843AR Super Shield Silver Coated Copper Conductive Paint (Aerosol)
MG Chemicals UK Limited

Version No: **A-2.00**
Safety Data Sheet (Conforms to Regulation (EU) No 2020/878)

Issue Date: **12/03/2021**
Revision Date: **12/03/2021**
L.REACH.GBR.EN

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

1.1. Product Identifier

Product name	843AR
Synonyms	SDS Code: 843AR-Aerosol; 843AR-140G, 843AR-340G | UFI:82M0-70V9-700X-8WTR
Other means of identification	Super Shield Silver Coated Copper 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

Classification according to regulation (EC) No 1272/2008 [CLP] and amendments [1]	H336 - Specific target organ toxicity - single exposure Category 3 (narcotic effects), H411 - Chronic Aquatic Hazard Category 2, H223+H229 - Aerosols Category 2, H319 - Eye Irritation Category 2
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

2.2. Label elements

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

Hazard statement(s)

H336	May cause drowsiness or dizziness.
H411	Toxic to aquatic life with long lasting effects.
H223+H229	Flammable aerosol; Pressurized container: may burst if heated.
H319	Causes serious eye irritation.

Supplementary statement(s)

EUH066	Repeated exposure may cause skin dryness or cracking.

Precautionary statement(s) Prevention

Page 1 continued...

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.
P261	Avoid breathing gas
P273	Avoid release to the environment.
P280	Wear protective gloves/protective clothing/eye protection/face protection/hearing protection/...

Precautionary statement(s) Response

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/... if you feel unwell.
P337+P313	If eye irritation persists: Get medical advice/attention.
P391	Collect spillage.
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.

2.3. Other hazards

Inhalation and/or skin contact may produce health damage*.

Cumulative effects may result following exposure*.

May produce discomfort of the respiratory system*.

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	Classification according to regulation (EC) No 1272/2008 [CLP] and amendments
1.67-64-1 2.200-662-2 3.606-001-00-8 4.01-2119471330-49-XXXX	32	acetone *	Flammable Liquid Category 2, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Eye Irritation Category 2; H225, H336, H319, EUH066 [2]
1.74-98-6 2.200-827-9 3.601-003-00-5 4.01-2119486944-21-XXXX	13	propane	Gas under Pressure, Flammable Gas Category 1; H280, H220 [2]
1.123-86-4 2.204-658-1 3.607-025-00-1 4.01-2119485493-29-XXXX	12	n-butyl acetate *	Flammable Liquid Category 3, Specific target organ toxicity - single exposure Category 3 (narcotic effects); H226, H336, EUH066 [2]
1.616-38-6 2.210-478-4 3.607-013-00-6 4.01-2119548399-23-XXXX|01-2119822377-36-XXXX	12	dimethyl carbonate	Flammable Liquid Category 2; H225 [2]
1.7440-50-8 2.231-159-6 3.029-024-00-X 4.01-2119475516-31-XXXX|01-2119480154-42-XXXX|01-2119480184-39-XXXX|01-2120762783-45-XXXX	10	copper	Chronic Aquatic Hazard Category 2; H411 [2]
1.75-28-5. 2.200-857-2 3.601-004-00-0|601-004-01-8 4.01-2119485395-27-XXXX	7	iso-butane	Flammable Gas Category 1, Gas under Pressure (Liquefied gas); H220, H280[1]
1.110-43-0 2.203-767-1 3.606-024-00-3 4.01-2119902391-49-	7	amyl methyl ketone *	Flammable Liquid Category 3, Acute Toxicity (Oral) Category 4, Acute Toxicity (Inhalation) Category 4; H226, H302, H332 [2]

Continued...

1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classification according to regulation (EC) No 1272/2008 [CLP] and amendments
XXXX|01-2120752829-39-XXXX			
1.108-65-6 2.203-603-9 3.607-195-00-7 4.01-2119475791-29-XXXX	2	propylene glycol monomethyl ether acetate, alpha-isomer *	Flammable Liquid Category 3; H226 [2]
1.7440-22-4 2.231-131-3 3.Not Available 4.01-2119513211-60-XXXX|01-2119555669-21-XXXX	1	silver	EUH210 [1]
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 3. Classification drawn from C&L; * EU IOELVs available*		

SECTION 4 First aid measures

4.1. Description of first aid measures

Eye Contact	If aerosols come in contact with the eyes: ▸ 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	If solids or aerosol mists are deposited upon the skin: ▸ 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	If aerosols, fumes or combustion products are inhaled: ▸ 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	▸ **IF SWALLOWED, REFER FOR MEDICAL ATTENTION, WHERE POSSIBLE, WITHOUT DELAY.** ▸ For advice, contact a Poisons Information Centre or a doctor. ▸ Urgent hospital treatment is likely to be needed. ▸ In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition. ▸ If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS should be provided. Further action will be the responsibility of the medical specialist. ▸ If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS. **Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:** ▸ **INDUCE** vomiting with fingers down the back of the throat, **ONLY IF CONSCIOUS**. Lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. **NOTE:** Wear a protective glove when inducing vomiting by mechanical means. ▸ 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

for copper intoxication:

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

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

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

[ELLENHORN & BARCELOUX: Medical Toxicology]

Treat symptomatically.

Continued...

For acute or short term repeated exposures to acetone:
- Symptoms of acetone exposure approximate ethanol intoxication.
- About 20% is expired by the lungs and the rest is metabolised. Alveolar air half-life is about 4 hours following two hour inhalation at levels near the Exposure Standard; in overdose, saturable metabolism and limited clearance, prolong the elimination half-life to 25-30 hours.
- There are no known antidotes and treatment should involve the usual methods of decontamination followed by supportive care.

[Ellenhorn and Barceloux: Medical Toxicology]
Management:
Measurement of serum and urine acetone concentrations may be useful to monitor the severity of ingestion or inhalation.
Inhalation Management:
- Maintain a clear airway, give humidified oxygen and ventilate if necessary.
- If respiratory irritation occurs, assess respiratory function and, if necessary, perform chest X-rays to check for chemical pneumonitis.
- Consider the use of steroids to reduce the inflammatory response.
- Treat pulmonary oedema with PEEP or CPAP ventilation.

Dermal Management:
- Remove any remaining contaminated clothing, place in double sealed, clear bags, label and store in secure area away from patients and staff.
- Irrigate with copious amounts of water.
- An emollient may be required.

Eye Management:
- Irrigate thoroughly with running water or saline for 15 minutes.
- Stain with fluorescein and refer to an ophthalmologist if there is any uptake of the stain.

Oral Management:
- No **GASTRIC LAVAGE OR EMETIC**
- Encourage oral fluids.

Systemic Management:
- Monitor blood glucose and arterial pH.
- Ventilate if respiratory depression occurs.
- If patient unconscious, monitor renal function.
- Symptomatic and supportive care.

The Chemical Incident Management Handbook:
Guy's and St. Thomas' Hospital Trust, 2000
BIOLOGICAL EXPOSURE INDEX
These represent the determinants observed in specimens collected from a healthy worker exposed at the Exposure Standard (ES or TLV):

Determinant	Sampling Time	Index	Comments
Acetone in urine	End of shift	50 mg/L	NS

NS: Non-specific determinant; also observed after exposure to other material

SECTION 5 Firefighting measures

5.1. Extinguishing media

Metal dust fires need to be smothered with sand, inert dry powders.
DO NOT USE WATER, CO2 or FOAM.
- Use DRY sand, graphite powder, dry sodium chloride based extinguishers, G-1 or Met L-X to smother fire.
- Confining or smothering material is preferable to applying water as chemical reaction may produce flammable and explosive hydrogen gas.
- Chemical reaction with CO2 may produce flammable and explosive methane.
- If impossible to extinguish, withdraw, protect surroundings and allow fire to burn itself out.
- Alcohol stable foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.
- Water spray or fog - Large fires only.
- **DO NOT** use halogenated fire extinguishing agents.

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

5.3. Advice for firefighters

Fire Fighting	‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear breathing apparatus plus protective gloves in the event of a fire. ‣ Prevent, by any means available, spillage from entering drains or water courses. ‣ Use fire fighting procedures suitable for surrounding area. ‣ **DO NOT** approach containers suspected to be hot. ‣ Cool fire exposed containers with water spray from a protected location. ‣ If safe to do so, remove containers from path of fire. ‣ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	‣ **DO NOT** disturb burning dust. Explosion may result if dust is stirred into a cloud, by providing oxygen to a large surface of hot metal. ‣ **DO NOT** use water or foam as generation of explosive hydrogen may result. With the exception of the metals that burn in contact with air or water (for example, sodium), masses of combustible metals do not represent unusual fire risks because they have the ability to conduct heat away from hot spots so efficiently that the heat of combustion cannot be maintained - this means that it will require a lot of heat to ignite a mass of combustible metal. Generally, metal fire risks exist when sawdust, machine shavings and other metal 'fines' are present. Metal powders, while generally regarded as non-combustible: ‣ May burn when metal is finely divided and energy input is high. ‣ May react explosively with water. ‣ May be ignited by friction, heat, sparks or flame. ‣ May **REIGNITE** after fire is extinguished. ‣ Will burn with intense heat. Note:

Continued...

	▸ Metal dust fires are slow moving but intense and difficult to extinguish. ▸ Containers may explode on heating. ▸ Dusts or fumes may form explosive mixtures with air. ▸ Gases generated in fire may be poisonous, corrosive or irritating. ▸ Hot or burning metals may react violently upon contact with other materials, such as oxidising agents and extinguishing agents used on fires involving ordinary combustibles or flammable liquids. ▸ Temperatures produced by burning metals can be higher than temperatures generated by burning flammable liquids ▸ Some metals can continue to burn in carbon dioxide, nitrogen, water, or steam atmospheres in which ordinary combustibles or flammable liquids would be incapable of burning. carbon dioxide (CO_2) metal oxides other pyrolysis products typical of burning organic material. **Contains low boiling substance:** Closed containers may rupture due to pressure buildup under fire conditions. May emit poisonous fumes. **WARNING**: Aerosol containers may present pressure related hazards.

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	▸ 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	▸ 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	▸ 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	▸ Store in original containers. ▸ Keep containers securely sealed. ▸ Store in a cool, dry, well-ventilated area. ▸ Store away from incompatible materials and foodstuff containers. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.

7.2. Conditions for safe storage, including any incompatibilities

Continued...

Suitable container	For low viscosity materials ▸ Drums and jerricans must be of the non-removable head type. ▸ Where a can is to be used as an inner package, the can must have a screwed enclosure. For materials with a viscosity of at least 2680 cSt. (23 deg. C) and solids (between 15 C deg. and 40 deg C.): ▸ Removable head packaging; ▸ Cans with friction closures and ▸ low pressure tubes and cartridges may be used. - Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages *. - In addition, where inner packagings are glass and contain liquids of packing group I and II there must be sufficient inert absorbent to absorb any spillage *. - * unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic. ▸ Aerosol dispenser. ▸ Check that containers are clearly labelled.
Storage incompatibility	Inorganic derivative of Group 11 metal. n-Butyl acetate: ▸ 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 Heptanones: ▸ react violently with strong oxidisers, aldehydes, nitric acid, perchloric acid ▸ form a variety of unstable peroxides following reaction with hydrogen peroxide ▸ are incompatible with aliphatic amines, aldehydes, strong bases ▸ Carbonates are incompatible with cerium compounds, germanium, lead diacetate, magnesium, mercurous chloride, silver nitrate ▸ Many metals may incandesce, react violently, ignite or react explosively upon addition of concentrated nitric acid. 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 alkalies 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) 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 ▸ Avoid reaction with oxidising agents, bases and strong reducing agents. Metals exhibit varying degrees of activity. Reaction is reduced in the massive form (sheet, rod, or drop), compared with finely divided forms. The less active metals will not burn in air but: ▸ can react exothermically with oxidising acids to form noxious gases. ▸ catalyse polymerisation and other reactions, particularly when finely divided ▸ react with halogenated hydrocarbons (for example, copper dissolves when heated in carbon tetrachloride), sometimes forming explosive compounds. ▸ Finely divided metal powders develop pyrophoricity when a critical specific surface area is exceeded; this is ascribed to high heat of oxide formation on exposure to air. ▸ Safe handling is possible in relatively low concentrations of oxygen in an inert gas. ▸ Several pyrophoric metals, stored in glass bottles have ignited when the container is broken on impact. Storage of these materials moist and in metal containers is recommended. ▸ The reaction residues from various metal syntheses (involving vacuum evaporation and co-deposition with a ligand) are often pyrophoric. Factors influencing the pyrophoricity of metals are particle size, presence of moisture, nature of the surface of the particle, heat of formation of the oxide, or nitride, mass, hydrogen content, stress, purity and presence of oxide, among others. ▸ Many metals in elemental form react exothermically with compounds having active hydrogen atoms (such as acids and water) to form flammable hydrogen gas and caustic products. ▸ Elemental metals may react with azo/diazo compounds to form explosive products. ▸ Some elemental metals form explosive products with halogenated hydrocarbons.

Continued...

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/m³ (Systemic, Chronic) Inhalation 2 420 mg/m³ (Local, Acute) *Dermal 62 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 200 mg/m³ (Systemic, Chronic) ** *Oral 62 mg/kg bw/day (Systemic, Chronic) **	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)
n-butyl acetate	Dermal 7 mg/kg bw/day (Systemic, Chronic) Inhalation 48 mg/m³ (Systemic, Chronic) Inhalation 300 mg/m³ (Local, Chronic) Dermal 11 mg/kg bw/day (Systemic, Acute) Inhalation 600 mg/m³ (Systemic, Acute) Inhalation 600 mg/m³ (Local, Acute) *Dermal 3.4 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 12 mg/m³ (Systemic, Chronic) ** *Oral 2 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 35.7 mg/m³ (Local, Chronic) ** *Dermal 6 mg/kg bw/day (Systemic, Acute) ** *Inhalation 300 mg/m³ (Systemic, Acute) ** *Oral 2 mg/kg bw/day (Systemic, Acute) ** *Inhalation 300 mg/m³ (Local, Acute) **	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)
dimethyl carbonate	Dermal 5 mg/kg bw/day (Systemic, Chronic) Inhalation 34.9 mg/m³ (Systemic, Chronic) *Dermal 2.5 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 8.7 mg/m³ (Systemic, Chronic) ** *Oral 2.5 mg/kg bw/day (Systemic, Chronic) **	0.5 mg/L (Water (Fresh)) 0.05 mg/L (Water - Intermittent release) 1 mg/L (Water (Marine)) 188 mg/L (STP)
copper	Dermal 137 mg/kg bw/day (Systemic, Chronic) Dermal 273 mg/kg bw/day (Systemic, Acute) *Dermal 137 mg/kg bw/day (Systemic, Chronic) ** *Oral 0.041 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 1 mg/m³ (Local, Chronic) ** *Dermal 273 mg/kg bw/day (Systemic, Acute) ** *Inhalation 1 mg/m³ (Local, Acute) **	3.1 µg/L (Water (Fresh)) 1.2 µg/L (Water - Intermittent release) 0 µg/L (Water (Marine)) 87 mg/kg sediment dw (Sediment (Fresh Water)) 12 mg/kg sediment dw (Sediment (Marine)) 0.7 mg/kg soil dw (Soil) 0.33 mg/L (STP) 0.12 mg/kg food (Oral)
amyl methyl ketone	Dermal 54.27 mg/kg bw/day (Systemic, Chronic) Inhalation 394.25 mg/m³ (Systemic, Chronic) Inhalation 1 516 mg/m³ (Systemic, Acute) *Dermal 23.32 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 84.31 mg/m³ (Systemic, Chronic) ** *Oral 23.32 mg/kg bw/day (Systemic, Chronic) **	0.098 mg/L (Water (Fresh)) 0.01 mg/L (Water - Intermittent release) 0.982 mg/L (Water (Marine)) 1.89 mg/kg sediment dw (Sediment (Fresh Water)) 0.189 mg/kg sediment dw (Sediment (Marine)) 0.321 mg/kg soil dw (Soil) 12.5 mg/L (STP)
propylene glycol monomethyl ether acetate, alpha-isomer	Dermal 796 mg/kg bw/day (Systemic, Chronic) Inhalation 275 mg/m³ (Systemic, Chronic) Inhalation 550 mg/m³ (Local, Acute) *Dermal 320 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 33 mg/m³ (Systemic, Chronic) ** *Oral 36 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 33 mg/m³ (Local, Chronic) **	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)
silver	Inhalation 0.1 mg/m³ (Systemic, Chronic) *Inhalation 0.04 mg/m³ (Systemic, Chronic) ** *Oral 1.2 mg/kg bw/day (Systemic, Chronic) **	0.04 µg/L (Water (Fresh)) 0.86 µg/L (Water - Intermittent release) 438.13 mg/kg sediment dw (Sediment (Fresh Water)) 438.13 mg/kg sediment dw (Sediment (Marine)) 1.41 mg/kg soil dw (Soil) 0.025 mg/L (STP)

* Values for General Population

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
UK Workplace Exposure Limits (WELs)	acetone	Acetone	500 ppm / 1210 mg/m3	3620 mg/m3 / 1500 ppm	Not Available	Not Available
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	acetone	Acetone	500 ppm / 1210 mg/m3	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	n-butyl acetate	Butyl acetate	150 ppm / 724 mg/m3	966 mg/m3 / 200 ppm	Not Available	Not Available
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	n-butyl acetate	n-Butyl acetate	50 ppm / 241 mg/m3	723 mg/m3 / 150 ppm	Not Available	Not Available

Continued...

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
UK Workplace Exposure Limits (WELs)	copper	Copper fume (as Cu)	0.2 mg/m3	Not Available	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	amyl methyl ketone	Heptan-2-one	50 ppm / 237 mg/m3	475 mg/m3 / 100 ppm	Not Available	Sk
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	amyl methyl ketone	Heptan-2-one	50 ppm / 238 mg/m3	475 mg/m3 / 100 ppm	Not Available	Skin
UK Workplace Exposure Limits (WELs)	propylene glycol monomethyl ether acetate, alpha-isomer	1-Methoxypropyl acetate	50 ppm / 274 mg/m3	548 mg/m3 / 100 ppm	Not Available	Sk
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/m3	550 mg/m3 / 100 ppm	Not Available	Skin
UK Workplace Exposure Limits (WELs)	silver	Silver, metallic	0.1 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
acetone	Not Available	Not Available	Not Available
propane	Not Available	Not Available	Not Available
n-butyl acetate	Not Available	Not Available	Not Available
dimethyl carbonate	11 ppm	120 ppm	700 ppm
copper	3 mg/m3	33 mg/m3	200 mg/m3
iso-butane	5500* ppm	17000** ppm	53000*** ppm
amyl methyl ketone	150 ppm	670 ppm	4000* ppm
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available	Not Available	Not Available
silver	0.3 mg/m3	170 mg/m3	990 mg/m3

Ingredient	Original IDLH	Revised IDLH
acetone	2,500 ppm	Not Available
propane	2,100 ppm	Not Available
n-butyl acetate	1,700 ppm	Not Available
dimethyl carbonate	Not Available	Not Available
copper	100 mg/m3	Not Available
iso-butane	Not Available	Not Available
amyl methyl ketone	800 ppm	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available	Not Available
silver	10 mg/m3	Not Available

MATERIAL DATA

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)

Continued...

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 sternabral ossification) - no effects on foetal development were seen in rabbits exposed at 3000 ppm.

For propane
Odour Safety Factor(OSF)
OSF=0.16 (PROPANE)
For amyl methyl ketone:
Odour Threshold Value: 0.18 ppm (detection)
The TLV-TWA is well below the highest level of vapour (1025 ppm) reported to be associated with adverse effects in animals including dermal irritation.
Odour Safety Factor (OSF)
OSF=1.4E2 (2-HEPTANONE)

8.2. Exposure controls

8.2.1. Appropriate engineering controls

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

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

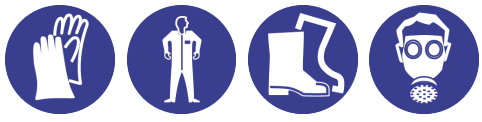
Type of Contaminant:	Speed:
aerosols, (released at low velocity into zone of active generation)	0.5-1 m/s
direct spray, spray painting in shallow booths, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)

Within each range the appropriate value depends on:

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

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

8.2.2. Personal protection



Eye and face protection

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

Skin protection

See Hand protection below

Hands/feet protection

NOTE:
- The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.
- Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.

For esters:
- Do NOT use natural rubber, butyl rubber, EPDM or polystyrene-containing materials.
- No special equipment needed when handling small quantities.
- **OTHERWISE:**
- 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.

Continued...

	▸ Insulated gloves: 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.
Body protection	See Other protection below
Other protection	▸ Overalls. ▸ Eyewash unit. ▸ Barrier cream. ▸ Skin cleansing cream. No special equipment needed when handling small quantities. **OTHERWISE:** ▸ 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:
843AR Super Shield Silver Coated Copper Conductive Paint (Aerosol)

Material	CPI
PE/EVAL/PE	A
TEFLON	B
BUTYL	C
BUTYL/NEOPRENE	C
CPE	C
HYPALON	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
VITON/BUTYL	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.

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.

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**
- Generally not applicable.

Aerosols, in common with most vapours/ mists, should never be used in confined spaces without adequate ventilation. Aerosols, containing agents designed to enhance or mask smell, have triggered allergic reactions in predisposed individuals.

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	Light brown metallic

Physical state	Liquified Gas	**Relative density (Water = 1)**	1.2
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	5 ppm	**Auto-ignition temperature (°C)**	>315
pH (as supplied)	Not Available	**Decomposition temperature**	Not Available

Continued...

Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	72.5
Initial boiling point and boiling range (°C)	>56	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	-17	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	HIGHLY FLAMMABLE.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	13	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	2	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	16	**Gas group**	Not Available
Solubility in water	Partly miscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	>2	**VOC g/L**	Not Available

9.2. Other information

Not Available

SECTION 10 Stability and reactivity

10.1.Reactivity	See section 7.2
10.2. Chemical stability	▸ 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	Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may produce toxic effects; these may be fatal. Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Strong evidence exists that exposure to the material may produce serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. 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. 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 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. Common, generalised symptoms associated with toxic gas inhalation include: ▸ 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. Copper poisoning following exposure to copper dusts and fume may result in headache, cold sweat and weak pulse. Capillary, kidney, liver and brain damage are the longer term manifestations of such poisoning. Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in 'metal fume fever'. Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional

Continued...

	vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure. 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. 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. **WARNING**:Intentional misuse by concentrating/inhaling contents may be lethal. Systemic effects of acetone inhalation exposure include central nervous system depression, light-headedness, incoherent speech, ataxia, stupor, hypotension, tachycardia, metabolic acidosis, hyperglycaemia and ketosis. Rarely, convulsions and tubular necrosis may be evident. Other symptoms of exposure may include restlessness, headache, vomiting, low blood-pressure and rapid and irregular pulse, eye and throat irritation, weakness of the legs and dizziness. Inhalation of high concentrations may produce dryness of the mouth and throat, nausea, uncoordinated movement, loss of coordinated speech, drowsiness and, in severe cases, coma. Inhalation of acetone vapours over long periods causes irritation of the respiratory tract, coughing and headache. Rats exposed to 52200 ppm vapour for 1 hour showed clear signs of narcosis; fatalities occurred at 126600 ppm. Exposure to ketone vapours may produce nose, throat and mucous membrane irritation. High concentrations of vapour may produce central nervous system depression characterised by headache, vertigo, loss of coordination, narcosis and cardiorespiratory failure. Some ketones produce neurological disorders (polyneuropathy) characterised by bilateral symmetrical paresthesia and muscle weakness primarily in the legs and arms.
Ingestion	Severely toxic effects may result from the accidental ingestion of the material; animal experiments indicate that ingestion of less than 5 gram may be fatal or may produce serious damage to the health of the individual. Strong evidence exists that exposure to the material may produce serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by swallowing. 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. 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. Numerous cases of a single oral exposure to high levels of copper have been reported. Consumption of copper-contaminated drinking water has been associated with mainly gastrointestinal symptoms including nausea, abdominal pain, vomiting and diarrhoea. A metallic taste, nausea, vomiting and epigastric burning often occur after ingestion of copper and its derivatives. The vomitus is usually green/blue and discolours contaminated skin. Acute poisonings from the ingestion of copper salts are rare due to their prompt removal by vomiting. Vomiting is due mainly to the local and astringent action of copper ion on the stomach and bowel. Emesis usually occurs within 5 to 10 minutes but may be delayed if food is present in the stomach. Should vomiting not occur, or is delayed, gradual absorption from the bowel may result in systemic poisoning with death, possibly, following within several days. Apparent recovery may be followed by lethal relapse. Systemic effects of copper resemble other heavy metal poisonings and produce wide-spread capillary damage, kidney and liver damage and central nervous system excitation followed by depression. Haemolytic anaemia (a result of red-blood cell damage) has been described in acute human poisoning. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products.] Other symptoms of copper poisoning include lethargy, neurotoxicity, and increased blood pressure and respiratory rates. Coma and death have followed attempted suicides using solutions of copper sulfate. Copper is an essential element and most animal tissues have measurable amounts of copper associated with them. Humans have evolved mechanisms which maintain is availability whilst limiting its toxicity (homeostasis). Copper is initially bound in the body to a blood-borne protein, serum albumin and thereafter is more firmly bound to another protein, alpha-ceruloplasmin. Such binding effectively 'inactivates' the copper, thus reducing its potential to produce toxic damage. In healthy individuals, bound copper can reach relatively high levels without producing adverse health effects. Excretion in the bile represents the major pathway by which copper is removed from the body when it reaches potentially toxic levels. Copper may also be stored in the liver and bone marrow where it is bound to another protein, metallothionein. A combination of binding and excretion ensures that the body is able to tolerate relatively high loadings of copper.
Skin Contact	Strong evidence exists that exposure to the material may produce serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact. Skin contact is not thought to produce harmful health effects (as classified under EC Directives using animal models). Systemic harm, however, has been identified following exposure of animals by at least one other route and the material may still produce health damage following entry through wounds, lesions or abrasions. Good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting. Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. Spray mist may produce discomfort Exposure to copper, by skin, has come from its use in pigments, ointments, ornaments, jewellery, dental amalgams and IUDs and as an antifungal agent and an algicide. Although copper algicides are used in the treatment of water in swimming pools and reservoirs, there are no reports of toxicity from these applications. Reports of allergic contact dermatitis following contact with copper and its salts have appeared in the literature, however the exposure concentrations leading to any effect have been poorly characterised. In one study, patch testing of 1190 eczema patients found that only 13 (1.1%) cross-reacted with 2% copper sulfate in petrolatum. The investigators warned, however, that the possibility of contamination with nickel (an established contact allergen) might have been the cause of the reaction. Copper salts often produce an itching eczema in contact with skin. This is, likely, of a non-allergic nature. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected. Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.
Eye	Copper salts, in contact with the eye, may produce conjunctivitis or even ulceration and turbidity of the cornea. 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 windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur. The liquid may produce eye discomfort and is capable of causing temporary impairment of vision and/or transient eye inflammation, ulceration
Chronic	Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.

Continued...

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.
There is sufficient evidence to provide a strong presumption that human exposure to the material may produce heritable genetic damage.
There is sufficient evidence to provide a strong presumption that human exposure to the material may result in the development of heritable genetic damage, generally on the basis of
- appropriate animal studies,
- other relevant information
Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.

Chronic copper poisoning is rarely recognised in man although in one instance, at least, symptoms more commonly associated with exposures to mercury, namely infantile acrodynia (pink disease), have been described. Tissue damage of mucous membranes may follow chronic dust exposure. A hazardous situation is exposure of a worker with the rare hereditary condition (Wilson's disease or hereditary hepatolenticular degeneration) to copper exposure which may cause liver, kidney, CNS, bone and sight damage and is potentially lethal. Haemolytic anaemia (a result of red-blood cell damage) is common in cows and sheep poisoned by copper derivatives. Overdosing of copper feed supplements has resulted in pigmentary cirrhosis of the liver. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products]
Workers exposed to 700 ppm acetone for 3 hours/day for 7-15 years showed inflammation of the respiratory tract, stomach and duodenum, attacks of giddiness and loss of strength. Exposure to acetone may enhance liver toxicity of chlorinated solvents.

843AR Super Shield Silver Coated Copper Conductive Paint (Aerosol)

TOXICITY	IRRITATION
Not Available	Not Available

acetone

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >11.899 mg/kg[1]	Eye (human): 500 ppm - irritant
Inhalation(Mouse) LC50; 44 mg/L4[2]	Eye (rabbit): 20mg/24hr -moderate
Oral(Rat) LD50; 2.785 mg/kg[1]	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]

propane

TOXICITY	IRRITATION
Inhalation(Rat) LC50; >13023 ppm4[1]	Not Available

n-butyl acetate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >14100 mg/kg[2]	Eye (human): 300 mg
Inhalation(Rat) LC50; 0.74 mg/l4[2]	Eye (rabbit): 20 mg (open)-SEVERE
Oral(Rat) LD50; 13.864 mg/kg[1]	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]

dimethyl carbonate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
Inhalation(Rat) LC50; >5.36 mg/l4[1]	Skin: no adverse effect observed (not irritating)[1]
Oral(Rat) LD50; >5000 mg/kg[1]	

copper

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
Inhalation(Rat) LC50; 0.733 mg/l4[1]	Skin: no adverse effect observed (not irritating)[1]
Oral(Mouse) LD50; 0.7 mg/kg[2]	

iso-butane

TOXICITY	IRRITATION
Inhalation(Rat) LC50; >13023 ppm4[1]	Not Available

amyl methyl ketone

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	Eye: adverse effect observed (irritating)[1]
Inhalation(Rat) LC50; >16.7 mg/l4[1]	Skin (rabbit): 14 mg/24h Mild
Oral(Mouse) LD50; 730 mg/kg[2]	Skin (rabbit): Primary Irritant
	Skin: adverse effect observed (irritating)[1]

Continued...

	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]
Oral(Rat) LD50; 5155 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]

silver	
TOXICITY	**IRRITATION**
dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
Inhalation(Rat) LC50; >5.16 mg/l4[1]	Skin: no adverse effect observed (not irritating)[1]
Oral(Rat) LD50; >2000 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*

843AR Super Shield Silver Coated Copper Conductive Paint (Aerosol)	Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production. 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.
PROPANE	No significant acute toxicological data identified in literature search.
N-BUTYL ACETATE	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
COPPER	WARNING: Inhalation of high concentrations of copper fume may cause 'metal fume fever', an acute industrial disease of short duration. Symptoms are tiredness, influenza like respiratory tract irritation with fever. for copper and its compounds (typically copper chloride): **Acute toxicity:** There are no reliable acute oral toxicity results available. In an acute dermal toxicity study (OECD TG 402), one group of 5 male rats and 5 groups of 5 female rats received doses of 1000, 1500 and 2000 mg/kg bw via dermal application for 24 hours. The LD50 values of copper monochloride were 2,000 mg/kg bw or greater for male (no deaths observed) and 1,224 mg/kg bw for female. Four females died at both 1500 and 2000 mg/kg bw, and one at 1,000 mg/kg bw. Symptom of the hardness of skin, an exudation of hardness site, the formation of scar and reddish changes were observed on application sites in all treated animals. Skin inflammation and injury were also noted. In addition, a reddish or black urine was observed in females at 2,000, 1,500 and 1,000 mg/kg bw. Female rats appeared to be more sensitive than male based on mortality and clinical signs. No reliable skin/eye irritation studies were available. The acute dermal study with copper monochloride suggests that it has a potential to cause skin irritation. **Repeat dose toxicity:** In repeated dose toxicity study performed according to OECD TG 422, copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39 - 51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL value was 5 and 1.3 mg/kg bw/day for male and female rats, respectively. No deaths were observed in male rats. One treatment-related death was observed in female rats in the high dose group. Erythropoietic toxicity (anaemia) was seen in both sexes at the 80 mg/kg bw/day. The frequency of squamous cell hyperplasia of the forestomach was increased in a dose-dependent manner in male and female rats at all treatment groups, and was statistically significant in males at doses of =20 mg/kg bw/day and in females at doses of =5 mg/kg bw/day doses. The observed effects are considered to be local, non-systemic effect on the forestomach which result from oral (gavage) administration of copper monochloride. **Genotoxicity:** An in vitro genotoxicity study with copper monochloride showed negative results in a bacterial reverse mutation test with Salmonella typhimurium strains (TA 98, TA 100, TA 1535, and TA 1537) with and without S9 mix at concentrations of up to 1,000 ug/plate. An in vitro test for chromosome aberration in Chinese hamster lung (CHL) cells showed that copper monochloride induced structural and numerical aberrations at the concentration of 50, 70 and 100 ug/mL without S9 mix. In the presence of the metabolic activation system, significant increases of structural aberrations were observed at 50 and 70 ug/mL and significant increases of numerical aberrations were observed at 70 ug/mL. In an in vivo mammalian erythrocyte micronucleus assay, all animals dosed (15 - 60 mg/kg bw) with copper monochloride exhibited similar PCE/(PCE+NCE) ratios and MNPCE frequencies compared to those of the negative control animals. Therefore copper monochloride is not an in vivo mutagen. **Carcinogenicity:** there was insufficient information to evaluate the carcinogenic activity of copper monochloride. Reproductive and developmental toxicity: In the combined repeated dose toxicity study with the reproduction/developmental toxicity screening test (OECD TG 422), copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39-51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL of copper monochloride for fertility toxicity was 80 mg/kg bw/day for the parental animals. No treatment-related effects were observed on the reproductive organs and the fertility parameters assessed. For developmental toxicity the NOAEL was 20 mg/kg bw/day. Three of 120 pups appeared to have icterus at birth; 4 of 120 pups appeared runted at the highest dose tested (80 mg/kg bw/day).
PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER	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

Continued...

	for propylene glycol ethers (PGEs): Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM). Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids. Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects). This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product. Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body. As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces. As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m3 for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m3. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m3), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating None are skin sensitisers. In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested). Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m3 (600 ppm) for PnB and 2,010 mg/m3 (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m3 (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m3 (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members. One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m3) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m3). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m3), with decreased body weights occurring at 3000 ppm (11058 mg/m3). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. in a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health. In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity. The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic *in vivo*. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice. 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]
843AR Super Shield Silver Coated Copper Conductive Paint (Aerosol) & N-BUTYL ACETATE	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 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 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. The JEFCA 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 **Internationl Program on Chemical Safety: the Joint FAO/WHO Expert Committee on Food Additives (JECFA)** **Esters of Aliphatic acyclic primary alcohols with aliphatic linear saturated carboxylic acids.; 1998**
843AR Super Shield Silver Coated Copper Conductive Paint (Aerosol) & ACETONE	for acetone: The acute toxicity of acetone is low. Acetone is not a skin irritant or sensitiser 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

Continued...

	rats and mice. 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. 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.
ACETONE & AMYL METHYL KETONE	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.

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

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

SECTION 12 Ecological information

12.1. Toxicity

843AR Super Shield Silver Coated Copper Conductive Paint (Aerosol)

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
LC50	96	Fish	13.303mg/L	4
NOEC(ECx)	12	Fish	0.001mg/L	4
EC50	48	Crustacea	6098.4mg/L	5
EC50	96	Algae or other aquatic plants	9.87327.684mg/l	4

propane

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

n-butyl acetate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48	Crustacea	32mg/l	1
LC50	96	Fish	18mg/l	2
EC50	72	Algae or other aquatic plants	246mg/l	2
EC50(ECx)	96	Fish	18mg/l	2

dimethyl carbonate

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	504	Crustacea	25mg/l	2
LC50	96	Fish	>=100mg/l	2
EC50	48	Crustacea	>74.16mg/l	2
EC50	72	Algae or other aquatic plants	>57.29mg/l	2
EC50	96	Algae or other aquatic plants	166.6211mg/l	2

copper

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	9	Crustacea	<0.001mg/L	4
LC50	96	Fish	<0.001mg/L	4
EC50	48	Crustacea	<0.001mg/L	4
EC50	72	Algae or other aquatic plants	<0.001mg/L	4
EC50	96	Algae or other aquatic plants	<0.001mg/L	4

iso-butane

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

Continued...

	LC50	96	Fish	24.11mg/l	2
	EC50	96	Algae or other aquatic plants	7.71mg/l	2

amyl methyl ketone

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	131mg/l	2
EC50	48	Crustacea	>90.1mg/l	2
NOEC(ECx)	72	Algae or other aquatic plants	42.68mg/l	2
EC50	72	Algae or other aquatic plants	75.5mg/l	2

propylene glycol monomethyl ether acetate, alpha-isomer

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	>100mg/l	2
EC50	48	Crustacea	373mg/l	2
NOEC(ECx)	336	Fish	47.5mg/l	2
EC50	72	Algae or other aquatic plants	>1000mg/l	2
EC50	96	Algae or other aquatic plants	>1000mg/l	2

silver

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	48	Crustacea	<0.001mg/L	4
LC50	96	Fish	<0.001mg/L	4
EC50	48	Crustacea	<0.001mg/L	4
EC50	72	Algae or other aquatic plants	11.89mg/l	2
EC50	96	Algae or other aquatic plants	0.002mg/L	4

Legend: *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*

Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
Metal-containing inorganic substances generally have negligible vapour pressure and are not expected to partition to air. Once released to surface waters and moist soils their fate depends on solubility and dissociation in water. Environmental processes (such as oxidation and the presence of acids or bases) may transform insoluble metals to more soluble ionic forms. Microbiological processes may also transform insoluble metals to more soluble forms. Such ionic species may bind to dissolved ligands or sorb to solid particles in aquatic or aqueous media. A significant proportion of dissolved/ sorbed metals will end up in sediments through the settling of suspended particles. The remaining metal ions can then be taken up by aquatic organisms.
When released to dry soil most metals will exhibit limited mobility and remain in the upper layer; some will leach locally into ground water and/ or surface water ecosystems when soaked by rain or melt ice. Environmental processes may also be important in changing solubilities.
Even though many metals show few toxic effects at physiological pHs, transformation may introduce new or magnified effects.
A metal ion is considered infinitely persistent because it cannot degrade further.
The current state of science does not allow for an unambiguous interpretation of various measures of bioaccumulation.
The counter-ion may also create health and environmental concerns once isolated from the metal. Under normal physiological conditions the counter-ion may be essentially insoluble and may not be bioavailable.
Environmental processes may enhance bioavailability.
Copper is unlikely to accumulate in the atmosphere due to a short residence time for airborne copper aerosols. Airborne coppers, however, may be transported over large distances.
Copper accumulates significantly in the food chain.
Drinking Water Standards:
3000 ug/l (UK max)
2000 ug/l (WHO provisional Guideline)
1000 ug/l (WHO level where individuals complain)
Soil Guidelines: Dutch Criteria
36 mg/kg (target)
190 mg/kg (intervention)
Air Quality Standards: no data available.
The toxic effect of copper in the aquatic biota depends on the bio-availability of copper in water which, in turn, depends on its physico-chemical form (ie.speciation). Bioavailability is decreased by complexation and adsorption of copper by natural organic matter, iron and manganese hydrated oxides, and chelating agents excreted by algae and other aquatic organisms. Toxicity is also affected by pH and hardness. Total copper is rarely useful as a predictor of toxicity. In natural sea water, more than 98% of copper is organically bound and in river waters a high percentage is often organically bound, but the actual percentage depends on the river water and its pH.
Copper exhibits significant toxicity in some aquatic organisms. Some algal species are very sensitive to copper with EC50 (96 hour) values as low as 47 ug/litre dissolved copper whilst for other algal species EC50 values of up to 481 ug/litre have been reported. However many of the reportedly high EC50 values may arise in experiments conducted with a culture media containing copper-complexing agents such as silicate, iron, manganese and EDTA which reduce bioavailability.

Toxic effects arising following exposure by aquatic species to copper are typically:

Algae EC50 (96 h)	Daphnia magna LC50 (48-96 h)	Amphipods LC50 (48-96 h)	Gastropods LC50 (48-96 h)	Crab larvae LC50 (48-96 h)
47-481 *	7-54 *	37-183 *	58-112 *	50-100 *

* ug/litre

Exposure to concentrations ranging from one to a few hundred micrograms per litre has led to sublethal effects and effects on long-term survival. For high bioavailability waters, effect concentrations for several sensitive species may be below 10 ug Cu/litre.

In fish, the acute lethal concentration of copper ranges from a few ug/litre to several mg/litre, depending both on test species and exposure conditions. Where the value is less than 50 ug Cu/litre, test waters generally have a low dissolved organic carbon (DOC) level, low hardness and neutral to slightly acidic pH. Exposure to concentrations ranging from one to a few hundred micrograms per litre has led to sublethal effects and effects on long-term survival. Lower effect concentrations are generally associated with test waters of high bioavailability.

Continued...

In summary:

Responses expected for high concentration ranges of copper *

Total dissolved Cu concentration range (ug/litre)	Effects of high availability in water
1-10	Significant effects are expected for diatoms and sensitive invertebrates, notably cladocerans. Effects on fish could be significant in freshwaters with low pH and hardness.
10-100	Significant effects are expected on various species of microalgae, some species of macroalgae, and a range of invertebrates, including crustaceans, gastropods and sea urchins. Survival of sensitive fish will be affected and a variety of fish show sublethal effects.
100-1000	Most taxonomic groups of macroalgae and invertebrates will be severely affected. Lethal levels for most fish species will be reached.
>1000	Lethal concentrations for most tolerant organisms are reached.

* Sites chosen have moderate to high bioavailability similar to water used in most toxicity tests.

In soil, copper levels are raised by application of fertiliser, fungicides, from deposition of highway dusts and from urban, mining and industrial sources. Generally, vegetation rooted in soils reflects the soil copper levels in its foliage. This is dependent upon the bioavailability of copper and the physiological requirements of species concerned.

Typical foliar levels of copper are:

Uncontaminated soils (0.3-250 mg/kg)	Contaminated soils (150-450 mg/kg)	Mining/smelting soils
6.1-25 mg/kg	80 mg/kg	300 mg/kg

Plants rarely show symptoms of toxicity or of adverse growth effects at normal soil concentrations of copper. Crops are often more sensitive to copper than the native flora, so protection levels for agricultural crops range from 25 mg Cu/kg to several hundred mg/kg, depending on country. Chronic and or acute effects on sensitive species occur at copper levels occurring in some soils as a result of human activities such as copper fertiliser addition, and addition of sludge.

When soil levels exceed 150 mg Cu/kg, native and agricultural species show chronic effects. Soils in the range 500-1000 mg Cu/kg act in a strongly selective fashion allowing the survival of only copper-tolerant species and strains. At 2000 Cu mg/kg most species cannot survive. By 3500 mg Cu/kg areas are largely devoid of vegetation cover. The organic content of the soil appears to be a key factor affecting the bioavailability of copper.

On normal forest soils, non-rooted plants such as mosses and lichens show higher copper concentrations. The fruiting bodies and mycorrhizal sheaths of soil fungi associated with higher plants in forests often accumulate copper to much higher levels than plants at the same site. International Programme on Chemical Safety (IPCS): Environmental Health Criteria 200
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 substrateThe higher molecular weight ketones do no 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 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 falls between propane 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 5x105 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 7.07x10-1 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 1.15x10-12 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: 3.67E-05
BOD 5: 0.31-1.76,46-55%
COD: 1.12-2.07
ThOD: 2.2

Continued...

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 (*Ephestia 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: 3.20E-04
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 2.8x10-4 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 2.8x10-4 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) 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)
propane	LOW	LOW
n-butyl acetate	LOW	LOW
dimethyl carbonate	HIGH	HIGH
iso-butane	HIGH	HIGH
amyl methyl ketone	LOW	LOW
propylene glycol monomethyl ether acetate, alpha-isomer	LOW	LOW

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
acetone	LOW (BCF = 0.69)
propane	LOW (LogKOW = 2.36)
n-butyl acetate	LOW (BCF = 14)
dimethyl carbonate	LOW (LogKOW = 0.2336)
iso-butane	LOW (BCF = 1.97)

Continued...

Ingredient	Bioaccumulation
amyl methyl ketone	LOW (LogKOW = 1.98)
propylene glycol monomethyl ether acetate, alpha-isomer	LOW (LogKOW = 0.56)

12.4. Mobility in soil

Ingredient	Mobility
acetone	HIGH (KOC = 1.981)
propane	LOW (KOC = 23.74)
n-butyl acetate	LOW (KOC = 20.86)
dimethyl carbonate	LOW (KOC = 8.254)
iso-butane	LOW (KOC = 35.04)
amyl methyl ketone	LOW (KOC = 24.01)
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 Applicable	Not Applicable	Not Applicable
PBT Criteria fulfilled?	Not Applicable	Not Applicable	Not Applicable

12.6. Other adverse effects

No data available

SECTION 13 Disposal considerations

13.1. Waste treatment methods

Product / Packaging disposal	▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ 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

Land transport (ADR-RID)

14.1. **UN number**	1950
14.2. **UN proper shipping name**	AEROSOLS
14.3. **Transport hazard class(es)**	Class: 2.1 Subrisk: Not Applicable
14.4. **Packing group**	Not Applicable
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	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)

Continued...

Air transport (ICAO-IATA / DGR)

14.1. **UN number**	1950
14.2. **UN proper shipping name**	Aerosols, flammable
14.3. **Transport hazard class(es)**	ICAO/IATA Class: 2.1 ICAO / IATA Subrisk: Not Applicable ERG Code: 10L
14.4. **Packing group**	Not Applicable
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	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)**	IMDG Class: 2.1 IMDG Subrisk: Not Applicable
14.4. **Packing group**	Not Applicable
14.5. **Environmental hazard**	Marine Pollutant
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**	Environmentally hazardous
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
propane	Not Available
n-butyl acetate	Not Available
dimethyl carbonate	Not Available
copper	Not Available
iso-butane	Not Available
amyl methyl ketone	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available
silver	Not Available

Continued...

14.9. Transport in bulk in accordance with the ICG Code

Product name	Ship Type
acetone	Not Available
propane	Not Available
n-butyl acetate	Not Available
dimethyl carbonate	Not Available
copper	Not Available
iso-butane	Not Available
amyl methyl ketone	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available
silver	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
UK Workplace Exposure Limits (WELs)

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

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
UK Workplace Exposure Limits (WELs)

dimethyl carbonate 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

copper is found on the following regulatory lists

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
UK Workplace Exposure Limits (WELs)

iso-butane 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 1A (Table 3.1)/category 1 (Table 3.2)
EU REACH Regulation (EC) No 1907/2006 - Annex XVII (Appendix 4) Mutagens: category 1B (Table 3.1)/category 2 (Table 3.2)
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

amyl methyl ketone 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
UK Workplace Exposure Limits (WELs)

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
UK Workplace Exposure Limits (WELs)

silver is found on the following regulatory lists

Continued...

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances	International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
Europe EC Inventory	UK Workplace Exposure Limits (WELs)
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

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (acetone; propane; n-butyl acetate; dimethyl carbonate; copper; iso-butane; amyl methyl ketone; propylene glycol monomethyl ether acetate, alpha-isomer; silver)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (copper; silver)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - ARIPS	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 and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 Other information

Revision Date	12/03/2021
Initial Date	23/11/2020

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	Harmful if swallowed.
H332	Harmful if inhaled.

SDS Version Summary

Version	Issue Date	Sections Updated
3.9.1.1.1	12/03/2021	Classification, Exposure Standard, Ingredients, Physical Properties, Storage (storage incompatibility)

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

Continued...

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

Reason for Change

A-2.00 - Update to the classification.