl ketone	Butan-2-one (methyl ethyl ketone)	200 ppm / 600 mg/m ³	899 mg/m ³ / 300 ppm	Not Available	Sk, BMGV
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	methyl isobutyl ketone	4-Methylpentan-2-one	20 ppm / 83 mg/m ³	208 mg/m ³ / 50 ppm	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	methyl isobutyl ketone	4-Methylpentan-2-one	50 ppm / 208 mg/m ³	416 mg/m ³ / 100 ppm	Not Available	Sk, BMGV
UK Workplace Exposure Limits (WELs)	isopropanol	Propan-2-ol	400 ppm / 999 mg/m ³	1250 mg/m ³ / 500 ppm	Not Available	Not Available

Continued...

838AR (Aerosol) Total Ground Carbon Conductive Paint

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	n-butyl acetate	n-Butyl acetate	50 ppm / 241 mg/m ³	723 mg/m ³ / 150 ppm	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	n-butyl acetate	Butyl acetate	150 ppm / 724 mg/m ³	966 mg/m ³ / 200 ppm	Not Available	Not Available
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	ethyl acetate	Ethyl acetate	200 ppm / 734 mg/m ³	1 468 mg/m ³ / 400 ppm	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	ethyl acetate	Ethyl acetate	200 ppm / 734 mg/m ³	1468 mg/m ³ / 400 ppm	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	carbon black	Carbon black	3.5 mg/m ³	7 mg/m ³	Not Available	Not Available
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	propylene glycol monomethyl ether acetate, alpha-isomer	1-Methoxypropyl-2-acetate	50 ppm / 275 mg/m ³	550 mg/m ³ / 100 ppm	Not Available	Skin
UK Workplace Exposure Limits (WELs)	propylene glycol monomethyl ether acetate, alpha-isomer	1-Methoxypropyl acetate	50 ppm / 274 mg/m ³	548 mg/m ³ / 100 ppm	Not Available	Sk

Emergency Limits			
Ingredient	TEEL-1	TEEL-2	TEEL-3
acetone	Not Available	Not Available	Not Available
methyl ethyl ketone	Not Available	Not Available	Not Available
methyl isobutyl ketone	75 ppm	500 ppm	3000* ppm
propane	Not Available	Not Available	Not Available
isopropanol	400 ppm	2000* ppm	12000** ppm
iso-butane	5500* ppm	17000** ppm	53000*** ppm
n-butyl acetate	Not Available	Not Available	Not Available
ethyl acetate	1,200 ppm	1,700 ppm	10000** ppm
carbon black	9 mg/m ³	99 mg/m ³	590 mg/m ³
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
acetone	2,500 ppm	Not Available
methyl ethyl ketone	3,000 ppm	Not Available
methyl isobutyl ketone	500 ppm	Not Available
propane	2,100 ppm	Not Available
isopropanol	2,000 ppm	Not Available
iso-butane	Not Available	Not Available
n-butyl acetate	1,700 ppm	Not Available
ethyl acetate	2,000 ppm	Not Available
carbon black	1,750 mg/m ³	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available	Not Available
barium dinonyl naphthalenesulfonate	Not Available	Not Available

Occupational Exposure Banding		
Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
barium dinonyl naphthalenesulfonate	E	≤ 0.01 mg/m ³
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

For ethyl acetate:

Odour Threshold Value: 6.4-50 ppm (detection), 13.3-75 ppm (recognition)

The TLV-TWA provides a significant margin of safety from the standpoint of adverse health effects. Unacclimated subjects found the odour objectionably strong at 200 ppm. Mild nose, eye and throat irritation was experienced at 400 ppm. Workers exposed regularly at concentrations ranging from 375 ppm to 1500 ppm for several months showed no unusual signs or symptoms.

Odour Safety Factor(OSF)

OSF=51 (ETHYL ACETATE)

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

Continued...

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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:

ClassOSF 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

Odour Threshold Value: 3.6 ppm (detection), 699 ppm (recognition)

Saturation vapour concentration: 237000 ppm @ 20 C

NOTE: Detector tubes measuring in excess of 40 ppm, are available.

Exposure at or below the recommended TLV-TWA is thought to protect the worker against mild irritation associated with brief exposures and the bioaccumulation, chronic irritation of the respiratory tract and headaches associated with long-term acetone exposures. The NIOSH REL-TWA is substantially lower and has taken into account slight irritation experienced by volunteer subjects at 300 ppm. Mild irritation to acclimatised workers begins at about 750 ppm - unacclimatised subjects will experience irritation at about 350-500 ppm but acclimatisation can occur rapidly. Disagreement between the peak bodies is based largely on the view by ACGIH that widespread use of acetone, without evidence of significant adverse health effects at higher concentrations, allows acceptance of a higher limit.

Half-life of acetone in blood is 3 hours which means that no adjustment for shift-length has to be made with reference to the standard 8 hour/day, 40 hours per week because body clearance occurs within any shift with low potential for accumulation.

A STEL has been established to prevent excursions of acetone vapours that could cause depression of the central nervous system.

Odour Safety Factor(OSF)

OSF=38 (ACETONE)

For n-butyl acetate

Odour Threshold Value: 0.0063 ppm (detection), 0.038-12 ppm (recognition)

Exposure at or below the recommended TLV-TWA is thought to prevent significant irritation of the eyes and respiratory passages as well as narcotic effects. In light of the lack of substantive evidence regarding teratogenicity and a review of acute oral data a STEL is considered inappropriate.

Odour Safety Factor(OSF)

OSF=3.8E2 (n-BUTYL ACETATE)

For butane:

Odour Threshold Value: 2591 ppm (recognition)

Butane in common with other homologues in the straight chain saturated aliphatic hydrocarbon series is not characterised by its toxicity but by its narcosis-inducing effects at high concentrations. The TLV is based on analogy with pentane by comparing their lower explosive limits in air. It is concluded that this limit will protect workers against the significant risk of drowsiness and other narcotic effects.

Odour Safety Factor(OSF)

OSF=0.22 (n-BUTANE)

for propylene glycol monomethyl ether acetate (PGMEA)

Saturated vapour concentration: 4868 ppm at 20 C.

A two-week inhalation study found nasal effects to the nasal mucosa in animals at concentrations up to 3000 ppm. Differences in the teratogenic potential of the alpha (commercial grade) and beta isomers of PGMEA may be explained by the formation of different metabolites. The beta-isomer is thought to be oxidised to methoxypropionic acid, a homologue to methoxyacetic acid which is a known teratogen. The alpha- form is conjugated and excreted. PGMEA mixture (containing 2% to 5% beta isomer) is a mild skin and eye irritant, produces mild central nervous system effects in animals at 3000 ppm and produces mild CNS impairment and upper respiratory tract and eye irritation in humans at 1000 ppm. In rats exposed to 3000 ppm PGMEA produced slight foetotoxic effects (delayed sternal ossification) - no effects on foetal development were seen in rabbits exposed at 3000 ppm.

For methyl ethyl ketone:

Odour Threshold Value: Various reported as 2 ppm and 4.8 ppm

Odour threshold: 2 ppm (detection); 5 ppm (recognition); 25 ppm (easy recognition); 300 ppm IRRITATING

Exposures at or below the recommended TLV-TWA are thought to prevent injurious systemic effects and to minimise objections to odour and irritation. Where synergism or potentiation may occur stringent control of the primary toxin (e.g. n-hexane or methyl butyl ketone) is desirable and additional consideration should be given to lowering MEK exposures.

Odour Safety Factor(OSF)

OSF=28 (METHYL ETHYL KETONE)

for methyl isobutyl ketone (MIBK):

Unfatigued, odour recognition threshold (100% test panel) is 0.3 - 0.5 ppm.

Distinct odour at 15 ppm.

Odour is objectionable and vapours are irritating to eyes at 200 ppm.

NOTE: Detector tubes for methyl isobutyl ketone, measuring in excess of 50 ppm, are commercially available.

Exposure at or below the recommended TLV-TWA should provide sufficient protection against the potential irritant effects, headache and nausea, neurasthenic symptoms and other systemic toxicities (including liver and kidney damage) produced by MIBK.

The low odour threshold (1.64 mg/m3) and the irritant effects can provide warning of high concentrations. Exposure to levels of 10-410 mg/m3 (2.4-100 ppm) produced perceptible irritation of the eyes, nose, or throat, and 820 mg/m3 (200 ppm) produced discomfort. Symptoms, such as headache, nausea, or vertigo, also occurred at 10-410 mg/m3 (2.4-100 ppm). A 2-h exposure of up to 200 mg/m3 (50 ppm) did not produce any significant effects on a simple reaction-time task or a test of mental arithmetic.

Odour Safety Factor(OSF)

OSF=29 (METHYL ISOBUTYL KETONE)

For propane

Odour Safety Factor(OSF)

OSF=0.16 (PROPANE)

Odour Threshold Value: 3.3 ppm (detection), 7.6 ppm (recognition)

Exposure at or below the recommended isopropanol TLV-TWA and STEL is thought to minimise the potential for inducing narcotic effects or significant irritation of the eyes or upper respiratory tract. It is believed, in the absence of hard evidence, that this limit also provides protection against the development of chronic health effects. The limit is intermediate to that set for ethanol, which is less toxic, and n-propyl alcohol, which is more toxic, than isopropanol

8.2. Exposure controls

8.2.1. Appropriate engineering

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can

Continued...

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	<p>be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>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.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>General exhaust is adequate under normal conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection.</p> <p>Provide adequate ventilation in warehouse or closed storage areas.</p> <p>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.</p> <table border="1" data-bbox="389 499 1439 600"> <tr> <td>Type of Contaminant:</td><td>Speed:</td></tr> <tr> <td>aerosols, (released at low velocity into zone of active generation)</td><td>0.5-1 m/s</td></tr> <tr> <td>direct spray, spray painting in shallow booths, gas discharge (active generation into zone of rapid air motion)</td><td>1-2.5 m/s (200-500 f/min.)</td></tr> </table> <p>Within each range the appropriate value depends on:</p> <table border="1" data-bbox="389 656 1091 824"> <tr> <td>Lower end of the range</td><td>Upper end of the range</td></tr> <tr> <td>1: Room air currents minimal or favourable to capture</td><td>1: Disturbing room air currents</td></tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td><td>2: Contaminants of high toxicity</td></tr> <tr> <td>3: Intermittent, low production.</td><td>3: High production, heavy use</td></tr> <tr> <td>4: Large hood or large air mass in motion</td><td>4: Small hood-local control only</td></tr> </table> <p>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.</p>	Type of Contaminant:	Speed:	aerosols, (released at low velocity into zone of active generation)	0.5-1 m/s	direct spray, spray painting in shallow booths, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)	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
Type of Contaminant:	Speed:																
aerosols, (released at low velocity into zone of active generation)	0.5-1 m/s																
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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																
8.2.2. Personal protection																	
Eye and face protection	<ul style="list-style-type: none"> ▶ Chemical goggles. ▶ Full face shield may be required for supplementary but never for primary protection of eyes. ▶ 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	<p>NOTE:</p> <ul style="list-style-type: none"> ▶ 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. <p>For esters:</p> <ul style="list-style-type: none"> ▶ Do NOT use natural rubber, butyl rubber, EPDM or polystyrene-containing materials. ▶ No special equipment needed when handling small quantities. <p>OTHERWISE:</p> <ul style="list-style-type: none"> ▶ For potentially moderate exposures: ▶ Wear general protective gloves, eg. light weight rubber gloves. ▶ For potentially heavy exposures: ▶ Wear chemical protective gloves, eg. PVC. and safety footwear. ▶ Insulated gloves: <p>NOTE: Insulated gloves should be loose fitting so that may be removed quickly if liquid is spilled upon them. Insulated gloves are not made to permit hands to be placed in the liquid; they provide only short-term protection from accidental contact with the liquid.</p>																
Body protection	See Other protection below																
Other protection	<p>No special equipment needed when handling small quantities.</p> <p>OTHERWISE:</p> <ul style="list-style-type: none"> ▶ Overalls. ▶ Skin cleansing cream. ▶ Eyewash unit. ▶ Do not spray on hot surfaces. 																

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:

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Respiratory protection

Type AX 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.

Continued...

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Material	CPI
PE/EVAL/PE	A
BUTYL	C
BUTYL/NEOPRENE	C
CPE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE	C
PVA	C
PVC	C
PVDC/PE/PVDC	C
SARANEX-23	C
SARANEX-23 2-PLY	C
TEFLON	C
VITON/BUTYL	C
VITON/CHLOROBUTYL	C
VITON/NEOPRENE	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.

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

^ - 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

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

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

** - Continuous-flow or positive pressure demand.

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

8.2.3. Environmental exposure controls

See section 12

SECTION 9 Physical and chemical properties

9.1. Information on basic physical and chemical properties

Appearance	Black		
Physical state	Liquified Gas	Relative density (Water = 1)	0.83
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	465
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)	56	Molecular weight (g/mol)	Not Available
Flash point (°C)	-17	Taste	Not Available
Evaporation rate	<1 BuAC = 1	Explosive properties	Not Available
Flammability	HIGHLY FLAMMABLE.	Oxidising properties	Not Available
Upper Explosive Limit (%)	9.4	Surface Tension (dyn/cm or mN/m)	Not Available

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Lower Explosive Limit (%)	2	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Partly miscible	pH as a solution (Not Available%)	Not Available
Vapour density (Air = 1)	>1	VOC g/L	Not Available
Nanoform Solubility	Not Available	Nanoform Particle Characteristics	Not Available
Particle Size	Not Available		

9.2. Other information

Not Available

SECTION 10 Stability and reactivity

10.1. Reactivity	See section 7.2
10.2. Chemical stability	<ul style="list-style-type: none"> ▸ Elevated temperatures. ▸ Presence of open flame. ▸ Product is considered stable. ▸ Hazardous polymerisation will not occur.
10.3. Possibility of hazardous reactions	See section 7.2
10.4. Conditions to avoid	See section 7.2
10.5. Incompatible materials	See section 7.2
10.6. Hazardous decomposition products	See section 5.3

SECTION 11 Toxicological information

11.1. Information on toxicological effects

Inhaled	<p>Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful. The material is not thought to produce respiratory irritation (as classified by EC Directives using animal models). Nevertheless inhalation, of the material, especially for prolonged periods, may produce respiratory discomfort and occasionally, distress.</p> <p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>The main effects of simple aliphatic esters are narcosis and irritation and anaesthesia at higher concentrations. These effects become greater as the molecular weights and boiling points increase. Central nervous system depression, headache, drowsiness, dizziness, coma and neurobehavioral changes may also be symptomatic of overexposure. Respiratory tract involvement may produce mucous membrane irritation, dyspnea, and tachypnea, pharyngitis, bronchitis, pneumonitis and, in massive exposures, pulmonary oedema (which may be delayed). Gastrointestinal effects include nausea, vomiting, diarrhoea and abdominal cramps. Liver and kidney damage may result from massive exposures.</p> <p>No health effects were seen in humans exposed at 1,000 ppm isobutane for up to 8 hours or 500 ppm for 8 hours/day for 10 days. Isobutane can have anaesthetic and asphyxiant effects at high concentrations, well above the lower explosion limit of 1.8% (18,000 ppm). Butane is a simple asphyxiant and is mildly anaesthetic at high concentrations (20-25%). 10000 ppm for 10 minutes causes drowsiness. Narcotic effects may be accompanied by exhilaration, dizziness, headache, nausea, confusion, incoordination and unconsciousness in severe cases.</p> <p>The paraffin gases C1-4 are practically nontoxic below the lower flammability limit, 18,000 to 50,000 ppm; above this, low to moderate incidental effects such as CNS depression and irritation occur, but are completely reversible upon cessation of the exposure.</p> <p>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.</p> <p>Common, generalised symptoms associated with toxic gas inhalation include:</p> <ul style="list-style-type: none"> ▸ central nervous system effects such as depression, headache, confusion, dizziness, progressive stupor, coma and seizures; ▸ respiratory system complications may include acute pulmonary oedema, dyspnoea, stridor, tachypnoea, bronchospasm, wheezing and other reactive airway symptoms, and respiratory arrest; ▸ cardiovascular effects may include cardiovascular collapse, arrhythmias and cardiac arrest; ▸ gastrointestinal effects may also be present and may include mucous membrane irritation, nausea and vomiting (sometimes bloody), and abdominal pain. <p>Material is highly volatile and may quickly form a concentrated atmosphere in confined or unventilated areas. The vapour may displace and replace air in breathing zone, acting as a simple asphyxiant. This may happen with little warning of overexposure.</p> <p>The use of a quantity of material in an unventilated or confined space may result in increased exposure and an irritating atmosphere developing. Before starting consider control of exposure by mechanical ventilation.</p> <p>WARNING: Intentional misuse by concentrating/inhaling contents may be lethal.</p>
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	<p>Acute exposure of humans to high concentrations of methyl ethyl ketone produces irritation to the eyes, nose, and throat. Other effects reported from acute inhalation exposure in humans include central nervous system depression, headache, and nausea.</p> <p>Easy odour recognition and irritant properties of methyl ethyl ketone means that high vapour levels are readily detected and should be avoided by application of control measures; however odour fatigue may occur with loss of warning of exposure.</p> <p>Human overexposure to MIBK vapour may produce weakness, loss of appetite, headache, a burning sensation to the eyes, stomach-ache, nausea and vomiting. Sore throat, insomnia, somnolence, heartburn and intestinal pain have been reported by some workers. Tolerance is reported to be acquired over the workweek and lost during the weekend.</p> <p>Exposure to high concentrations (>1000 ppm) can produce central nervous system depression and narcosis. Lower doses (80-500 ppm) can cause weakness, headache and nausea.</p> <p>Rats, mice, dogs and monkeys that inhaled 100 or 200 ppm MIBK 24 hrs/day showed no outward adverse effects during 2 weeks of exposure. At 200 ppm rats showed increased absolute liver and kidney weights and increased organ-to-body weight ratios. Examination of the proximal tubules showed toxic nephrosis (hyaline droplet degeneration and occasional focal tubular necrosis) in rats exposed to 100 ppm. This damage was considered transient and reversible. Discriminatory behaviour and memory in baboons was effected at exposures of 50 ppm for 7 days.</p> <p>The odour of isopropanol may give some warning of exposure, but odour fatigue may occur. Inhalation of isopropanol may produce irritation of the nose and throat with sneezing, sore throat and runny nose. The effects in animals subject to a single exposure, by inhalation, included inactivity or anaesthesia and histopathological changes in the nasal canal and auditory canal.</p>
Ingestion	<p>Effects on the nervous system characterise over-exposure to higher aliphatic alcohols. These include headache, muscle weakness, giddiness, ataxia, (loss of muscle coordination), confusion, delirium and coma. Gastrointestinal effects may include nausea, vomiting and diarrhoea. In the absence of effective treatment, respiratory arrest is the most common cause of death in animals acutely poisoned by the higher alcohols.</p> <p>Aspiration of liquid alcohols produces an especially toxic response as they are able to penetrate deeply in the lung where they are absorbed and may produce pulmonary injury. Those possessing lower viscosity elicit a greater response. The result is a high blood level and prompt death at doses otherwise tolerated by ingestion without aspiration. In general the secondary alcohols are less toxic than the corresponding primary isomers. As a general observation, alcohols are more powerful central nervous system depressants than their aliphatic analogues. In sequence of decreasing depressant potential, tertiary alcohols with multiple substituent OH groups are more potent than secondary alcohols, which, in turn, are more potent than primary alcohols. The potential for overall systemic toxicity increases with molecular weight (up to C7), principally because the water solubility is diminished and lipophilicity is increased.</p> <p>Within the homologous series of aliphatic alcohols, narcotic potency may increase even faster than lethality</p> <p>Only scanty toxicity information is available about higher homologues of the aliphatic alcohol series (greater than C7) but animal data establish that lethality does not continue to increase with increasing chain length. Aliphatic alcohols with 8 carbons are less toxic than those immediately preceding them in the series. 10 -Carbon n-decyl alcohol has low toxicity as do the solid fatty alcohols (e.g. lauryl, myristyl, cetyl and stearyl).</p> <p>However the rat aspiration test suggests that decyl and melted dodecyl (lauryl) alcohols are dangerous if they enter the trachea. In the rat even a small quantity (0.2 ml) of these behaves like a hydrocarbon solvent in causing death from pulmonary oedema.</p> <p>Primary alcohols are metabolised to corresponding aldehydes and acids; a significant metabolic acidosis may occur. Secondary alcohols are converted to ketones, which are also central nervous system depressants and which, in the case of the higher homologues persist in the blood for many hours. Tertiary alcohols are metabolised slowly and incompletely so their toxic effects are generally persistent.</p> <p>Not normally a hazard due to physical form of product.</p> <p>Considered an unlikely route of entry in commercial/industrial environments</p> <p>Many aliphatic hydrocarbons create a burning sensation because they are irritating to the GI mucosa. Vomiting has been reported in up to one third of all hydrocarbon exposures. While most aliphatic hydrocarbons have little GI absorption, aspiration frequently occurs, either initially or in a semi-delayed fashion as the patient coughs or vomits, thereby resulting in pulmonary effects. Once aspirated, the hydrocarbons can create a severe pneumonitis.</p> <p>Rats given isoparaffinic hydrocarbons - isoalkanes- (after 18-24 hours fasting) showed lethargy and/or general weakness, ataxia and diarrhoea. Symptoms disappeared within 24-28 hours.</p> <p>Following ingestion, a single exposure to isopropyl alcohol produced lethargy and non-specific effects such as weight loss and irritation. Ingestion of near-lethal doses of isopropanol produces histopathological changes of the stomach, lungs and kidneys, incoordination, lethargy, gastrointestinal tract irritation, and inactivity or anaesthesia.</p> <p>Swallowing 10 ml. of isopropanol may cause serious injury; 100 ml. may be fatal if not promptly treated. The adult single lethal doses is approximately 250 ml. The toxicity of isopropanol is twice that of ethanol and the symptoms of intoxication appear to be similar except for the absence of an initial euphoric effect; gastritis and vomiting are more prominent. Ingestion may cause nausea, vomiting, and diarrhoea.</p> <p>There is evidence that a slight tolerance to isopropanol may be acquired.</p>
Skin Contact	<p>The material may accentuate any pre-existing dermatitis condition</p> <p>Dermatitis has been reported in humans following dermal exposure to methyl ethyl ketone. Tests involving acute exposure of rabbits has shown methyl ethyl ketone to have high acute toxicity from dermal exposure.</p> <p>Spray mist may produce discomfort</p> <p>Most liquid alcohols appear to act as primary skin irritants in humans. Significant percutaneous absorption occurs in rabbits but not apparently in man.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>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.</p> <p>Vapourising liquid causes rapid cooling and contact may cause cold burns, frostbite, even through normal gloves. Frozen skin tissues are painless and appear waxy and yellow. Signs and symptoms of frost-bite may include 'pins and needles', paleness followed by numbness, a hardening and stiffening of the skin, a progression of colour changes in the affected area, (first white, then mottled and blue and eventually black; on recovery, red, hot, painful and blistered).</p> <p>511ipa</p> <p>The material may produce mild skin irritation; limited evidence or practical experience suggests, that the material either:</p> <ul style="list-style-type: none"> ▸ produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or ▸ produces significant, but mild, 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. <p>Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (non allergic). 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.</p>
Eye	<p>Direct contact with the eye may not cause irritation because of the extreme volatility of the gas; however concentrated atmospheres may produce irritation after brief exposures..</p> <p>At concentrations of 100-200 ppm MIBK, the vapour may irritate the eyes and respiratory tract</p> <p>Isopropanol vapour may cause mild eye irritation at 400 ppm. Splashes may cause severe eye irritation, possible corneal burns and eye damage.</p> <p>Eye contact may cause tearing or blurring of vision.</p> <p>The liquid may produce eye discomfort and is capable of causing temporary impairment of vision and/or transient eye inflammation, ulceration</p> <p>Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to</p>

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	windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.	
Chronic	<p>On the basis, primarily, of animal experiments, concern has been expressed 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. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.</p> <p>Substances that can cause occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers. Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.</p> <p>Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.</p> <p>Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.</p> <p>Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.</p> <p>Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects.</p> <p>Principal route of occupational exposure to the gas is by inhalation.</p> <p>Limited information is available on the chronic (long-term) effects of methyl ethyl ketone in humans. Chronic inhalation studies in animals have reported slight neurological, liver, kidney, and respiratory effects. No information is available on the developmental, reproductive, or carcinogenic effects of methyl ethyl ketone in humans. Developmental effects, including decreased foetal weight and foetal malformations, have been reported in mice and rats exposed to methyl ethyl ketone via inhalation and ingestion.</p> <p>Methyl ethyl ketone is considered to have a low order of toxicity; however methyl ethyl ketone is often used in combination with other solvents and the toxic effects of the mix may be greater than either solvent alone. Combinations of n-hexane with methyl ethyl ketone and also methyl n-butyl ketone with methyl ethyl ketone show increase in peripheral neuropathy, a progressive disorder of nerves of extremities.</p> <p>Combinations with chloroform also show increase in toxicity.</p> <p>Experiments with rats exposed to MIBK have shown nerve changes characteristic of neuropathy (disease of the peripheral nerves usually causing weakness and numbness).</p> <p>Chronic occupational exposure to 500 ppm MIBK in air (20-30 mins/day, and 80 ppm for the remainder of the workday resulted in nausea, headache, burning eyes, and weakness in over half the workers. Some workers reported somnolence, insomnia and intestinal pain, and 4/19 appeared to have enlarged livers. This study was continued 5 years after MIBK concentrations had been reduced to 100-105 ppm for the 20-30 minutes exposures and 50 ppm for the general exposure. A few workers still experienced gastrointestinal and neurological problems and slight liver enlargement was found in two individuals.</p> <p>Long term or repeated ingestion exposure of isopropanol may produce incoordination, lethargy and reduced weight gain.</p> <p>Repeated inhalation exposure to isopropanol may produce narcosis, incoordination and liver degeneration. Animal data show developmental effects only at exposure levels that produce toxic effects in the adult animals. Isopropanol does not cause genetic damage in bacterial or mammalian cell cultures or in animals.</p> <p>There are inconclusive reports of human sensitisation from skin contact with isopropanol. Chronic alcoholics are more tolerant of systemic isopropanol than are persons who do not consume alcohol; alcoholics have survived as much as 500 ml. of 70% isopropanol.</p> <p>Continued voluntary drinking of a 2.5% aqueous solution through two successive generations of rats produced no reproductive effects.</p> <p>NOTE: Commercial isopropanol does not contain 'isopropyl oil'. An excess incidence of sinus and laryngeal cancers in isopropanol production workers has been shown to be caused by the byproduct 'isopropyl oil'. Changes in the production processes now ensure that no byproduct is formed. Production changes include use of dilute sulfuric acid at higher temperatures.</p>	
838AR (Aerosol) Total Ground Carbon Conductive Paint	TOXICITY	IRRITATION
	Not Available	Not Available
acetone	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 20000 mg/kg ^[2]	Eye (human): 500 ppm - irritant
	Inhalation(Mouse) LC50; 44 mg/L4h ^[2]	Eye (rabbit): 20mg/24hr -moderate
	Oral (Rat) LD50; 5800 mg/kg ^[2]	Eye (rabbit): 3.95 mg - SEVERE
		Eye: adverse effect observed (irritating) ^[1]
		Skin (rabbit): 500 mg/24hr - mild
		Skin (rabbit):395mg (open) - mild
		Skin: no adverse effect observed (not irritating) ^[1]
methyl ethyl ketone	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 6480 mg/kg ^[2]	Eye (human): 350 ppm -irritant
	Inhalation(Mouse) LC50; 32 mg/L4h ^[2]	Eye (rabbit): 80 mg - irritant
	Oral (Rat) LD50; 2054 mg/kg ^[1]	Skin (rabbit): 402 mg/24 hr - mild
		Skin (rabbit):13.78mg/24 hr open

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methyl isobutyl ketone	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >16000 mg/kg ^[1]	Eye (human): 200 ppm/15m
	Inhalation(Rat) LC50; ~8.2-16.4 mg/l4h ^[2]	Eye (rabbit): 40 mg - SEVERE
	Oral (Rat) LD50; 2080 mg/kg ^[2]	Eye (rabbit): 500 mg/24h - mild Skin (rabbit): 500 mg/24h - mild
propane	TOXICITY	IRRITATION
	Inhalation(Rat) LC50; >13023 ppm4h ^[1]	Not Available
isopropanol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 12800 mg/kg ^[2]	Eye (rabbit): 10 mg - moderate
	Inhalation(Mouse) LC50; 53 mg/L4h ^[2]	Eye (rabbit): 100 mg - SEVERE
	Oral (Mouse) LD50; 3600 mg/kg ^[2]	Eye (rabbit): 100mg/24hr-moderate Skin (rabbit): 500 mg - mild
iso-butane	TOXICITY	IRRITATION
	Inhalation(Rat) LC50; >13023 ppm4h ^[1]	Not Available
n-butyl acetate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 3200 mg/kg ^[2]	Eye (human): 300 mg
	Inhalation(Rat) LC50; 0.74 mg/l4h ^[2]	Eye (rabbit): 20 mg (open)-SEVERE
	Oral (Rabbit) LD50; 3200 mg/kg ^[2]	Eye (rabbit): 20 mg/24h - moderate
		Eye: no adverse effect observed (not irritating) ^[1]
		Skin (rabbit): 500 mg/24h-moderate Skin: no adverse effect observed (not irritating) ^[1]
ethyl acetate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >18000 mg/kg ^[2]	Eye (human): 400 ppm
	Inhalation(Mouse) LC50; >18 mg/l4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1] Skin: no adverse effect observed (not irritating) ^[1]
carbon black	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >3000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1] Skin: no adverse effect observed (not irritating) ^[1]
propylene glycol monomethyl ether acetate, alpha-isomer	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1] Skin: no adverse effect observed (not irritating) ^[1]
barium dinonyl naphthalenesulfonate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	Eye (rabbit): 250 mg/5d mild
	Inhalation(Rat) LC50; >5.25 mg/L4h ^[2]	
	Oral (Rat) LD50; 3000 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	

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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 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.

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METHYL ISOBUTYL KETONE	<p>For methyl isobutyl ketone (MIBK):</p> <p>MIBK is primarily absorbed by the lungs in animals and humans; it can however be absorbed by the gastrointestinal system and through skin. In two cases involving individuals exposed to the vapour MIBK was found in the brain, liver, lung, vitreous fluid, kidney and blood.</p> <p>Experiments in guinea pigs show that MIBK is metabolised to 4-hydroxy-4-methyl-2-pentanone and 4-methyl-2-pentanol. Ketones are generally excreted rapidly in expired air. Small amounts of MIBK are also excreted in the urine. Humans excreted less than 0.1% of the dose as unmetabolised MIBK in the urine within the first 3 hours post exposure. Serum half-life in guinea pigs is about 55 minutes with a clearance time of 6 hours</p> <p>In animal studies, the acute systemic toxicity of MIBK, via the oral and inhalation routes of exposure, is low. In a 90-day gavage study on rats, a no-observed-effect level (NOEL) of 50 mg/kg per day was found. In 90-day inhalation studies on rats and mice, concentrations of up to 4100 mg/m³ (1000 ppm) did not result in significant toxicity, though compound-related reversible morphological changes were reported in the liver and kidney. Evidence of central nervous system depression was seen in animals exposed to a level of 4100 mg/m³ (1000 ppm). In a number of studies, exposure to MIBK concentrations as low as 1025 mg/m³ (250 ppm) resulted in an increase in liver size and induced hepatic microsomal metabolism. This may be responsible for the exacerbation of haloalkane toxicity and for the potentiation of the neurotoxicity of <i>n</i>-hexane. MIBK was also found to potentiate the cholestatic effects of manganese given with, or without, bilirubin. In 90-day studies on mice, rats, dogs, and monkeys, only male rats developed hyaline droplets in the proximal tubules of the kidney. Effects on behaviour were reported in baboons exposed for 7 days to 205 mg/m³ (50 ppm). At a concentration of 4100 mg/m³ (1000 ppm), MIBK was not embryotoxic, foetotoxic, or teratogenic in rats or mice. Foetotoxicity was only observed at concentrations of MIBK that caused maternal toxicity. MIBK did not induce gene mutations in <i>in vitro</i> bacterial test systems with, or without, metabolic activation. Negative results were also obtained <i>in vitro</i> with, or without, metabolic activation, in tests for mitotic gene conversion in yeast, and for gene mutation in cultured mammalian cells. The results of <i>in vitro</i> assays for unscheduled DNA synthesis in primary rat hepatocytes and for structural chromosome damage in cultured rat liver cells were negative. An <i>in vivo</i> micronucleus test on mice was negative. These data indicate that MIBK is not genotoxic. No long-term or carcinogenicity studies are available. The toxicity of MIBK for aquatic organisms and microorganisms is low.</p>
ISOPROPANOL	<p>For isopropanol (IPA):</p> <p>Acute toxicity: Isopropanol has a low order of acute toxicity. It is irritating to the eyes, but not to the skin. Very high vapor concentrations are irritating to the eyes, nose, and throat, and prolonged exposure may produce central nervous system depression and narcosis. Human volunteers reported that exposure to 400 ppm isopropanol vapors for 3 to 5 min. caused mild irritation of the eyes, nose and throat.</p> <p>Although isopropanol produced little irritation when tested on the skin of human volunteers, there have been reports of isolated cases of dermal irritation and/or sensitization. The use of isopropanol as a sponge treatment for the control of fever has resulted in cases of intoxication, probably the result of both dermal absorption and inhalation. There have been a number of cases of poisoning reported due to the intentional ingestion of isopropanol, particularly among alcoholics or suicide victims. These ingestions typically result in a comatose condition. Pulmonary difficulty, nausea, vomiting, and headache accompanied by various degrees of central nervous system depression are typical. In the absence of shock, recovery usually occurred.</p> <p>Repeat dose studies: The systemic (non-cancer) toxicity of repeated exposure to isopropanol has been evaluated in rats and mice by the inhalation and oral routes. The only adverse effects-in addition to clinical signs identified from these studies were to the kidney.</p> <p>Reproductive toxicity: A recent two-generation reproductive study characterised the reproductive hazard for isopropanol associated with oral gavage exposure. This study found that the only reproductive parameter apparently affected by isopropanol exposure was a statistically significant decrease in male mating index of the F1 males. It is possible that the change in this reproductive parameter was treatment related and significant, although the mechanism of this effect could not be discerned from the results of the study. However, the lack of a significant effect of the female mating index in either generation, the absence of any adverse effect on litter size, and the lack of histopathological findings of the testes of the high-dose males suggest that the observed reduction in male mating index may not be biologically meaningful.</p> <p>Developmental toxicity: The developmental toxicity of isopropanol has been characterized in rat and rabbit developmental toxicity studies. These studies indicate that isopropanol is not a selective developmental hazard. Isopropanol produced developmental toxicity in rats, but not in rabbits. In the rat, the developmental toxicity occurred only at maternally toxic doses and consisted of decreased foetal body weights, but no teratogenicity</p> <p>Genotoxicity: All genotoxicity assays reported for isopropanol have been negative</p> <p>Carcinogenicity: rodent inhalation studies were conducted to evaluate isopropanol for cancer potential. The only tumor rate increase seen was for interstitial (Leydig) cell tumors in the male rats. Interstitial cell tumors of the testis is typically the most frequently observed spontaneous tumor in aged male Fischer 344 rats. These studies demonstrate that isopropanol does not exhibit carcinogenic potential relevant to humans. Furthermore, there was no evidence from this study to indicate the development of carcinomas of the testes in the male rat, nor has isopropanol been found to be genotoxic. Thus, the testicular tumors seen in the isopropanol exposed male rats are considered of no significance in terms of human cancer risk assessment</p> <p>The substance is classified by IARC as Group 3:</p> <p>NOT classifiable as to its carcinogenicity to humans.</p> <p>Evidence of carcinogenicity may be inadequate or limited in animal testing.</p>
N-BUTYL ACETATE	<p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
CARBON BLACK	<p>Inhalation (rat) TCLo: 50 mg/m³/6h/90D-I Nil reported</p>
PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER	<p>A BASF report (in ECETOC) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects. The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.I] *Shin-Etsu SDS</p> <p>for propylene glycol ethers (PGEs):</p> <p>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).</p> <p>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.</p> <p>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).</p> <p>This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product.</p> <p>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.</p> <p>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.</p>

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	<p>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.</p> <p>None are skin sensitizers.</p> <p>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).</p> <p>Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m3 (600 ppm) for PnB and 2,010 mg/m3 (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m3 (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m3 (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members.</p> <p>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.</p> <p>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.</p> <p>The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. <i>In vitro</i>, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic <i>in vivo</i>. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice. A BASF report (in ECETOC) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects.</p> <p>The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.]</p>
BARIUM DINONYL NAPHTHALENESULFONATE	<p>Toxicity information for barium sulfonates (barium salts of various alkyl and aryl sulfonic acids in oil solution):</p> <p>For dinonylnaphthalenes:</p> <p>The chemicals exhibit a very low order of toxicity to rats or rabbits by the oral, inhalation, or dermal routes.</p> <p>Human sensitisation study results are available for two members of the category (dinonylnaphthalene sulfonic acid, calcium salt; dinonylnaphthalene sulfonic acid, barium salt). Neither is a sensitizer.</p> <p>Based on the available toxicity results, dinonylnaphthalene sulfonic acid, barium salt appears to be the most biologically active member of the category.</p> <p>for alkaryl sulfonate petroleum additives:</p> <p>Mammalian Toxicology - Acute. Existing data on acute mammalian toxicity indicates a low concern for acute toxicity.</p> <p>Acute oral toxicity: In all but one studies, there were no deaths that could be attributed to treatment with the test material when administered at the limit dose of 2000 or 5000 mg/kg. In some studies, the primary clinical observations were diarrhea and reduced food consumption (without a change in body weight). These effects are consistent with the gastrointestinal irritant properties of detergents in an oil-based vehicle. In other studies, decreased body weight gain or ruffled fur was observed. In one study where deaths occurred, animals were administered dose levels well above the 2000 mg/kg limit dose. Overall, the acute oral LD50 for these substances was greater than the 2000 mg/kg limit dose indicating a relatively low order of toxicity.</p> <p>Acute dermal toxicity: No mortality was observed for any tested substance when administered at the limit dose of 2000 or 5000 mg/kg. The principal clinical observation was erythema and/or edema at the site of dermal application. In some cases, the cutaneous findings included dry, flaky skin, desquamation and hyperkeratosis. Overall, the acute dermal LD50 for these substances was greater than the 2000 mg/kg limit dose indicating a relatively low order of toxicity.</p> <p>Acute inhalation toxicity: One member of the petroleum additive alkaryl sulfonate category (CAS RN: 6878396-0) was tested for acute inhalation toxicity (OECD Guideline 403, Acute Inhalation Toxicity). Rats were exposed whole-body to an aerosol of the substance at a nominal atmospheric concentration of 1.9 mg/L for four hours. This was the maximum attainable concentration due to the low volatility and high viscosity of the test material. No mortality was noted, and all animals fully recovered following depuration. Clinical signs of toxicity during exposure included reduced activity, matted coat, and closed eyes. Clinical signs of toxicity observed post exposure included lacrimation, nasal discharge, salivation rates, matted coat, hunched appearance, soft stools and closed eyes. No treatment-related macroscopic findings were noted. The lack of mortality at a concentration just below the limit dose of 2.0 mg/L indicates a relatively low order of toxicity for this substance.</p> <p>Mammalian Toxicology - Subchronic Toxicity. Existing data from repeated-dose toxicity studies indicates minimal signs of toxicity following repeated oral exposure. Adverse effects at the site of contact were observed following repeated dermal exposure (injury to the skin) and repeated inhalation (injury to the lungs).</p> <p>NOAELs range from 49.5 mg/m3 to 1000 mg/kg/day</p> <p>Mammalian Toxicology - Reproductive and Developmental Toxicity. A one-generation reproductive toxicity test was conducted on one member of the category (CAS # 115733-09-0). Exposure to the alkaryl sulfonate did not significantly impact reproduction or development and these results were bridged to the remainder of the category.</p> <p>Mammalian Toxicology - Mutagenicity. Existing data from bacterial reverse mutation assays and in vitro and in vivo chromosome aberration studies indicate a low concern for mutagenicity.</p> <p>Animal Irritation</p> <p>An acute eye irritation study indicates that calcium dodecylbenzenesulfonate caused irritation.</p> <p>Result: irritating at 0.1 ml</p> <p>An acute skin study indicate that calcium dodecylbenzenesulfonate is irritant to skin 0.5 ml according to OECD GHS guidelines.</p> <p>Respiratory irritation was not observed. There were no treatment-related changes in the haematological or urinalysis values in any of the animals. No signs of irritation of respiratory tract and nasal effects were observed.</p> <p>For dinonylnaphthalenesulfonic acid (DNNSA) and its salts:</p> <p>In general, a compound needs to be dissolved before it can be taken up from the gastro-intestinal tract after oral administration. Calcium bis(di C8-C10, branched, C9 rich, alkylnaphthalene sulphonate) (CaDNNSA) has a measured water solubility of 0.266 mg/L and therefore it is expected to dissolve into the gastrointestinal fluids to a very limited extent. Uptake by passive diffusion is possible, but limited due to the high molecular weight of the salt (average MW 959) and its dissociation product DNNSA (MW 461). CaDNNSA has a high log Pow 6.6), which makes the compound relatively hydrophobic. This characteristic will enable micellar solubilisation by bile salts in the gastro-intestinal tract which allows some crossing of lipid biomembranes. The structure contains an ionizable group (SO3H), which might hamper diffusion across biological membranes. In addition, the molecular size of the molecule of 19 Å does not favor uptake across the biological membranes.</p>

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In the 90-day study on CaDNNSA in the highest dose group 6/10 females died showing alterations in the gastro-intestinal tract, a small thymus and bone marrow atrophy. The surviving females at 1000 mg/kg bw showed similar effects and a reduced body weight (gain). The effects on the gastro-intestinal tract also became apparent in males at 300 and 1000 mg/kg bw. These animals also had a reduced body weight (gain). Other effects included changes in numbers of white blood cells, lymphocytes, platelets as well as effects on several biochemical parameters. Macroscopy and histopathology indicated that next to the GI-tract mainly the thymus and bone marrow could be considered as potentially affected in males at 300 mg/kg bw and above and in females at 1000 mg/kg bw. The effects on blood and blood forming organs as well as on the immune system are indicative for some absorption of the substance. This absorption may be enhanced due to the effects on the gastro intestinal tract lining.

The metabolism of DNNSA salts is mainly contingent on both the nature of the alkyl groups and the nature and extent of naphthalene ring substitutions. There are currently no metabolism studies of CaDNNSA, however, the US EPA has evaluated the metabolism of analogs in the sodium alkyl naphthalenesulfonate cluster (SANS), a group of sodium salts of naphthalenesulfonic acids. In a US EPA final rule for SANS, it was stated that "the 1- or 2-sulfonic acid sodium salt moieties on the naphthalene ring may provide a handle by which these compounds can be readily conjugated and eliminated." Though the available information is not definitive for CaDNNSA, where the alkyl chains are much larger than for the naphthalenesulfonic acids evaluated by EPA, it is expected that the metabolism of the substance will be a factor, enhancing elimination. If absorbed, wide distribution of the CaDNNSA throughout the body is not expected based on its molecular size (18 Å). In general, molecules of this size do not pass readily through cell membranes, thus limiting wide distribution. Excretion of CaDNNSA and its potential metabolites will occur via the bile (high molecular weight) or the urine (low molecular weight).

Irritation:

Calcium bis(di-C8-10, branched, C9 rich, alkylnaphthalenesulphonate) is irritating to skin and eyes. It is not corrosive.

Sensitisation:

In the Buehler assay the substance was shown to be a weak skin sensitiser, while a human patch test showed no sensitization in human volunteers.

Genetic toxicity:

The Barium analog was found to be non-mutagenic in the Ames bacterial reverse mutation assay and the mouse lymphoma test (MLA). The substance was did not cause chromosomal aberrations in human peripheral lymphocytes.

Reproductive toxicity:

DNNSA (di C8-C10, branched, C9 rich, alkylnaphthalene sulphonic acid) is the major structural component of Calcium bis(di c8-c10, branched, c9 rich, alkylnaphthalene sulphonate). The OECD 422 repeat dose and reproduction/development study with DNNSA provides reliable read-across for developmental endpoints for Calcium bis(di c8-c10, branched, c9 rich, alkylnaphthalene sulphonate).

A second OECD 422 study conducted with another analog, Barium bis(di c8-c10, branched, c9 rich, alkylnaphthalene sulphonate), showed no effects on development at the highest dose in the study of 150 mg/kg/day. Together these studies show that Calcium bis(di c8-c10, branched, c9 rich, alkylnaphthalene sulphonate) is not a developmental toxin.

*REACH Dossier

Animal studies show that calcium sulfonates with a TBN greater than 300 are not skin sensitisers while the results in animals at a TBN (Total Base Number) of 300 exhibit a mixed skin sensitisation response. However, human repeat insult patch tests clearly show that high TBN overbased calcium sulfonates (TBN = 300) are not sensitisers and that low TBN calcium sulfonates do not cause sensitisation in a substantial number of persons at concentrations of 10% or lower within the definition of sensitisation under EU Regulation (EC) No. 1272/2008.

The weight-of-evidence indicates that low TBN sodium and calcium sulfonates (TBN < 300) are skin sensitisers with a specific concentration limit (SCL) of 10% and that high TBN sodium and calcium sulfonates (TBN = 300) are not skin sensitisers. Studies in guinea pigs show that low TBN benzenesulfonic acid, mono-C20-24 (even)-sec-alkyl derivs., para-, sodium salts (EC No. None; CAS No. None; TBN = 3) is a skin sensitizer while benzenesulfonic acid, mono-C20-24 (even)-sec-alkyl derivs., para-, sodium salts TBN = 448) is not a skin sensitiser. Studies in guinea pigs and human volunteers show that low TBN benzenesulfonic acid, 4-(mono-C15-36 branched alkyl derivs., C24 rich) and benzenesulfonic acid, 4-octadecyl, calcium salts (EC 939-141-9; TBN = 13) are skin sensitisers. Numerous well-conducted, reliable, controlled human (HRIPT) studies with benzene, polypropylene derivs., sulfonated, calcium salts (EC 616-278-7; TBN values ranging from 13 to 85), sulfonic acids, petroleum, calcium salts (EC 263-093-9; TBN = 30 to 100), and benzenesulfonic acid, 4-(mono-C15-36 branched alkyl derivs., C24 rich) and benzenesulfonic acid, 4-octadecyl, calcium salts (EC 939-141-6; TBN = 13) show that low TBN calcium sulfonates do not cause sensitisation in a substantial number of subjects at 10% and lower. High TBN calcium sulfonates, sulfonic acids, petroleum, calcium salts (EC 263-093-9; TBN = 375 and 400) do not cause skin sensitisation in guinea pigs. Results of guinea pigs studies at TBN = 300 are mixed; two studies of sulfonic acids, petroleum, calcium salts, (EC 263-093-9) report no skin sensitisation while one study of sulfonic acids, petroleum, calcium salts (EC 263-093-9) and one study of benzene, polypropylene derivs., sulfonated, calcium salts (EC 616-278-7) report skin sensitisation. However, numerous well-conducted, reliable, controlled human (HRIPT) studies with benzene, polypropylene derivs., sulfonated, calcium salts (EC 616-278-7; TBN = 300) and sulfonic acids, petroleum, calcium salts (EC 263-093-9; TBN = 300) also show that high TBN (TBN = 300) do not cause skin sensitisation. In accordance with EU CLP Regulation (EC) No. 1272/2008, classification is required for low TBN sodium and calcium sulfonates (TBN < 300) with a specific concentration limit of 10% and classification is not required for high TBN calcium sulfonates (TBN = 300).

Linear alkylbenzene sulfonates (LAS) are classified as Irritant (Xi) with the risk phrases R38 (Irritating to skin) and R41 (Risk of serious damage to eyes) according to CESIO (CESIO 2000). LAS are not included in Annex 1 of list of dangerous substances of Council Directive 67/548/EEC. Linear alkylbenzene sulfonic acids (LABS) are strong acids (pKa<2) are classified as corrosive (R34)

Branched materials exhibit comparable toxicity to linear species.

Acute toxicity: The available data indicate minimal to moderate toxicity, with LD50 values ranging from 500 to 2000 mg/kg body weight (bw).

Acute inhalation data also indicate a lack of significant toxicity. Available dermal exposure data also shows a lack of significant toxicity.

LAS are readily absorbed by the gastrointestinal tract after oral administration in animals. LAS are not readily absorbed through the skin. The bulk is metabolised in the liver to sulfophenyl carboxylic acids. The metabolites are excreted primarily via the urine and faeces. The main urinary metabolites in rats are sulfophenyl butanoic acid and sulfophenyl pentanoic acid. Accumulation of LAS or its main metabolites has not been established in any organ after repeated oral ingestion.

No serious injuries or fatalities in man have been reported following accidental ingestion of LAS-containing detergent. The main clinical signs observed after oral administration to rats of doses near or greater than the LD50 values consisted of reduced voluntary activity, diarrhoea, weakness etc. Death usually occurred within 24 hours of administration. Rats appear to be more sensitive to LAS than mice.

LAS and branched alkylbenzene sulfonates may cause irritation of the eyes, skin and mucous membranes. LAS are relatively more irritating to the skin than the corresponding branched alkylbenzene sulfonates. The potential of LAS to irritate the skin depends on the concentration applied. LAS have been classified as irritating to skin at concentrations above 20% according to EU-criteria. Human skin can tolerate contact with solution of up to 1% LAS for 24 hours resulting in only mild irritation. Application of > 5% LAS to the eyes of rabbits produced irritation. Concentration of < 0.1% LAS produced mild to no irritation.

Skin sensitization was not seen in 2,294 volunteers exposed to LAS or in 17,887 exposed to formulations of LAS.

Repeat dose toxicity: A feeding study indicated that LAS, when administered for 2 years at extremely high levels (0.5%) in the diets to rats, produced no adverse effects on growth, health or feed efficiency.

Genotoxicity: The mutagenic potential of LAS was tested using *Salmonella typhimurium* strains, using Ames test. In these studies, LAS was not mutagenic. The available long-term studies are inadequate for evaluating the carcinogenic potential of LAS in laboratory animals. The studies available (oral administration to rats and mice) do not show any evidence of carcinogenicity.

Reproductive toxicity: In general no specific effect of LAS on reproductive processes has been seen, although dosages causing maternal toxicity may also induce some effects on reproduction. No teratogenic effects attributed to LAS exposure have been observed.

Environmental and Health Assessment of Substances in Household Detergents and Cosmetic Detergent Products, Environment Project, 615, 2001. Torben Madsen et al: Miljøministeriet (Danish Environmental Protection Agency)

For aromatic sulfonic acids

Aromatic sulfonic acids are very corrosive as was demonstrated in skin and eye irritation studies, in the acute oral studies, and in the single repeated dose oral study.

Health records from industrial manufacturing exposure, including manufacturing plant book of injuries and a physician report, show toluene-4-sulphonic acid (as handled in manufacturing plants; i.e., a 65% aqueous solution with < 5% free sulphuric acid) is an irritant to the eye and skin.

Sensitisation:

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	<p>There is a single, key study for sensitization of the aromatic sulphonic acids. None of the tested animals showed positive responses in a, well documented, GLP guinea pig sensitization study with toluene-4-sulphonic acid (CAS No. 104-15-4). The test substance can be considered a non-sensitizer in guinea pigs as none of the test animals showed a positive response to combined intradermal and topical induction followed by topical challenge.</p> <p>Repeat dose toxicity:</p> <p>A GLP guideline study with p-toluenesulphonic acid (CAS No. 104-15-4) reported no adverse effects to male and female rats exposed orally for 28 days. The highest dose was 500 mg/kg bw/day (>490 mg/kg bw/day based on >98% active ingredient). Therefore the NOAEL was set at 500 mg/kg bw/day.</p> <p>Toxicity to reproduction:</p> <p>No fertility studies are reported for the aromatic sulphonic acids. There are however studies for the chemically related hydrotrope substances that looked at reproductive organs and development of offspring. Hydrotropes are the salt form of the sulphonic acids and therefore are used as read-across for this endpoint. The 90-day oral rat and oral mouse studies and the 2-year chronic dermal rat and mouse studies with the closely related compound sodium xylene sulfonate (CAS No. 1300-72-7) included examination of sex organs of both sexes. No treatment related effects on reproductive organs were reported at doses roughly equivalent to those in the developmental toxicity study. The NOAEL for both maternal and foetal toxicity was the highest dose tested - 3000 mg/kg bw /day which is equivalent to 936 mg active ingredient per kilogram body weight per day. The conclusion of the study was no indications of developmental toxicity including teratogenesis.</p> <p>Genetic toxicity:</p> <p>There is a fully documented, GLP Guideline (OECD 471) Ames Test and a fully documented, GLP Guideline (OECD 473) Chromosome Aberration Test for one of the aromatic sulphonic acids, p-toluenesulphonic acid (CAS No. 104-15-4). Both tests were conducted with and without metabolic activation. The Ames test exposed up to 5000 micrograms/plate and the chromosome aberration test exposed up to 1902 micrograms per liter of the test substance. These studies conclude the substance is neither mutagenic nor cytotoxic.</p> <p>There is an additional, published report of an Ames Test for another of the aromatic sulphonic acids, benzenesulfonic acid (CAS No. 98-11-3). Exposures up to 10,000 micrograms/plate were done with and without metabolic activation. The conclusion is the same as for the p-toluenesulphonic acid; that is, not mutagenic and not cytotoxic.</p> <p>There are no in vivo mutagenicity studies for the aromatic sulphonic acids, but there are two in vivo mouse micronucleus studies for the related hydrotropes – sodium cumene sulfonate (CAS 28348-53-0) and calcium xylene sulfonate (CAS 28088-63-3). Both are GLP-compliant Guideline mouse micronucleus studies with full documentation. Both studies conclude the test substances were not mutagenic in these assays.</p> <p>Disulfonic acids have not been the subject of concern.</p> <p>Carcinogenicity:</p> <p>There are no carcinogenicity studies for the aromatic sulphonic acids. Two hydrotrope studies involve 2-year rat and mouse dermal exposures conducted under GLP. Up to 240 mg (rats) and 727 mg (mice) sodium xylenesulfonate/kg body weight in 50% ethanol were dosed 5 days per week for 104 weeks. There were no treatment related incidences of mononuclear cell leukemia, neoplasms, or nonneoplastic lesions of the skin and other organs. The increased incidence of epidermal hyperplasia may have been related to exposure to the test substance. The NOAEL was reported as 240 mg/kg bw/day for rats and 727 mg/kg bw/day for mice.</p> <p>Elimination:</p> <p>The US EPA has evaluated the metabolism of analogs in the sodium alkyl naphthalenesulfonate cluster (SANS), a group of sodium salts of naphthalenesulfonic acids. In a US EPA final rule for SANS, it was stated that "the 1- or 2-sulfonic acid sodium salt moieties on the naphthalene ring may provide a handle by which these compounds can be readily conjugated and eliminated."</p>
838AR (Aerosol) Total Ground Carbon Conductive Paint & N-BUTYL ACETATE	<p>Generally, linear and branched-chain alkyl esters are hydrolysed to their component alcohols and carboxylic acids in the intestinal tract, blood and most tissues throughout the body. Following hydrolysis the component alcohols and carboxylic acids are metabolized.</p> <p>Oral acute toxicity studies have been reported for 51 of the 67 esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids. The very low oral acute toxicity of this group of esters is demonstrated by oral LD50 values greater than 1850 mg/kg bw.</p> <p>Genotoxicity studies have been performed in vitro using the following esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids: methyl acetate, butyl acetate, butyl stearate and the structurally related isoamyl formate and demonstrates that these substances are not genotoxic.</p> <p>The JECFA Committee concluded that the substances in this group would not present safety concerns at the current levels of intake the esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids are generally used as flavouring substances up to average maximum levels of 200 mg/kg. Higher levels of use (up to 3000 mg/kg) are permitted in food categories such as chewing gum and hard candy. In Europe the upper use levels for these flavouring substances are generally 1 to 30 mg/kg foods and in special food categories like candy and alcoholic beverages up to 300 mg/kg foods.</p> <p>International Program on Chemical Safety: the Joint FAO/WHO Expert Committee on Food Additives (JECFA)</p> <p>Esters of Aliphatic acyclic primary alcohols with aliphatic linear saturated carboxylic acids.; 1998</p>
838AR (Aerosol) Total Ground Carbon Conductive Paint & ACETONE	<p>for acetone:</p> <p>The acute toxicity of acetone is low. Acetone is not a skin irritant or sensitizer but is a defatting agent to the skin. Acetone is an eye irritant. The subchronic toxicity of acetone has been examined in mice and rats that were administered acetone in the drinking water and again in rats treated by oral gavage. Acetone-induced increases in relative kidney weight changes were observed in male and female rats used in the oral 13-week study. Acetone treatment caused increases in the relative liver weight in male and female rats that were not associated with histopathologic effects and the effects may have been associated with microsomal enzyme induction. Haematologic effects consistent with macrocytic anaemia were also noted in male rats along with hyperpigmentation in the spleen. The most notable findings in the mice were increased liver and decreased spleen weights. Overall, the no-observed-effect-levels in the drinking water study were 1% for male rats (900 mg/kg/d) and male mice (2258 mg/kg/d), 2% for female mice (5945 mg/kg/d), and 5% for female rats (3100 mg/kg/d). For developmental effects, a statistically significant reduction in foetal weight, and a slight, but statistically significant increase in the percent incidence of later resorptions were seen in mice at 15,665 mg/m3 and in rats at 26,100 mg/m3. The no-observable-effect level for developmental toxicity was determined to be 5220 mg/m3 for both rats and mice.</p> <p>Teratogenic effects were not observed in rats and mice tested at 26,110 and 15,665 mg/m3, respectively. Lifetime dermal carcinogenicity studies in mice treated with up to 0.2 mL of acetone did not reveal any increase in organ tumor incidence relative to untreated control animals.</p> <p>The scientific literature contains many different studies that have measured either the neurobehavioural performance or neurophysiological response of humans exposed to acetone. Effect levels ranging from about 600 to greater than 2375 mg/m3 have been reported. Neurobehavioral studies with acetone-exposed employees have recently shown that 8-hr exposures in excess of 2375 mg/m3 were not associated with any dose-related changes in response time, vigilance, or digit span scores. Clinical case studies, controlled human volunteer studies, animal research, and occupational field evaluations all indicate that the NOAEL for this effect is 2375 mg/m3 or greater.</p>
838AR (Aerosol) Total Ground Carbon Conductive Paint & METHYL ETHYL KETONE	<p>Methyl ethyl ketone is considered to have a low order of toxicity; however methyl ethyl ketone is often used in combination with other solvents and the toxic effects of the mix may be greater than either solvent alone. Combinations of n-hexane with methyl ethyl ketone and also methyl n-butyl ketone with methyl ethyl ketone show increase in peripheral neuropathy, a progressive disorder of nerves of extremities. Combinations with chloroform also show increase in toxicity.</p>
ACETONE & METHYL ISOBUTYL KETONE & ISOPROPANOL	<p>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 epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p>
METHYL ETHYL KETONE & METHYL ISOBUTYL KETONE & ISOPROPANOL & ETHYL ACETATE	<p>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.</p>

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	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.
METHYL ETHYL KETONE & N-BUTYL ACETATE	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.
METHYL ISOBUTYL KETONE & CARBON BLACK	WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
PROPANE & CARBON BLACK & BARIUM DINONYL NAPHTHALENESULFONATE	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

11.2.1. Endocrine Disruption Properties

Not Available

SECTION 12 Ecological information

12.1. Toxicity

838AR (Aerosol) Total Ground Carbon Conductive Paint	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available

acetone	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	12h	Fish	0.001mg/L	4
	LC50	96h	Fish	3744.6-5000.7mg/L	4
	EC50	48h	Crustacea	6098.4mg/L	5
	EC50	96h	Algae or other aquatic plants	9.873-27.684mg/l	4

methyl ethyl ketone	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	48h	Crustacea	68mg/l	2
	LC50	96h	Fish	>324mg/L	4
	EC50	72h	Algae or other aquatic plants	1972mg/l	2
	EC50	48h	Crustacea	308mg/l	2
	EC50	96h	Algae or other aquatic plants	>500mg/l	4

methyl isobutyl ketone	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	48h	Crustacea	170mg/l	1
	LC50	96h	Fish	>179mg/l	2
	EC50	48h	Crustacea	170mg/l	1
	EC50	96h	Algae or other aquatic plants	400mg/l	1

propane	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	96h	Algae or other aquatic plants	7.71mg/l	2
	LC50	96h	Fish	24.11mg/l	2
	EC50	96h	Algae or other aquatic plants	7.71mg/l	2

isopropanol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	24h	Algae or other aquatic plants	0.011mg/L	4
	LC50	96h	Fish	4200mg/l	4
	EC50	72h	Algae or other aquatic plants	>1000mg/l	1
	EC50	48h	Crustacea	7550mg/l	4
	EC50	96h	Algae or other aquatic plants	>1000mg/l	1

Continued...

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iso-butane	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	96h	Algae or other aquatic plants	7.71mg/l	2
	LC50	96h	Fish	24.11mg/l	2
	EC50	96h	Algae or other aquatic plants	7.71mg/l	2
n-butyl acetate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	96h	Fish	18mg/l	2
	LC50	96h	Fish	18mg/l	2
	EC50	72h	Algae or other aquatic plants	246mg/l	2
	EC50	48h	Crustacea	32mg/l	1
ethyl acetate	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	72h	Algae or other aquatic plants	>100mg/l	1
	LC50	96h	Fish	>75.6mg/l	2
	EC50	48h	Crustacea	164mg/l	1
carbon black	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	24h	Crustacea	3200mg/l	1
	LC50	96h	Fish	>100mg/l	2
	EC50	72h	Algae or other aquatic plants	>0.2mg/l	2
	EC50	48h	Crustacea	33.076-41.968mg/l	4
propylene glycol monomethyl ether acetate, alpha-isomer	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	336h	Fish	47.5mg/l	2
	LC50	96h	Fish	>100mg/l	2
	EC50	72h	Algae or other aquatic plants	>1000mg/l	2
	EC50	48h	Crustacea	373mg/l	2
	EC50	96h	Algae or other aquatic plants	>1000mg/l	2
barium dinonyl naphthalenesulfonate	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
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			

for methyl isobutyl ketone (MIBK)

log Kow : 1.19-1.31

Koc : 19-106

Half-life (hr) air : 15-17

Half-life (hr) H₂O surface water : 15-33

Henry's atm m³ /mol: 9.40E-05

BOD 5: 0.12-2.14, 4. 4%

COD : 2.16, 79%

ThOD : 2.72

BCF : 2-5

Environmental fate:

MIBK has a short half-life in the atmosphere and is also biodegraded in water. It is not expected to bioaccumulate. The toxicity of MIBK for microorganisms and aquatic organisms is low

MIBK is not expected to be retarded by absorption to soils rich in organic matter; therefore it is expected to be mobile in soil and subject to leaching.

MIBK may contribute to the formation of photochemical smog.

The relatively high vapour pressure (14.5 mm Hg at 20 °C) and estimated Henry's Law Constant (9.4 x 10⁻⁵ atm-m³/mol, 20 °C) indicate that it volatilise from moist and dry soil. When released to water it does not adsorb significantly to suspended solids, and will volatilise to the atmosphere.

Transformation and Persistence:

Air: The main degradation pathway for MIBK in the atmosphere is reaction with photochemically produced hydroxyl radicals. The half-life of MIBK from the reaction with hydroxyl radicals has been estimated to be 16-17 hours. based on its UV light absorption spectrum, direct photolysis of MIBK is expected to occur with a half-life of about 15 hours; acetone is a photo-oxidation product.

Smog chamber studies indicate MIBK is moderately reactive with nitrogen oxides producing acetone, peroxyacetyl nitrate and methyl nitrate. As a volatile organic chemical (VOC)

MIBK can contribute to photochemical smog in the presence of other VOCs

Soil: In wet or dry soil, MIBK may undergo volatilisation to air and photolysis on the soil surface. It is highly mobile and may be leached from the soil by water, and is susceptible to aerobic degradation by mixed populations of microorganisms.

Water: MIBK is not expected to bioconcentrate in fish and other aquatic organisms; its estimated bioconcentration factor is 2.

Ecotoxicity:

The toxicity of MIBK in aquatic organisms is low; toxicity values are greater than 100 mg/l.

MIBK also has low toxicity in terrestrial rodents for oral and inhalation exposure. It is moderately toxic to birds, based on oral LD50 values between 50 to 500 mg/kg (redwinged blackbirds)

Fish LC50 (96 h): fathead minnow (Pimephales promelas) 505 mg/l; (24 h): goldfish (Carassius auratus) 460 mg/l

Daphnia magna LC50 (24 h): 4280 mg/l

Brine shrimp LC50 (24 h): 1230 mg/l

Continued...

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For methyl ethyl ketone:

log Kow : 0.26-0.69

log Koc : 0.69

Koc : 34

Half-life (hr) air : 2.3

Half-life (hr) H₂O surface water : 72-288

Henry's atm m³/mol: 1.05E-05

BOD 5 : 1.5-2.24, 46%

COD : 2.2-2.31, 100%

ThOD : 2.44

BCF : 1

Environmental fate:

TERRESTRIAL FATE: Measured Koc values of 29 and 34 were obtained for methyl ethyl ketone in silt loams. Methyl ethyl ketone is expected to have very high mobility in soil. Volatilisation of methyl ethyl ketone from dry soil surfaces is expected based upon an experimental vapor pressure of 91 mm Hg at 25 deg C. Volatilization from moist soil surfaces is also expected given the measured Henry's Law constant of 4.7x10⁻⁵ atm-cu m/mole. The volatilisation half-life of methyl ethyl ketone from silt and sandy loams was measured as 4.9 days. Methyl ethyl ketone is expected to biodegrade under both aerobic and anaerobic conditions as indicated by numerous screening tests.

AQUATIC FATE: Based on Koc values, methyl ethyl ketone is not expected to adsorb to suspended solids and sediment in water. Methyl ethyl ketone is expected to volatilise from water surfaces based on the measured Henry's Law constant. Estimated half-lives for a model river and model lake are 19 and 197, hours respectively. Biodegradation of this compound is expected based upon numerous screening tests. An estimated BCF value of 1 based on an experimental log Kow of 0.29, suggests that bioconcentration in aquatic organisms is low.

ATMOSPHERIC FATE: According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere, methyl ethyl ketone, which has an experimental vapor pressure of 91 mm Hg at 25 deg C, will exist solely as a vapor in the ambient atmosphere. Vapour-phase methyl ethyl ketone is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be about 14 days. Methyl ethyl ketone is also expected to undergo photodecomposition in the atmosphere by natural sunlight. Photochemical degradation of methyl ethyl ketone by natural sunlight is expected to occur at approximately 1/5 the rate of degradation by photochemically produced hydroxyl radicals.

Ecotoxicity:

Fish LC50 (24 h): bluegill sunfish (*Lepomis macrochirus*) 1690-5640 mg/l; guppy (*Lebistes reticulatus*) 5700 mg/l; goldfish (*Carassius auratus*) >5000 mg/l

Fish LC50 (96 h): fathead minnow (*Pimephales promelas*) 3200 mg/l; bluegill sunfish (*Lepomis macrochirus*) 4467 mg/l; mosquito fish (*Gambusia affinis*) 5600 mg/l

Daphnia magna LC50 (48 h): <520-1382 mg/l

Daphnia magna LC50 (24 h): 8890 mg/l

Brine shrimp (*Artemia salina*) LC50 (24 h): 1950 mg/l

For isopropanol (IPA):

log Kow : -0.16- 0.28

Half-life (hr) air : 33-84

Half-life (hr) H₂O surface water : 130

Henry's atm m³/mol: 8.07E-06

BOD 5: 1.19,60%

COD : 1.61-2.30,97%

ThOD : 2.4

BOD 20: >70% * [Akzo Nobel]

Environmental Fate

Based on calculated results from a lever 1 fugacity model, IPA is expected to partition primarily to the aquatic compartment (77.7%) with the remainder to the air (22.3%). IPA has been shown to biodegrade rapidly in aerobic, aqueous biodegradation tests and therefore, would not be expected to persist in aquatic habitats. IPA is also not expected to persist in surface soils due to rapid evaporation to the air. In the air, physical degradation will occur rapidly due to hydroxyl radical (OH) attack. Overall, IPA presents a low potential hazard to aquatic or terrestrial biota.

IPA is expected to volatilise slowly from water based on a calculated Henry's Law constant of 7.52 x 10⁻⁶ atm.m³/mole. The calculated half-life for the volatilisation from surface water (1 meter depth) is predicted to range from 4 days (from a river) to 31 days (from a lake). Hydrolysis is not considered a significant degradation process for IPA. However, aerobic biodegradation of IPA has been shown to occur rapidly under non-acclimated conditions, based on a result of 49% biodegradation from a 5 day BOD test. Additional biodegradation data developed using standardized test methods show that IPA is readily biodegradable in both freshwater and saltwater media (72 to 78% biodegradation in 20 days).

IPA will evaporate quickly from soil due to its high vapor pressure (43 hPa at 20°C), and is not expected to partition to the soil based on a calculated soil adsorption coefficient (log Koc) of 0.03.

IPA has the potential to leach through the soil due to its low soil adsorption

In the air, isopropanol is subject to oxidation predominantly by hydroxyl radical attack. The room temperature rate constants determined by several investigators are in good agreement for the reaction of IPA with hydroxyl radicals. The atmospheric half-life is expected to be 10 to 25 hours, based on measured degradation rates ranging from 5.1 to 7.1 x 10⁻¹² cm³/molecule-sec, and an OH concentration of 1.5 x 10⁶ molecule/cm³, which is a commonly used default value for calculating atmospheric half-lives. Using OH concentrations representative of polluted (3 x 10⁶) and pristine (3 x 10⁵) air, the atmospheric half-life of IPA would range from 9 to 126 hours, respectively. Direct photolysis is not expected to be an important transformation process for the degradation of IPA.

Ecotoxicity:

IPA has been shown to have a low order of acute aquatic toxicity. Results from 24- to 96-hour LC50 studies range from 1,400 to more than 10,000 mg/L for freshwater and saltwater fish and invertebrates. In addition, 16-hour to 8-day toxicity threshold levels (equivalent to 3% inhibition in cell growth) ranging from 104 to 4,930 mg/L have been demonstrated for various microorganisms.

Chronic aquatic toxicity has also been shown to be of low concern, based on 16- to 21-day NOEC values of 141 to 30 mg/L, respectively, for a freshwater invertebrate.

Bioconcentration of IPA in aquatic organisms is not expected to occur based on a measured log octanol/water partition coefficient (log Kow) of 0.05, a calculated bioconcentration factor of 1 for a freshwater fish, and the unlikelihood of constant, long-term exposures.

Toxicity to Plants

Toxicity of IPA to plants is expected to be low, based on a 7-day toxicity threshold value of 1,800 mg/L for a freshwater algae, and an EC50 value of 2,100 mg/L from a lettuce seed germination test.

For ketones:

Ketones, unless they are alpha, beta--unsaturated ketones, can be considered as narcosis or baseline toxicity compounds

Hydrolysis may also involve the addition of water to ketones to yield ketals under mild acid conditions. However, this addition of water is thermodynamically favorable only for low molecular weight ketones. This addition is an equilibrium reaction that is reversible upon a change of water concentration and the reaction ultimately leads to no permanent change in the structure of the ketone substrate. The higher molecular weight ketones do not form stable ketals. Therefore, the ketones are stable to water under ambient environmental conditions. Another possible reaction of ketones in water involves the enolic hydrogen on the carbons bonded to the carbonyl function. Under conditions of high pH (pH greater than 10), the enolic proton is abstracted by base (OH-) forming a carbanion intermediate that may react with other organic substrates (e.g., ketones, esters, aldehydes) containing a center for nucleophilic attack. The reactions, commonly recognized as condensation reactions, produce higher molecular weight products. Under ambient conditions of temperature, pH, and low concentration, these condensation reactions are unfavorable.

Based on its reactions in air, it seems likely that ketones undergo photolysis in water. It is probable that ketones will be biodegraded to an appreciable degree by micro-organisms in soil and water. They are unlikely to bioconcentrate or biomagnify.

For isobutane:

Refrigerant Gas: Saturated Hydrocarbons have zero ozone depletion potential (ODP) and will photodegrade under atmospheric conditions. [Calor Gas]

Environmental Fate

Terrestrial fate: An estimated Koc value of 35 suggests that isobutane will have very high mobility in soil. Its very high Henry's Law constant, 4.08 atm-cu m/mole, (calculated from its vapor pressure and water solubility, high vapor pressure, 2611 mm Hg at 25 deg C, and low adsorptivity to soil indicate that volatilisation will be an important fate process from both moist and dry soil surfaces. Isobutane is biodegradable, especially under acclimated conditions, and may biodegrade in soil.

Aquatic fate: The estimated Koc value suggests that isobutane would not adsorb to sediment and particulate matter in the water column. Additional evidence that isobutane is not removed to sediment has been obtained from microcosm experiments. Isobutane will readily volatilise from water based on its estimated Henry's Law constant of 4.08 atm-cu m/mole. Estimated half-lives for a model river and model lake are 2.2 hr and 3.0 days, respectively. An estimated BCF value of 74 based on the log Kow suggests that isobutane will not bioconcentrate in aquatic organisms.

Results indicate that gas exchange is the dominant removal mechanism for isobutane gases from the water column following a hypothetical input. The volatilisation half-lives for

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isobutane from the water columns in natural estuaries are estimated to be 4.4 and 6.8 days at 20 and 10 deg C, respectively.

Isobutane also biodegrades in the microcosm at a rate that is slower than for n-butane and ethane in susceptibility. Biodegradation of isobutane initially occurs with a half-lives of 16-26 days at 20 deg C and 33-139 days at 10 deg C, significantly slower than the loss predicted by gas exchange from typical natural estuaries. However, after a lag of 2-4 weeks, the biodegradation rate increases markedly so that in the case of chronic inputs, biodegradation can become the dominant removal mechanism.

Atmospheric fate: Isobutane is a gas at ordinary temperatures. It is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is 6.9 days, assuming a hydroxyl radical concn of 5×10^5 radicals per cubic cm. When isobutane was exposed to sunlight for 6 hr in a tedlar bag filled with Los Angeles air, 6% of the isobutane degraded. The air contained 4529 ppb-C hydrocarbons and 870 ppb of NOX. The tropospheric loss of volatile hydrocarbons such as isobutane by wet and dry deposition are believed to be of minor importance. Indeed, isobutane assimilated into precipitation may evaporate during transport as well as being reemitted into the atmosphere after deposition. Isobutane is a contributor to the production of PAN (peroxyacyl nitrates) under photochemical smog conditions

For propane:

Environmental Fate

Terrestrial fate: An estimated Koc value of 460 determined from a log Kow of 2.36 indicates that propane is expected to have moderate mobility in soil. Volatilisation of propane from moist soil surfaces is expected to be an important fate process given an estimated Henry's Law constant of 7.07×10^{-1} atm-cu m/mole, derived from its vapor pressure, 7150 mm Hg, and water solubility, 62.4 mg/L. Propane is expected to volatilise from dry soil surfaces based upon its vapor pressure. Using cell suspensions of microorganisms isolated from soil and water, propane was oxidised to acetone within 24 hours, suggesting that biodegradation may be an important fate process in soil and sediment.

Aquatic fate: The estimated Koc value indicates that propane is expected to adsorb to suspended solids and sediment. Volatilisation from water surfaces is expected based upon an estimated Henry's Law constant. Using this Henry's Law constant volatilisation half-lives for a model river and model lake are estimated to be 41 minutes and 2.6 days, respectively.

An estimated BCF of 13.1 using log Kow suggests the potential for bioconcentration in aquatic organisms is low. After 192 hr, the trace concentration of propane contained in gasoline remained unchanged for both a sterile control and a mixed culture sample collected from ground water contaminated with gasoline. This indicates that biodegradation may not be an important fate process in water.

Atmospheric fate: According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere and vapour pressure, propane is expected to exist solely as a gas in the ambient atmosphere. Gas-phase propane is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 14 days, calculated from its rate constant of 1.15×10^{-12} cu cm/molecule-sec at 25 deg C. Propane does not contain chromophores that absorb at wavelengths >290 nm and therefore is not expected to be susceptible to direct photolysis by sunlight.

DO NOT discharge into sewer or waterways.

for acetone:

log Kow: -0.24

Half-life (hr) air: 312-1896

Half-life (hr) H₂O surface water: 20

Henry's atm m³/mol: 3.67×10^{-5}

BOD 5: 0.31-1.76, 46-55%

COD: 1.12-2.07

ThOD: 2.2

BCF: 0.69

Environmental fate:

Acetone preferentially locates in the air compartment when released to the environment. A substantial amount of acetone can also be found in water, which is consistent with the high water to air partition coefficient and its small, but detectable, presence in rain water, sea water, and lake water samples. Very little acetone is expected to reside in soil, biota, or suspended solids. This is entirely consistent with the physical and chemical properties of acetone and with measurements showing a low propensity for soil absorption and a high preference for moving through the soil and into the ground water.

In air, acetone is lost by photolysis and reaction with photochemically produced hydroxyl radicals; the estimated half-life of these combined processes is about 22 days. The relatively long half-life allows acetone to be transported long distances from its emission source.

Acetone is highly soluble and slightly persistent in water, with a half-life of about 20 hours; it is minimally toxic to aquatic life.

Acetone released to soil volatilises although some may leach into the ground where it rapidly biodegrades.

Acetone does not concentrate in the food chain.

Acetone meets the OECD definition of readily biodegradable which requires that the biological oxygen demand (BOD) is at least 70% of the theoretical oxygen demand (THOD) within the 28-day test period.

Drinking Water Standard: none available.

Soil Guidelines: none available.

Air Quality Standards: none available.

Ecotoxicity:

Testing shows that acetone exhibits a low order of toxicity

Fish LC50: brook trout 6070 mg/l; fathead minnow 15000 mg/l

Bird LC0 (5 day): Japanese quail, ring-neck pheasant 40,000 mg/l

Daphnia magna LC50 (48 h): 15800 mg/l; NOEC 8500 mg/l

Aquatic invertebrate 2100 - 16700 mg/l

Aquatic plant NOEC: 5400-7500 mg/l

Daphnia magna chronic NOEC 1660 mg/l

Acetone vapors were shown to be relatively toxic to two types insects and their eggs. The time to 50% lethality (LT50) was found to be 51.2 hr and 67.9 hr when the flour beetle (*Tribolium confusum*) and the flour moth (*Ephesia kuehniella*) were exposed to an airborne acetone concentration of 61.5 mg/m³. The LT50 values for the eggs were 30-50% lower than for the adult. The direct application of acetone liquid to the body of the insects or surface of the eggs did not, however, cause any mortality.

The ability of acetone to inhibit cell multiplication has been examined in a wide variety of microorganisms. The results have generally indicated mild to minimal toxicity with NOECs greater than 1700 mg/L for exposures lasting from 6 hr to 4 days. Longer exposure periods of 7 to 8 days with bacteria produced mixed results; but overall the data indicate a low degree of toxicity for acetone. The only exception to these findings were the results obtained with the flagellated protozoa (*Entosiphon sulcatum*) which yielded a 3-day NOEC of 28 mg/L.

For n-butyl acetate:

Half-life (hr) air: 144

Half-life (hr) H₂O surface water: 178-27156

Henry's atm m³/mol: 3.20×10^{-4}

BOD 5 if unstated: 0.15-1.02, 7%

COD: 78%

ThOD: 2.207

BCF: 4-14

Environmental Fate:

TERRESTRIAL FATE: An estimated Koc value of 200 determined from a measured log Kow of 1.78 indicates that n-butyl acetate is expected to have moderate mobility in soil.

Volatilisation of n-butyl acetate is expected from moist soil surfaces given its Henry's Law constant of 2.8×10^{-4} atm-cu m/mole. Volatilisation from dry soil surfaces is expected based on a measured vapor pressure of 11.5 mm Hg. Using a standard BOD dilution technique and a sewage inoculum, theoretical BODs of 56 % to 86 % were observed during 5-20 day incubation periods, which suggests that n-butyl acetate may biodegrade in soil.

AQUATIC FATE: An estimated Koc value indicates that n-butyl acetate is not expected to adsorb to suspended solids and sediment in water. Butyl acetate is expected to volatilise from water surfaces based on a Henry's Law constant of 2.8×10^{-4} atm-cu m/mole. Estimated half-lives for a model river and model lake are 7 and 127, hours respectively. An estimated BCF value of 10 based on the log Kow, suggests that bioconcentration in aquatic organisms is low. Using a filtered sewage seed, 5-day and 20-day theoretical BODs of 58 % and 83 % were measured in freshwater dilution tests; 5-day and 20-day theoretical BODs of 40 % and 61 % were measured in salt water. A 5-day theoretical BOD of 56.8 % and 51.8 % were measured for n-butyl acetate in distilled water and seawater, respectively. Hydrolysis may be an important environmental fate for this compound based upon experimentally determined hydrolysis half-lives of 114 and 11 days at pH 8 and 9 respectively.

ATMOSPHERIC FATE: According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere, n-butyl acetate, which has a vapour pressure of 11.5 mm Hg at 25 deg C, is expected to exist solely as a vapor in the ambient atmosphere. Vapour-phase n-butyl acetate is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be about 4 days

Environmental fate:

Fish LC50 (96 h, 23 C): island silverside (Menidia beryllina) 185 ppm (static bioassay in synthetic seawater, mild aeration applied after 24 h); bluegill sunfish (Lepomis macrochirus)

Continued...

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100 ppm (static bioassay in fresh water, mild aeration applied after 24 h)

Fish EC50 (96 h): fathead minnow (*Pimephales promelas*) 18 mg/l (affected fish lost equilibrium prior to death)

Daphnia LC50 (48 h): 44 ppm

Algal LC50 (96 h): *Scenedesmus* 320 ppm

12.2. Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
acetone	LOW (Half-life = 14 days)	MEDIUM (Half-life = 116.25 days)
methyl ethyl ketone	LOW (Half-life = 14 days)	LOW (Half-life = 26.75 days)
methyl isobutyl ketone	HIGH (Half-life = 7001 days)	LOW (Half-life = 1.9 days)
propane	LOW	LOW
isopropanol	LOW (Half-life = 14 days)	LOW (Half-life = 3 days)
iso-butane	HIGH	HIGH
n-butyl acetate	LOW	LOW
ethyl acetate	LOW (Half-life = 14 days)	LOW (Half-life = 14.71 days)
propylene glycol monomethyl ether acetate, alpha-isomer	LOW	LOW

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
acetone	LOW (BCF = 0.69)
methyl ethyl ketone	LOW (LogKOW = 0.29)
methyl isobutyl ketone	LOW (LogKOW = 1.31)
propane	LOW (LogKOW = 2.36)
isopropanol	LOW (LogKOW = 0.05)
iso-butane	LOW (BCF = 1.97)
n-butyl acetate	LOW (BCF = 14)
ethyl acetate	HIGH (BCF = 3300)
propylene glycol monomethyl ether acetate, alpha-isomer	LOW (LogKOW = 0.56)

12.4. Mobility in soil

Ingredient	Mobility
acetone	HIGH (KOC = 1.981)
methyl ethyl ketone	MEDIUM (KOC = 3.827)
methyl isobutyl ketone	LOW (KOC = 10.91)
propane	LOW (KOC = 23.74)
isopropanol	HIGH (KOC = 1.06)
iso-butane	LOW (KOC = 35.04)
n-butyl acetate	LOW (KOC = 20.86)
ethyl acetate	LOW (KOC = 6.131)
propylene glycol monomethyl ether acetate, alpha-isomer	HIGH (KOC = 1.838)

12.5. Results of PBT and vPvB assessment

	P	B	T
Relevant available data	Not Available	Not Available	Not Available
PBT	✗	✗	✗
vPvB	✗	✗	✗
PBT Criteria fulfilled?	No		
vPvB	No		

12.6. Endocrine Disruption Properties

Not Available

12.7. Other adverse effects

Not Available

SECTION 13 Disposal considerations

13.1. Waste treatment methods

Product / Packaging disposal	<p>▶ DO NOT allow wash water from cleaning or process equipment to enter drains.</p> <p>▶ It may be necessary to collect all wash water for treatment before disposal.</p>
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Continued...

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	<ul style="list-style-type: none"> ▶ 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. ▶ Consult State Land Waste Management Authority for disposal. ▶ Discharge contents of damaged aerosol cans at an approved site. ▶ Allow small quantities to evaporate. ▶ DO NOT incinerate or puncture aerosol cans. ▶ Bury residues and emptied aerosol cans at an approved site.
Waste treatment options	Not Available
Sewage disposal options	Not Available

SECTION 14 Transport information

Labels Required

	
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Land transport (ADR-RID)

14.1. UN number	1950												
14.2. UN proper shipping name	AEROSOLS												
14.3. Transport hazard class(es)	<table> <tr> <td>Class</td><td>2.1</td></tr> <tr> <td>Subrisk</td><td>Not Applicable</td></tr> </table>	Class	2.1	Subrisk	Not Applicable								
Class	2.1												
Subrisk	Not Applicable												
14.4. Packing group	Not Applicable												
14.5. Environmental hazard	Not Applicable												
14.6. Special precautions for user	<table> <tr> <td>Hazard identification (Kemler)</td><td>Not Applicable</td></tr> <tr> <td>Classification code</td><td>5F</td></tr> <tr> <td>Hazard Label</td><td>2.1</td></tr> <tr> <td>Special provisions</td><td>190 327 344 625</td></tr> <tr> <td>Limited quantity</td><td>1 L</td></tr> <tr> <td>Tunnel Restriction Code</td><td>2 (D)</td></tr> </table>	Hazard identification (Kemler)	Not Applicable	Classification code	5F	Hazard Label	2.1	Special provisions	190 327 344 625	Limited quantity	1 L	Tunnel Restriction Code	2 (D)
Hazard identification (Kemler)	Not Applicable												
Classification code	5F												
Hazard Label	2.1												
Special provisions	190 327 344 625												
Limited quantity	1 L												
Tunnel Restriction Code	2 (D)												

Air transport (ICAO-IATA / DGR)

14.1. UN number	1950														
14.2. UN proper shipping name	Aerosols, flammable														
14.3. Transport hazard class(es)	<table> <tr> <td>ICAO/IATA Class</td><td>2.1</td></tr> <tr> <td>ICAO / IATA Subrisk</td><td>Not Applicable</td></tr> <tr> <td>ERG Code</td><td>10L</td></tr> </table>	ICAO/IATA Class	2.1	ICAO / IATA Subrisk	Not Applicable	ERG Code	10L								
ICAO/IATA Class	2.1														
ICAO / IATA Subrisk	Not Applicable														
ERG Code	10L														
14.4. Packing group	Not Applicable														
14.5. Environmental hazard	Not Applicable														
14.6. Special precautions for user	<table> <tr> <td>Special provisions</td><td>A145 A167 A802</td></tr> <tr> <td>Cargo Only Packing Instructions</td><td>203</td></tr> <tr> <td>Cargo Only Maximum Qty / Pack</td><td>150 kg</td></tr> <tr> <td>Passenger and Cargo Packing Instructions</td><td>203</td></tr> <tr> <td>Passenger and Cargo Maximum Qty / Pack</td><td>75 kg</td></tr> <tr> <td>Passenger and Cargo Limited Quantity Packing Instructions</td><td>Y203</td></tr> <tr> <td>Passenger and Cargo Limited Maximum Qty / Pack</td><td>30 kg G</td></tr> </table>	Special provisions	A145 A167 A802	Cargo Only Packing Instructions	203	Cargo Only Maximum Qty / Pack	150 kg	Passenger and Cargo Packing Instructions	203	Passenger and Cargo Maximum Qty / Pack	75 kg	Passenger and Cargo Limited Quantity Packing Instructions	Y203	Passenger and Cargo Limited Maximum Qty / Pack	30 kg G
Special provisions	A145 A167 A802														
Cargo Only Packing Instructions	203														
Cargo Only Maximum Qty / Pack	150 kg														
Passenger and Cargo Packing Instructions	203														
Passenger and Cargo Maximum Qty / Pack	75 kg														
Passenger and Cargo Limited Quantity Packing Instructions	Y203														
Passenger and Cargo Limited Maximum Qty / Pack	30 kg G														

Sea transport (IMDG-Code / GGVSee)

14.1. UN number	1950				
14.2. UN proper shipping name	AEROSOLS				
14.3. Transport hazard class(es)	<table> <tr> <td>IMDG Class</td><td>2.1</td></tr> <tr> <td>IMDG Subrisk</td><td>Not Applicable</td></tr> </table>	IMDG Class	2.1	IMDG Subrisk	Not Applicable
IMDG Class	2.1				
IMDG Subrisk	Not Applicable				
14.4. Packing group	Not Applicable				
14.5. Environmental hazard	Not Applicable				

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14.6. Special precautions for user	EMS Number	F-D , S-U
	Special provisions	63 190 277 327 344 381 959
	Limited Quantities	1000 ml

Inland waterways transport (ADN)

14.1. UN number	1950	
14.2. UN proper shipping name	AEROSOLS	
14.3. Transport hazard class(es)	2.1	Not Applicable
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Classification code	5F
	Special provisions	190; 327; 344; 625
	Limited quantity	1 L
	Equipment required	PP, EX, A
	Fire cones number	1

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

Not Applicable

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

Product name	Group
acetone	Not Available
methyl ethyl ketone	Not Available
methyl isobutyl ketone	Not Available
propane	Not Available
isopropanol	Not Available
iso-butane	Not Available
n-butyl acetate	Not Available
ethyl acetate	Not Available
carbon black	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available
barium dinonyl naphthalenesulfonate	Not Available

14.9. Transport in bulk in accordance with the ICG Code

Product name	Ship Type
acetone	Not Available
methyl ethyl ketone	Not Available
methyl isobutyl ketone	Not Available
propane	Not Available
isopropanol	Not Available
iso-butane	Not Available
n-butyl acetate	Not Available
ethyl acetate	Not Available
carbon black	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available
barium dinonyl naphthalenesulfonate	Not Available

SECTION 15 Regulatory information

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

acetone is found on the following regulatory lists

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)
EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles
Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)
European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

Continued...

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methylethyl ketone is found on the following regulatory lists

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)
 EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances
 EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

Europe EC Inventory
 European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)
 European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

methylethyl ketone is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
 EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)
 EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles
 Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)
 European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
 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 2B: Possibly carcinogenic to humans

propane is found on the following regulatory lists

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles
 Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)
 European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

isopropanol is found on the following regulatory lists

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles
 Europe EC Inventory
 European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
 International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

isobutane is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
 EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles
 EU REACH Regulation (EC) No 1907/2006 - Annex XVII (Appendix 1) Carcinogens: Category 1 A
 EU REACH Regulation (EC) No 1907/2006 - Annex XVII (Appendix 4) Germ cell mutagens: Category 1 B

Europe EC Inventory
 European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)
 European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

n-butyl acetate is found on the following regulatory lists

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)
 EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles
 Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)
 European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

ethyl acetate is found on the following regulatory lists

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)
 EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles
 Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)
 European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

carbon black is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
 EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances
 Europe EC Inventory
 European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

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 2B: Possibly carcinogenic to humans
 International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

propylene glycol monomethyl ether acetate, alpha-isomer is found on the following regulatory lists

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)
 EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles
 Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)
 European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

barium dinonyl naphthalenesulfonate is found on the following regulatory lists

Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

This safety data sheet is in compliance with the following EU legislation and its adaptations - as far as applicable - : Directives 98/24/EC, - 92/85/EEC, - 94/33/EC, - 2008/98/EC, - 2010/75/EU; Commission Regulation (EU) 2020/878; Regulation (EC) No 1272/2008 as updated through ATPs.

15.2. Chemical safety assessment

No Chemical Safety Assessment has been carried out for this substance/mixture by the supplier.

National Inventory Status

Continued...

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National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (acetone; methyl ethyl ketone; methyl isobutyl ketone; propane; isopropanol; iso-butane; n-butyl acetate; ethyl acetate; carbon black; propylene glycol monomethyl ether acetate, alpha-isomer; barium dinonyl naphthalenesulfonate)
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	Yes
Vietnam - NCI	Yes
Russia - FBEPH	Yes
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	26/01/2022
Initial Date	01/09/2018

Full text Risk and Hazard codes

H220	Extremely flammable gas.
H225	Highly flammable liquid and vapour.
H226	Flammable liquid and vapour.
H280	Contains gas under pressure; may explode if heated.
H302+H332	Harmful if swallowed or if inhaled.
H315	Causes skin irritation.
H318	Causes serious eye damage.
H332	Harmful if inhaled.
H335	May cause respiratory irritation.
H411	Toxic to aquatic life with long lasting effects.

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.

For detailed advice on Personal Protective Equipment, refer to the following EU CEN Standards:

EN 166 Personal eye-protection

EN 340 Protective clothing

EN 374 Protective gloves against chemicals and micro-organisms

EN 13832 Footwear protecting against chemicals

EN 133 Respiratory protective devices

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

838AR (Aerosol) Total Ground Carbon Conductive Paint

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

Reason for Change

A-2.00 - Added UFI number and format changes to the safety data sheet