Revive 476S Solvent

On-Crete Australia Pty Ltd

Chemwatch Hazard Alert Code: **3**

Version No: **1.3**
Safety Data Sheet according to WHS and ADG requirements

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L.GHS.AUS.EN

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

Product Identifier

Product name	Revive 476S Solvent
Synonyms	Not Available
Proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound) (contains mibk and mek and xylene (mixed isomers) and ipa)
Other means of identification	Not Available

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

Relevant identified uses	Solvent wash reactivation of old delaminating solvent based sealers

Details of the manufacturer/importer

Registered company name	On-Crete Australia Pty Ltd
Telephone	+61 7 5593 6884
Fax	+61 7 5593 6885
Website	www.on-crete.com.au
Email	info@on-crete.com.au

Emergency telephone number

Association / Organisation	Poisons Information Centre
Emergency telephone numbers	13 11 26
Other emergency telephone numbers	Not Available

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

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

CHEMWATCH HAZARD RATINGS

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

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

Poisons Schedule	Not Applicable

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GHS Classification [1]	Acute Toxicity (Inhalation) Category 4, Aspiration Hazard Category 1, Flammable Liquid Category 2, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2, Emit Flammable Gases with Water Category 2
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HSIS ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI*

Label elements

GHS label elements	
SIGNAL WORD	**DANGER**

Hazard statement(s)

H225	Highly flammable liquid and vapour
H261	In contact with water releases flammable gas
H304	May be fatal if swallowed and enters airways
H315	Causes skin irritation
H319	Causes serious eye irritation
H332	Harmful if inhaled

Precautionary statement(s) Prevention

P231+P232	Handle under inert gas. Protect from moisture.
P233	Keep container tightly closed.
P271	Use only outdoors or in a well-ventilated area.
P261	Avoid breathing dust/fume/gas/mist/vapours/spray.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P223	Do not allow contact with water.
P240	Ground/bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use only non-sparking tools.
P243	Take precautionary measures against static discharge.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider
P331	Do NOT induce vomiting.
P335+P334	Brush off loose particles from skin. Immerse in cool water/wrap in wet bandages.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam for extinction.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P337+P313	If eye irritation persists: Get medical advice/attention.
P302+P352	IF ON SKIN: Wash with plenty of water and soap
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water/shower.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

P403+P235	Store in a well-ventilated place. Keep cool.
P405	Store locked up.

Precautionary statement(s) Disposal

Continued...

P501	Dispose of contents/container to authorised chemical landfill or if organic to high temperature incineration

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
108-10-1	10-30	methyl isobutyl ketone
78-93-3	10-30	methyl ethyl ketone
1330-20-7	10-30	xylene
64742-95-6.	10-30	aromatic 150
111-76-2	10-30	ethylene glycol monobutyl ether
67-63-0	<10	isopropanol
2530-83-8	<10	gamma-glycidoxypropyltrimethoxysilane

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: • Wash out immediately with fresh running water. • Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. • Seek medical attention without delay; if pain persists or recurs seek medical attention. Removal of • contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: • Immediately remove all contaminated clothing, including footwear. • Flush skin and hair with running water (and soap if available). • Seek medical attention in event of irritation.
Inhalation	• If fumes or combustion products are inhaled remove from contaminated area. Lay • patient down. Keep warm and rested. • Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. • Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. • Transport to hospital, or doctor, without delay.
Ingestion	• If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus. • If swallowed do **NOT** induce vomiting. • If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. • Observe the patient carefully. • Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. Give • water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. • Seek medical advice. • Avoid giving milk or oils. • Avoid giving alcohol.

Indication of any immediate medical attention and special treatment needed

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.

Followed acute or short term repeated exposures to ethylene glycol monoalkyl ethers and their acetates:
- Hepatic metabolism produces ethylene glycol as a metabolite.
- Clinical presentation, following severe intoxication, resembles that of ethylene glycol exposures.
- Monitoring the urinary excretion of the alkoxyacetic acid metabolites may be a useful indication of exposure.

[Ellenhorn and Barceloux: Medical Toxicology]

For acute or short term repeated exposures to ethylene glycol:
- Early treatment of ingestion is important. Ensure emesis is satisfactory.
- Test and correct for metabolic acidosis and hypocalcaemia. Apply
- sustained diuresis when possible with hypertonic mannitol.

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- Evaluate renal status and begin haemodialysis if indicated. [I.L.O]
- Rapid absorption is an indication that emesis or lavage is effective only in the first few hours. Cathartics and charcoal are generally not effective.
- Correct acidosis, fluid/electrolyte balance and respiratory depression in the usual manner. Systemic acidosis (below 7.2) can be treated with intravenous sodium bicarbonate solution.
- Ethanol therapy prolongs the half-life of ethylene glycol and reduces the formation of toxic metabolites.
- Pyridoxine and thiamine are cofactors for ethylene glycol metabolism and should be given (50 to 100 mg respectively) intramuscularly, four times per day for 2 days.
- Magnesium is also a cofactor and should be replenished. The status of 4-methylpyrazole, in the treatment regime, is still uncertain. For clearance of the material and its metabolites, haemodialysis is much superior to peritoneal dialysis.

[Ellenhorn and Barceloux: Medical Toxicology]

It has been suggested that there is a need for establishing a new biological exposure limit before a workshift that is clearly below 100 mmol ethoxy-acetic acids per mole creatinine in morning urine of people occupationally exposed to ethylene glycol ethers. This arises from the finding that an increase in urinary stones may be associated with such exposures.
Laitinen J., et al: Occupational & Environmental Medicine 1996; 53, 595-600 for
simple ketones:

BASIC TREATMENT

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

ADVANCED TREATMENT

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

EMERGENCY DEPARTMENT

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

BRONSTEIN, A.C. and CURRANCE, P.L.
EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

For acute and short term repeated exposures to methanol:

- Toxicity results from accumulation of formaldehyde/formic acid.
- Clinical signs are usually limited to CNS, eyes and GI tract Severe metabolic acidosis may produce dyspnea and profound systemic effects which may become intractable. All symptomatic patients should have arterial pH measured. Evaluate airway, breathing and circulation.
- Stabilise obtunded patients by giving naloxone, glucose and thiamine.
- Decontaminate with Ipecac or lavage for patients presenting 2 hours post-ingestion. Charcoal does not absorb well; the usefulness of cathartic is not established.
- Forced diuresis is not effective; haemodialysis is recommended where peak methanol levels exceed 50 mg/dL (this correlates with serum bicarbonate levels below 18 mEq/L).
- Ethanol, maintained at levels between 100 and 150 mg/dL, inhibits formation of toxic metabolites and may be indicated when peak methanol levels exceed 20 mg/dL. An intravenous solution of ethanol in D5W is optimal.
- Folate, as leucovorin, may increase the oxidative removal of formic acid. 4-methylpyrazole may be an effective adjunct in the treatment. 8.Phenytoin may be preferable to diazepam for controlling seizure.

[Ellenhorn Barceloux: Medical Toxicology]

BIOLOGICAL EXPOSURE INDEX - BEI

Determinant	Index	Sampling Time	Comment
1. Methanol in urine	15 mg/l	End of shift	B, NS
2. Formic acid in urine	80 mg/gm creatinine	Before the shift at end of workweek	B, NS

B: Background levels occur in specimens collected from subjects **NOT** exposed.

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NS: Non-specific determinant - observed following exposure to other materials. For acute or short term repeated exposures to xylene:

- Gastro-intestinal absorption is significant with ingestions. For ingestions exceeding 1-2 ml (xylene)/kg, intubation and lavage with cuffed endotracheal tube is recommended. The use of charcoal and cathartics is equivocal.
- Pulmonary absorption is rapid with about 60-65% retained at rest.
- Primary threat to life from ingestion and/or inhalation, is respiratory failure.
- Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO2 < 50 mm Hg or pCO2 > 50 mm Hg) should be intubated.
- Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial injury has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
- A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
- Epinephrine (adrenalin) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.

BIOLOGICAL EXPOSURE INDEX - BEI

These represent the determinants observed in specimens collected from a healthy worker exposed at the Exposure Standard (ES or TLV):

Determinant	Index	Sampling Time	Comments
Methylhippu-ric acids in urine	1.5 gm/gm creatinine	End of shift	
	2 mg/min	Last 4 hrs of shift	

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

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

Special hazards arising from the substrate or mixture

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

Advice for firefighters

Fire Fighting	‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ May be violently or explosively reactive. ‣ Wear breathing apparatus plus protective gloves in the event of a fire. ‣ Prevent, by any means available, spillage from entering drains or water course. ‣ Consider evacuation (or protect in place). ‣ Fight fire from a safe distance, with adequate cover. ‣ If safe, switch off electrical equipment until vapour fire hazard removed. ‣ Use water delivered as a fine spray to control the fire and cool adjacent area. ‣ Avoid spraying water onto liquid pools. ‣ **Do not** approach containers suspected to be hot. ‣ Cool fire exposed containers with water spray from a protected location. ‣ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	‣ Liquid and vapour are highly flammable. ‣ Severe fire hazard when exposed to heat, flame and/or oxidisers. ‣ Vapour may travel a considerable distance to source of ignition. ‣ Heating may cause expansion or decomposition leading to violent rupture of containers. ‣ On combustion, may emit toxic fumes of carbon monoxide (CO). Combustion products include:, carbon dioxide (CO_2), formaldehyde, sulfur oxides (SOx), other pyrolysis products typical of burning organic material **WARNING**: Long standing in contact with air and light may result in the formation of potentially explosive peroxides.

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Minor Spills	‣ Remove all ignition sources. ‣ Clean up all spills immediately. ‣ Avoid breathing vapours and contact with skin and eyes. ‣ Control personal contact with the substance, by using protective equipment. ‣ Contain and absorb small quantities with vermiculite or other absorbent material. ‣ Wipe up.

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	‣ Collect residues in a flammable waste container.
Major Spills	Chemical Class: ketones For release onto land: recommended sorbents listed in order of priority.

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS
LAND SPILL - SMALL				
cross-linked polymer - particulate	1	shovel	shovel	R, W, SS
cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT
sorbent clay - particulate	2	shovel	shovel	R,I, P
wood fiber - pillow	3	throw	pitchfork	R, P, DGC, RT
treated wood fiber - pillow	3	throw	pitchfork	DGC, RT
foamed glass - pillow	4	throw	pitchfork	R, P, DGC, RT
LAND SPILL - MEDIUM				
cross-linked polymer - particulate	1	blower	skiploader	R,W, SS
cross-linked polymer - pillow	2	throw	skiploader	R, DGC, RT
sorbent clay - particulate	3	blower	skiploader	R, I, P
polypropylene - particulate	3	blower	skiploader	R, SS, DGC
expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC
polypropylene - mat	4	throw	skiploader	DGC, RT

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

- 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 course.
- Consider evacuation (or protect in place).
- 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.
- Contain spill with sand, earth or vermiculite.
- Use only spark-free shovels and explosion proof equipment.
- Collect recoverable product into labelled containers for recycling.
- Absorb remaining product with sand, earth or vermiculite.
- Collect solid residues and seal in labelled drums for disposal.
- Wash area and prevent runoff into drains.
- If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

	‣ Containers, even those that have been emptied, may contain explosive vapours. Do ‣ NOT cut, drill, grind, weld or perform similar operations on or near containers. ‣ **DO NOT** allow clothing wet with material to stay in contact with skin
adjacent to the ether link are thought to be relatively safe	The tendency of many ethers to form explosive peroxides is well documented. Ethers lacking non-methyl hydrogen atoms **Safe handling** ‣ **DO NOT** concentrate by evaporation, or evaporate extracts to dryness, as residues may contain explosive peroxides with DETONATION potential. ‣ Any static discharge is also a source of hazard. ‣ Before any distillation process remove trace peroxides by shaking with excess 5% aqueous ferrous sulfate solution or by

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	▸ percolation through a column of activated alumina. ▸ Distillation results in uninhibited ether distillate with considerably increased hazard because of risk of peroxide formation on storage. ▸ Add inhibitor to any distillate as required. ▸ When solvents have been freed from peroxides by percolation through columns of activated alumina, the absorbed peroxides must promptly be desorbed by treatment with polar solvents such as methanol or water, which should then be disposed of safely. ▸ Electrostatic discharge may be generated during pumping - this may result in fire. ▸ Ensure electrical continuity by bonding and grounding (earthing) all equipment. ▸ Restrict line velocity during pumping in order to avoid generation of electrostatic discharge (<=1 m/sec until fill pipe submerged to twice its diameter, then <= 7 m/sec). ▸ Avoid splash filling. ▸ Do NOT use compressed air for filling discharging or handling operations. The substance accumulates peroxides which may become hazardous only if it evaporates or is distilled or otherwise treated to concentrate the peroxides. The substance may concentrate around the container opening for example. Purchases of peroxidisable chemicals should be restricted to ensure that the chemical is used completely before it can become peroxidised. ▸ A responsible person should maintain an inventory of peroxidisable chemicals or annotate the general chemical inventory to indicate which chemicals are subject to peroxidation. An expiration date should be determined. The chemical should either be treated to remove peroxides or disposed of before this date. ▸ The person or laboratory receiving the chemical should record a receipt date on the bottle. The individual opening the container should add an opening date. ▸ Unopened containers received from the supplier should be safe to store for 18 months. ▸ Opened containers should not be stored for more than 12 months. ▸ 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, heat or ignition sources. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Vapour may ignite on pumping or pouring due to static electricity. ▸ **DO NOT** use plastic buckets. ▸ Earth and secure metal containers when dispensing or pouring product. ▸ Use spark-free tools when handling. ▸ Avoid contact with incompatible materials. ▸ Keep containers securely sealed. ▸ 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 MSDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
Other information	▸ Store in original containers in approved flame-proof area. ▸ No smoking, naked lights, heat or ignition sources. ▸ **DO NOT** store in pits, depressions, basements or areas where vapours may be trapped. ▸ Keep containers securely sealed. ▸ Store away from incompatible materials in a cool, dry well ventilated area. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this MSDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Packing as supplied by manufacturer. ▸ Plastic containers may only be used if approved for flammable liquid. ▸ Check that containers are clearly labelled and free from leaks. ▸ For low viscosity materials (i) : Drums and jerry cans must be of the non-removable head type. (ii) : 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) ▸ For manufactured product having a viscosity of at least 250 cSt. (23 deg. C) ▸ Manufactured product that requires stirring before use and having a viscosity of at least 20 cSt (25 deg. C): (i) Removable head packaging; (ii) Cans with friction closures and (iii) 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 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.
Storage incompatibility	Ethylene glycol monobutyl ether (2-butoxyethanol) and its acetate: ▸ May form unstable peroxides in storage ▸ is incompatible with oxidisers, permanganates, peroxides, ammonium persulfate, bromine dioxide, nitrates, strong acids, sulfuric acid, nitric acid, perchloric acid

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Methyl ethyl ketone:
- reacts violently with strong oxidisers, aldehydes, nitric acid, perchloric acid, potassium tert-butoxide, oleum
- is incompatible with inorganic acids, aliphatic amines, ammonia, caustics, isocyanates, pyridines, chlorosulfonic aid
- forms unstable peroxides in storage, or on contact with propanol or hydrogen peroxide
- attacks some plastics
- may generate electrostatic charges, due to low conductivity, on flow or agitation Methyl

isobutyl ketone (MIBK)
- forms unstable and explosive peroxides on contact with air and/ or when in contact with hydrogen peroxide
- reacts violently with strong oxidisers, aldehydes, aliphatic amines, nitric acid, perchloric acid, potassium tert-butoxide, strong acids, reducing agents
- dissolves some plastics, resins and rubber

Xylenes:
- may ignite or explode in contact with strong oxidisers, 1,3-dichloro-5,5-dimethylhydantoin, uranium fluoride
- attack some plastics, rubber and coatings
- may generate electrostatic charges on flow or agitation due to low conductivity.
- Vigorous reactions, sometimes amounting to explosions, can result from the contact between aromatic rings and strong oxidising agents.
- Aromatics can react exothermically with bases and with diazo compounds. For

alkyl aromatics:
The alkyl side chain of aromatic rings can undergo oxidation by several mechanisms. The most common and dominant one is the attack by oxidation at benzylic carbon as the intermediate formed is stabilised by resonance structure of the ring.
- Following reaction with oxygen and under the influence of sunlight, a hydroperoxide at the alpha-position to the aromatic ring, is the primary oxidation product formed (provided a hydrogen atom is initially available at this position) - this product is often short-lived but may be stable dependent on the nature of the aromatic substitution; a secondary C-H bond is more easily attacked than a primary C-H bond whilst a tertiary C-H bond is even more susceptible to attack by oxygen
- Monoalkylbenzenes may subsequently form monocarboxylic acids; alkyl naphthalenes mainly produce the corresponding naphthalene carboxylic acids.
- Oxidation in the presence of transition metal salts not only accelerates but also selectively decomposes the hydroperoxides.
- Hock-rearrangement by the influence of strong acids converts the hydroperoxides to hemiacetals. Peresters formed from the hydroperoxides undergo Criegee rearrangement easily.
- Alkali metals accelerate the oxidation while CO2 as co-oxidant enhances the selectivity.
- Microwave conditions give improved yields of the oxidation products.
- Photo-oxidation products may occur following reaction with hydroxyl radicals and NOx - these may be components of photochemical smogs.

Oxidation of Alkylaromatics: T.S.S Rao and Shubhra Awasthi: E-Journal of Chemistry Vol 4, No. 1, pp 1-13 January 2007
- Contact with water liberates highly flammable gases

Epoxides:
- are highly reactive with acids, bases, and oxidising and reducing agents.
- react, possibly violently, with anhydrous metal chlorides, ammonia, amines and group 1 metals.
- may polymerise in the presence of peroxides or heat - polymerisation may be violent
- may react, possibly violently, with water in the presence of acids and other catalysts. 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).

+	X	X	X	X	+

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

PACKAGE MATERIAL INCOMPATIBILITIES

Not Available

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Continued...

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	methyl isobutyl ketone	Methyl isobutyl ketone	205 mg/m3 / 50 ppm	307 mg/m3 / 75 ppm	Not Available	Not Available
Australia Exposure Standards	methyl ethyl ketone	Methyl ethyl ketone (MEK)	445 mg/m3 / 150 ppm	890 mg/m3 / 300 ppm	Not Available	Not Available
Australia Exposure Standards	xylene	Xylene (o-, m-, p-isomers)	350 mg/m3 / 80 ppm	655 mg/m3 / 150 ppm	Not Available	Not Available
Australia Exposure Standards	ethylene glycol monobutyl ether	2-Butoxyethanol	96.9 mg/m3 / 20 ppm	242 mg/m3 / 50 ppm	Not Available	Sk
Australia Exposure Standards	isopropanol	Isopropyl alcohol	983 mg/m3 / 400 ppm	1230 mg/m3 / 500 ppm	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
methyl isobutyl ketone	Methyl isobutyl ketone; (Hexone)	75 ppm	75 ppm	3000 ppm
methyl ethyl ketone	Butanone, 2-; (Methyl ethyl ketone; MEK)	Not Available	Not Available	Not Available
xylene	Xylenes	Not Available	Not Available	Not Available
ethylene glycol monobutyl ether	Butoxyethanol, 2-; (Glycol ether EB)	20 ppm	20 ppm	700 ppm
isopropanol	Isopropyl alcohol	400 ppm	400 ppm	12000 ppm
gamma-glycidoxypropyltrimethoxysilane	Glycidoxypropyltrimethoxysilane; (3-(2,3-Epoxypropoxy) propyltrimethoxysilane)	1.7 mg/m3	19 mg/m3	210 mg/m3

Ingredient	Original IDLH	Revised IDLH
methyl isobutyl ketone	3,000 ppm	500 ppm
methyl ethyl ketone	3,000 ppm	3,000 [Unch] ppm
xylene	1,000 ppm	900 ppm
aromatic hydrocarbons, C9-11	Not Available	Not Available
ethylene glycol monobutyl ether	700 ppm	700 [Unch] ppm
isopropanol	12,000 ppm	2,000 [LEL] ppm
gamma-glycidoxypropyltrimethoxysilane	Not Available	Not Available

MATERIAL DATA

for methyl isobutyl ketone (MIBK):
Unfatigued, odour recognition threshold (100% test panel) is 0.3 - 0.5 ppm.
Distinct odour at 15 ppm.
Odour is objectionable and vapours are irritating to eyes at 200 ppm.
NOTE: Detector tubes for methyl isobutyl ketone, measuring in excess of 50 ppm, are commercially available.
Exposure at or below the recommended TLV-TWA should provide sufficient protection against the potential irritant effects, headache and nausea, neurasthemic symptoms and other systemic toxicities (including liver and kidney damage) produced by MIBK.
The low odour threshold (1.64 mg/m3) and the irritant effects can provide warning of high concentrations. Exposure to levels of 10-410 mg/m3 (2.4-100 ppm) produced perceptible irritation of the eyes, nose, or throat, and 820 mg/m3 (200 ppm) produced discomfort. Symptoms, such as headache, nausea, or vertigo, also occurred at 10-410 mg/m3 (2.4-100 ppm). A 2-h exposure of up to 200 mg/m3 (50 ppm) did not produce any significant effects on a simple reaction-time task or a test of mental arithmetic.
Odour Safety Factor(OSF)
OSF=29 (METHYL ISOBUTYL KETONE)

for xylenes:
IDLH Level: 900 ppm
Odour Threshold Value: 20 ppm (detection), 40 ppm (recognition)
NOTE: Detector tubes for o-xylene, measuring in excess of 10 ppm, are available commercially. (m-xylene and p-xylene give almost the same response).

Xylene vapour is an irritant to the eyes, mucous membranes and skin and causes narcosis at high concentrations. Exposure to doses sufficiently high to produce intoxication and unconsciousness also produces transient liver and kidney toxicity. Neurologic impairment is NOT evident amongst volunteers inhaling up to 400 ppm though complaints of ocular and upper respiratory tract irritation occur at 200 ppm for 3 to 5 minutes.
Exposure to xylene at or below the recommended TLV-TWA and STEL is thought to minimise the risk of irritant effects and to produce neither significant narcosis or chronic injury. An earlier skin notation was deleted because percutaneous absorption is gradual and protracted and does not substantially

Continued...

contribute to the dose received by inhalation.
Odour Safety Factor(OSF)
OSF=4 (XYLENE)

For ethylene glycol monobutyl ether (2-butoxyethanol)
Odour Threshold Value: 0.10 ppm (detection), 0.35 ppm (recognition)
Although rats appear to be more susceptible than other animals anaemia is not uncommon amongst humans following exposure. The TLV reflects the need to maintain exposures below levels found to cause blood changes in experimental animals. It is concluded that this limit will reduce the significant risk of irritation, haematologic effects and other systemic effects observed in humans and animals exposed to higher vapour concentrations. The toxic effects typical of some other glycol ethers (pancytopenia, testis atrophy and teratogenic effects) are not found with this substance.
Odour Safety Factor (OSF)
OSF=2E2 (2-BUTOXYETHANOL)
Odour Threshold Value: 3.3 ppm (detection), 7.6 ppm (recognition)
Exposure at or below the recommended isopropanol TLV-TWA and STEL is thought to minimise the potential for inducing narcotic effects or significant irritation of the eyes or upper respiratory tract. It is believed, in the absence of hard evidence, that this limit also provides protection against the development of chronic health effects. The limit is intermediate to that set for ethanol, which is less toxic, and n-propyl alcohol, which is more toxic, than isopropanol

Exposure controls

Appropriate engineering controls

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

For flammable liquids and flammable gases, local exhaust ventilation or a process enclosure ventilation system may be required. Ventilation equipment should be explosion-resistant.
Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

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

Within each range the appropriate value depends on:

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

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

Personal protection

Continued...

Eye and face protection	‣ Safety glasses with side shields. ‣ Chemical goggles. ‣ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	‣ Wear chemical protective gloves, e.g. PVC. ‣ Wear safety footwear or safety gumboots, e.g. Rubber **NOTE:** ‣ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ‣ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and.has to be observed when making a final choice. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: ‣ frequency and duration of contact, ‣ chemical resistance of glove material, ‣ glove thickness and ‣ dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). ‣ When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. ‣ When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. ‣ Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. ‣ Contaminated gloves should be replaced. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Body protection	See Other protection below
Other protection	‣ Overalls. ‣ PVC Apron. ‣ PVC protective suit may be required if exposure severe. ‣ Eyewash unit. ‣ Ensure there is ready access to a safety shower. · Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity. · For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets). · Non sparking safety or conductive footwear should be considered. Conductive footwear describes a boot or shoe with a sole made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground the foot an shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.
Thermal hazards	Not Available

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:
Revive 476S Solvent

Material	CPI
PE/EVAL/PE	A
BUTYL	C
BUTYL/NEOPRENE	C
HYPALON	C

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.
Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

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

Continued...

NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PVA	C
PVC	C
PVDC/PE/PVDC	C
SARANEX-23	C
TEFLON	C
VITON	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.

up to 50 x ES	Air-line*	-	-
up to 100 x ES	-	A-3 P2	-
100+ x ES	-	Air-line**	-

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

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Clear

Physical state	Liquid	**Relative density (Water = 1)**	0.8
Odour	Sharp	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	-9	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	HIGHLY FLAMMABLE.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water (g/L)	Miscible	**pH as a solution**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7

Continued...

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

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful. 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. 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 acute toxicity of inhaled alkylbenzenes is best described by central nervous system depression. As a rule, these compounds may also act as general anaesthetics. Systemic poisoning produced by general anaesthesia is characterised by lightheadedness, nervousness, apprehension, euphoria, confusion, dizziness, drowsiness, tinnitus, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness and respiratory depression and arrest. Cardiac arrest may result from cardiovascular collapse. Bradycardia, and hypotension may also be produced. Inhaled alkylbenzene vapours cause death in animals at air levels that are relatively similar (typically LC50s are in the range 5000 - 8000 ppm for 4 to 8 hour exposures). It is likely that acute inhalation exposure to alkylbenzenes resembles that to general anaesthetics. Alkylbenzenes are not generally toxic other than at high levels of exposure. This may be because their metabolites have a low order of toxicity and are easily excreted. There is little or no evidence to suggest that metabolic pathways can become saturated leading to spillover to alternate pathways. Nor is there evidence that toxic reactive intermediates, which may produce subsequent toxic or mutagenic effects, are formed Inhalation hazard is increased at higher temperatures. Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination Human overexposure to MIBK vapour may produce weakness, loss of appetite, headache, a burning sensation to the eyes, stomach-ache, nausea and vomiting. Sore throat, insomnia, somnolence, heartburn and intestinal pain have been reported by some workers. Tolerance is reported to be acquired over the workweek and lost during the weekend. Exposure to high concentrations (>1000 ppm) can produce central nervous system depression and narcosis. Lower doses (80-500 ppm) can cause weakness, headache and nausea. Rats, mice, dogs and monkeys that inhaled 100 or 200 ppm MIBK 24 hrs/day showed no outward adverse effects during 2 weeks of exposure. At 200 ppm rats showed increased absolute liver and kidney weights and increased organ-to-body weight ratios. Examination of the proximal tubules showed toxic nephrosis (hyaline droplet degeneration and occasional focal tubular necrosis) in rats exposed to 100 ppm. This damage was considered transient and reversible. Discriminatory behaviour and memory in baboons was effected at exposures of 50 ppm for 7 days. Ethylene glycol monobutyl ether (2-butoxyethanol) and its metabolite butoxyacetic acid are haemolytic agents, causing red blood cell destruction. On the basis of industrial experience and volunteer short-term exposure humans are shown to be less susceptible than experimental animals to exposure. In 8-hour exposures at concentrations of 200 or 100 ppm no objective effects were seen other than raised urinary excretion of the metabolite butoxyacetic acid. No increased osmotic fragility of the red blood cell is observed. Subjectively these concentrations were uncomfortable with mild eye, nose and throat irritation occurring. No clinical signs of adverse effects nor subjective complaints were produced when male volunteers were exposed for 2 hours to 20 ppm during light physical exercise. Other studies have established that the most sensitive indicators of toxic effect observed from many of the glycol ethers is an increase in erythrocyte osmotic fragility in rats. This appears to be related to the development of haemoglobinuria at higher exposure levels. The odour of isopropanol may give some warning of exposure, but odour fatigue may occur. Inhalation of isopropanol may produce irritation of the nose and throat with sneezing, sore throat and runny nose. The effects in animals subject to a single exposure, by inhalation, included inactivity or anaesthesia and histopathological changes in the nasal canal and auditory canal. Headache, fatigue, lassitude, irritability and gastrointestinal disturbances (e.g., nausea, anorexia and flatulence) are the most common symptoms of xylene overexposure. Injury to the heart, liver, kidneys and nervous system has also been noted amongst workers. Transient memory loss, renal impairment, temporary confusion and some evidence of disturbance of liver

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	function was reported in three workers overcome by gross exposure to xylene (10000 ppm). One worker died and autopsy revealed pulmonary congestion, oedema and focal alveolar haemorrhage. Volunteers inhaling xylene at 100 ppm for 5 to 6 hours showed changes in manual coordination reaction time and slight ataxia. Tolerance developed during the workweek but was lost over the weekend. Physical exercise may antagonise this effect. Xylene body burden in humans exposed to 100 or 200 ppm xylene in air depends on the amount of body fat with 4% to 8% of total absorbed xylene accumulating in adipose tissue. Xylene is a central nervous system depressant. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.
Ingestion	Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result. Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis). The material is not thought to produce adverse health effects following ingestion (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum. Severe acute exposure to ethylene glycol monobutyl ether, by ingestion, may cause kidney damage, haemoglobinuria, (blood in urine) and is potentially fatal. Following ingestion, a single exposure to isopropyl alcohol produced lethargy and non-specific effects such as weight loss and irritation. Ingestion of near-lethal doses of isopropanol produces histopathological changes of the stomach, lungs and kidneys, incoordination, lethargy, gastrointestinal tract irritation, and inactivity or anaesthesia. Swallowing 10 ml. of isopropanol may cause serious injury; 100 ml. may be fatal if not promptly treated. The adult single lethal doses is approximately 250 ml. The toxicity of isopropanol is twice that of ethanol and the symptoms of intoxication appear to be similar except for the absence of an initial euphoric effect; gastritis and vomiting are more prominent. Ingestion may cause nausea, vomiting, and diarrhoea. There is evidence that a slight tolerance to isopropanol may be acquired.
Skin Contact	Skin contact with the material may be harmful; systemic effects may result following absorption. The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either ‣ produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or ‣ produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. 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. Ethylene glycol monobutyl ether (2-butoxyethanol) penetrates the skin easily and toxic effects via this route may be more likely than by inhalation. Percutaneous uptake rate in the guinea pig was estimated to be 0.25 umole/min/cm2. 511ipa
Eye	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. At concentrations of 100-200 ppm MIBK, the vapour may irritate the eyes and respiratory tract When instilled in rabbit eyes ethylene glycol monobutyl ether produced pain, conjunctival irritation, and transient corneal injury. Isopropanol vapour may cause mild eye irritation at 400 ppm. Splashes may cause severe eye irritation, possible corneal burns and eye damage. Eye contact may cause tearing or blurring of vision.
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals. On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Experiments with rats exposed to MIBK have shown nerve changes characteristic of neuropathy (disease of the peripheral nerves usually causing weakness and numbness). Chronic occupational exposure to 500 ppm MIBK in air (20-30 mins/day, and 80 ppm for the remainder of the workday resulted in nausea, headache, burning eyes, and weakness in over half the workers. Some workers reported somnolence, insomnia and intestinal pain, and 4/19 appeared to have enlarged livers. This study was continued 5 years after MIBK concentrations had been reduced to 100-105 ppm for the 20-30 minutes exposures and 50 ppm for the general exposure. A few workers still experienced gastrointestinal and neurological problems and slight liver enlargement was found in two individuals.

Continued...

Prolonged or repeated contact with xylenes may cause defatting dermatitis with drying and cracking. Chronic inhalation of xylenes has been associated with central nervous system effects, loss of appetite, nausea, ringing in the ears, irritability, thirst anaemia, mucosal bleeding, enlarged liver and hyperplasia. Exposure may produce kidney and liver damage. In chronic occupational exposure, xylene (usually mix ed with other solvents) has produced irreversible damage to the central nervous system and ototoxicity (damages hearing and increases sensitivity to noise), probably due to neurotoxic mechanisms. Industrial workers exposed to xylene with a maximum level of ethyl benzene of 0.06 mg/l (14 ppm) reported headaches and irritability and tired quickly. Functional nervous system disturbances were found in some workers employed for over 7 years whilst other workers had enlarged livers.

Xylene has been classed as a developmental toxin in some jurisdictions.
Small excess risks of spontaneous abortion and congenital malformation were reported amongst women exposed to xylene in the first trimester of pregnancy. In all cases, however, the women were also been exposed to other substances. Evaluation of workers chronically exposed to xylene has demonstrated lack of genotoxicity. Exposure to xylene has been associated with increased risks of haemopoietic malignancies but, again, simultaneous exposure to other substances (including benzene) complicates the picture. A long-term gavage study to mixed xylenes (containing 17% ethyl benzene) found no evidence of carcinogenic activity in rats and mice of either sex.
Long term or repeated ingestion exposure of isopropanol may produce incoordination, lethargy and reduced weight gain.

Repeated inhalation exposure to isopropanol may produce narcosis, incoordination and liver degeneration. Animal data show developmental effects only at exposure levels that produce toxic effects in the adult animals. Isopropanol does not cause genetic damage in bacterial or mammalian cell cultures or in animals.

There are inconclusive reports of human sensitisation from skin contact with isopropanol. Chronic alcoholics are more tolerant of systemic isopropanol than are persons who do not consume alcohol; alcoholics have survived as much as 500 ml. of 70% isopropanol.

Continued voluntary drinking of a 2.5% aqueous solution through two successive generations of rats produced no reproductive effects.
NOTE: Commercial isopropanol does not contain "isopropyl oil". An excess incidence of sinus and laryngeal cancers in isopropanol production workers has been shown to be caused by the byproduct "isopropyl oil". Changes in the production processes now ensure that no byproduct is formed. Production changes include use of dilute sulfuric acid at higher temperatures.

Revive 476S Solvent	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
methyl isobutyl ketone	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >16000 mg/kg[1]	Eye (human): 200 ppm/15m
	Oral (rat) LD50: 2984 mg/kg[1]	Eye (rabbit): 40 mg - SEVERE
		Eye (rabbit): 500 mg/24h - mild
		Skin (rabbit): 500 mg/24h - mild
methyl ethyl ketone	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >8100 mg/kg[1]	- mild
	Inhalation (rat) LC50: 23.5 mg/L/8H[2]	Eye (human): 350 ppm -irritant
	Inhalation (rat) LC50: 50.1 mg/L/8 hr[2]	Eye (rabbit): 80 mg - irritant
	Oral (rat) LD50: 3474.9 mg/kg[1]	Skin (rabbit): 402 mg/24 hr - mild
		Skin (rabbit):13.78mg/24 hr open
xylene	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >1700 mg/kg[2]	Eye (human): 200 ppm irritant
	Inhalation (rat) LC50: 5000 ppm/4h[2]	Eye (rabbit): 5 mg/24h SEVERE
	Oral (rat) LD50: 4300 mg/kgt[2]	Eye (rabbit): 87 mg mild
		Skin (rabbit):500 mg/24h moderate
aromatic hydrocarbons, C9-11	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
ethylene glycol monobutyl ether	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	* [Union Carbide]
	Inhalation (rat) LC50: 450 ppm/4H[2]	Eye (rabbit): 100 mg SEVERE
	Oral (rat) LD50: 250 mg/kg[2]	Eye (rabbit): 100 mg/24h-moderate

Continued...

		Skin (rabbit): 500 mg, open; mild
isopropanol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 12792 mg/kg[1]	Eye (rabbit): 10 mg - moderate
	Inhalation (rat) LC50: 72.6 mg/L/4h[2]	Eye (rabbit): 100 mg - SEVERE
	Oral (rat) LD50: 5000 mg/kg[2]	Eye (rabbit): 100mg/24hr-moderate
		Skin (rabbit): 500 mg - mild
gamma-glycidoxypropyltrimethoxysilane	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 4247.9 mg/kg[1]	Not Available
	Oral (rat) LD50: >5350 mg/kg[1]	

Legend: *1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's msds. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*

Revive 476S Solvent

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.

No significant acute toxicological data identified in literature search. For
alkoxysilanes:
Low molecular weight alkoxysilanes (including alkyl orthosilicates) are a known concern for lung toxicity, due to inhalation of vapours or aerosols causing irreversible lung damage at low doses.
Alkoxysilane groups that rapidly hydrolyse when in contact with water, result in metabolites that may only cause mild skin irritation. Although there appears to be signs of irritation under different test conditions, based on the available information, the alkoxysilanes cannot be readily classified as a skin irritant.

The trimethoxysilane group of chemicals have previously been associated with occupational eye irritation in exposed workers who experienced severe inflammation of the cornea . Based on the collective information, these substances are likely to be severe irritants to the eyes. Methoxysilanes are generally reported to possess higher reactivity and toxicity compared to
ethoxysilanes; some methoxysilanes appear to be carcinogenic .In the US, alkoxysilanes with alkoxy groups greater than C2 are classified as moderate concern.
Based on available information on methoxysilanes, the possibility that this family causes skin sensitisation cannot be ruled out. Amine-functional methoxysilanes have previously been implicated as a cause of occupational contact dermatitis, often as a result of repeated skin exposure with workers involved in the manufacture or use of the resins containing the chemical during fibreglass production.

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.
Oxiranes (including glycidyl ethers and alkyl oxides, and epoxides) exhibit many common characteristics with respect to animal toxicology. One such oxirane is ethyloxirane; data presented here may be taken as representative.
for 1,2-butylene oxide (ethyloxirane):
Ethyloxirane increased the incidence of tumours of the respiratory system in male and female rats exposed via inhalation. Significant increases in nasal papillary adenomas and combined alveolar/bronchiolar adenomas and carcinomas were observed in male rats exposed to 1200 mg/m3 ethyloxirane via inhalation for 103 weeks. There was also a significant positive trend in the incidence of combined alveolar/bronchiolar adenomas and carcinomas. Nasal papillary adenomas were also observed in 2/50 high-dose female rats with none occurring in control or low-dose animals. In mice exposed chronically via inhalation, one male mouse developed a squamous cell papilloma in the nasal cavity (300 mg/m3) but other tumours were not observed. Tumours were not observed in mice exposed chronically via dermal exposure. When trichloroethylene containing 0.8% ethyloxirane was administered

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orally to mice for up to 35 weeks, followed by 0.4% from weeks 40 to 69, squamous-cell carcinomas of the forestomach occurred in 3/49 males (p=0.029, age-adjusted) and 1/48 females at week 106. Trichloroethylene administered alone did not induce these tumours and they were not observed in control animals . Two structurally related substances, oxirane (ethylene oxide) and methyloxirane (propylene oxide), which are also direct-acting alkylating agents, have been classified as carcinogenic Exposure of pregnant rats to ethylene glycol monobutyl ether (2-butoxyethanol) at 100 ppm or rabbits at 200 ppm during organogenesis resulted in maternal toxicity and embryotoxicity including a decreased number of viable implantations per litter. Slight foetoxicity in the form of poorly ossified or unossified skeletal elements was also apparent in rats. Teratogenic effects were not observed in other species.
At least one researcher has stated that the reproductive effects were less than that of other monoalkyl ethers of ethylene glycol.
Chronic exposure may cause anaemia, macrocytosis, abnormally large red cells and abnormal red cell fragility.
Exposure of male and female rats and mice for 14 weeks to 2 years produced a regenerative haemolytic anaemia and subsequent effects on the haemopoietic system in rats and mice. In addition, 2-butoxyethanol exposures caused increases in the incidence of neoplasms and nonneoplastic lesions
(1). The occurrence of the anaemia was concentration-dependent and more pronounced in rats and females. In this study it was proposed that 2-butoxyethanol at concentrations of 500 ppm and greater produced an acute disseminated thrombosis and bone infarction in male and female rats as a result of severe acute haemolysis and reduced deformability of erythrocytes or through anoxic damage to endothelial cells that compromise blood flow. In two-year studies, 2-butoxyethanol continued to affect circulating erythroid mass, inducing a responsive anaemia. Rats showed a marginal increase in the incidence of benign or malignant pheochromocytomas (combined) of the adrenal gland. In mice, 2-butoxyethanol exposure resulted in a concentration dependent increase in the incidence of squamous cell papilloma or carcinoma of the forestomach. It was hypothesised that exposure-induced irritation produced inflammatory and hyperplastic effects in the forestomach and that the neoplasia were associated with a continuation of the injury/ degeneration process. Exposure also produced a concentration -dependent increase in the incidence of haemangiosarcoma of the liver of male mice and hepatocellular carcinoma.

1: NTP Toxicology Program Technical report Series 484, March 2000.

METHYL ISOBUTYL KETONE

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 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.
For methyl isobutyl ketone (MIBK):
MIBK is primarily absorbed by the lungs in animals and humans; it can however be absorbed by the gastrointestinal system and through skin.
In two cases involving individuals exposed to the vapour MIBK was found in the brain, liver, lung, vitreous fluid, kidney and blood.
Experiments in guinea pigs show that MIBK is metabolised to 4-hydroxy-4-methyl-2-pentanone and 4-methyl-2-pentanol. Ketones are generally excreted rapidly in expired air. Small amounts of MIBK are also excreted in the urine. Humans excreted less than 0.1% of the dose as unmetabolised MIBK in the urine within the first 3 hours post exposure. Serum half-life in guinea pigs is about 55 minutes with a clearance time of 6 hours

In animal studies, the acute systemic toxicity of MIBK, via the oral and inhalation routes of exposure, is low. In a 90-day gavage study on rats, a no-observed-effect level (NOEL) of 50 mg/kg per day was found. In 90-day inhalation studies on rats and mice, concentrations of up to 4100 mg/m3 (1000 ppm) did not result in significant toxicity, though compound-related reversible morphological changes were reported in the liver and kidney. Evidence of central nervous system depression was seen in animals exposed to a level of 4100 mg/m3 (1000 ppm). In a number of studies, exposure to MIBK concentrations as low as 1025 mg/m3 (250 ppm) resulted in an increase in liver size and induced hepatic microsomal metabolism. This may be responsible for the exacerbation of haloalkane toxicity and for the potentiation of the neurotoxicity of *n*-hexane. MIBK was also found to potentiate the cholestatic effects of manganese given with, or without, bilirubin. In 90-day studies on mice, rats, dogs, and monkeys, only male rats developed hyaline droplets in the proximal tubules of the kidney. Effects on behaviour were reported in baboons exposed for 7 days to 205 mg/m3 (50 ppm). At a

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	concentration of 4100 mg/m3 (1000 ppm), MIBK was not embryotoxic, foetotoxic, or teratogenic in rats or mice. Foetotoxicity was only observed at concentrations of MIBK that caused maternal toxicity. MIBK did not induce gene mutations in *in vitro* bacterial test systems with, or without, metabolic activation. Negative results were also obtained *in vitro* with, or without, metabolic activation, in tests for mitotic gene conversion in yeast, and for gene mutation in cultured mammalian cells. The results of *in vitro* assays for unscheduled DNA synthesis in primary rat hepatocytes and for structural chromosome damage in cultured rat liver cells were negative. An *in vivo* micronucleus test on mice was negative. These data indicate that MIBK is not genotoxic. No long-term or carcinogenicity studies are available. The toxicity of MIBK for aquatic organisms and microorganisms is low.
METHYL ETHYL KETONE	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 material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Methyl ethyl ketone is considered to have a low order of toxicity; however methyl ethyl ketone is often used in combination with other solvents and the toxic effects of the mix may be greater than either solvent alone. Combinations of n-hexane with methyl ethyl ketone and also methyl n-butyl ketone with methyl ethyl ketone show increase in peripheral neuropathy, a progressive disorder of nerves of extremities. Combinations with chloroform also show increase in toxicity
XYLENE	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. The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing. Reproductive effector in rats
AROMATIC HYDROCARBONS, C9-11	No significant acute toxicological data identified in literature search.
ETHYLENE GLYCOL MONOBUTYL ETHER	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 epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. For ethylene glycol monoalkyl ethers and their acetates (EGMAEs): Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates. EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers. **Acute Toxicity:** Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m3) for EGHE, LC50 > 400ppm (2620 mg/m3) for EGBEA to LC50 > 2132 ppm (9061 mg/m3) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered

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to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitisers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE *in vitro* than those of rats.

Repeat dose toxicity: The fact that the NOAEL for repeated dose toxicity of EGBE is less than that of EGPE is consistent with red blood cells being more sensitive to EGBE than EGPE. Blood from mice, rats, hamsters, rabbits and baboons were sensitive to the effects of BAA *in vitro* and displayed similar responses, which included erythrocyte swelling (increased haematocrit and mean corpuscular hemoglobin), followed by hemolysis. Blood from humans, pigs, dogs, cats, and guinea pigs was less sensitive to haemolysis by BAA *in vitro*.
Mutagenicity: In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in *S. typhimurium* strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. *In vitro* cytogenicity and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and in vivo micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic.
Carcinogenicity: In a 2-year inhalation chronic toxicity and carcinogenicity study with EGBE in rats and mice a significant increase in the incidence of liver haemangiosarcomas was seen in male mice and forestomach tumours in female mice. It was decided that based on the mode of action data available, there was no significant hazard for human carcinogenicity
Reproductive and developmental toxicity. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes).
Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m3 and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m3), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m3), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m3) indicate that the members of the category are not teratogenic.
The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m3 (rabbit-EGPE), 100 ppm or 425 mg/m3 (rat-EGPE), 50 ppm or 241 mg/m3 (rat EGBE) and 100 ppm or 483 mg/m3 (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m3 (rat and rabbit-EGHE).
Exposure of pregnant rats to ethylene glycol monobutyl ether (2-butoxyethanol) at 100 ppm or rabbits at 200 ppm during organogenesis resulted in maternal toxicity and embryotoxicity including a decreased number of viable implantations per litter. Slight foetoxicity in the form of poorly ossified or unossified skeletal elements was also apparent in rats. Teratogenic effects were not observed in other species.
At least one researcher has stated that the reproductive effects were less than that of other monoalkyl ethers of ethylene glycol.
Chronic exposure may cause anaemia, macrocytosis, abnormally large red cells and abnormal red cell fragility.
Exposure of male and female rats and mice for 14 weeks to 2 years produced a regenerative haemolytic anaemia and subsequent effects on the haemopoietic system in rats and mice. In addition, 2-butoxyethanol exposures caused increases in the incidence of neoplasms and nonneoplastic lesions
(1). The occurrence of the anaemia was concentration-dependent and more pronounced in rats and females. In this study it was proposed that 2-butoxyethanol at concentrations of 500 ppm and greater produced an acute disseminated thrombosis and bone infarction in male and female rats as a result of severe acute haemolysis and reduced deformability of erythrocytes or through anoxic damage to endothelial cells that compromise blood flow. In two-year studies, 2-butoxyethanol continued to affect circulating erythroid mass, inducing a responsive anaemia. Rats showed a marginal increase in the incidence of benign or malignant pheochromocytomas (combined) of the adrenal gland. In mice, 2-butoxyethanol exposure resulted in a concentration dependent increase in the incidence of squamous cell papilloma or carcinoma of the forestomach. It was hypothesised that exposure-induced irritation produced inflammatory and hyperplastic effects in the forestomach and that the neoplasia were associated with a continuation of the injury/ degeneration process. Exposure also produced a concentration -dependent increase in the incidence of haemangiosarcoma of the liver of male mice and hepatocellular carcinoma.

1: NTP Toxicology Program Technical report Series 484, March 2000. For
ethylene glycol:
Ethylene glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol.

dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by

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aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO2, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO2, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested.

Respiratory Effects. Respiratory system involvement occurs 12-24 hours after ingestion of sufficient amounts of ethylene glycol and is considered to be part of a second stage in ethylene glycol poisoning The symptoms include hyperventilation, shallow rapid breathing, and generalized pulmonary edema with calcium oxalate crystals occasionally present in the lung parenchyma. Respiratory system involvement appears to be dose-dependent and occurs concomitantly with cardiovascular changes. Pulmonary infiltrates and other changes compatible with adult respiratory distress syndrome (ARDS) may characterise the second stage of ethylene glycol poisoning Pulmonary oedema can be secondary to cardiac failure, ARDS, or aspiration of gastric contents. Symptoms related to acidosis such as hyperpnea and tachypnea are frequently observed; however, major respiratory morbidities such as pulmonary edema and bronchopneumonia are relatively rare and usually only observed with extreme poisoning (e.g., in only 5 of 36 severely poisoned cases).

Cardiovascular Effects. Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12- 24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of ethylene glycol. Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases.Therefore, it appears that acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown.

Gastrointestinal Effects. Nausea, vomiting with or without blood, pyrosis, and abdominal cramping and pain are common early effects of acute ethylene glycol ingestion. Acute effects of ethylene glycol ingestion in one patient included intermittent diarrhea and abdominal pain, which were attributed to mild colonic ischaemia; severe abdominal pain secondary to colonic stricture and perforation developed 3 months after ingestion, and histology of the resected colon showed birefringent crystals highly suggestive of oxalate deposition.

Musculoskeletal Effects. Reported musculoskeletal effects in cases of acute ethylene glycol poisoning have included diffuse muscle tenderness and myalgias associated with elevated serum creatinine phosphokinase levels, and myoclonic jerks and tetanic contractions associated with hypocalcaemia.

Hepatic Effects. Central hydropic or fatty degeneration, parenchymal necrosis, and calcium oxalate crystals in the liver have been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol.
Renal Effects. Adverse renal effects after ethylene glycol ingestion in humans can be observed during the third stage of ethylene glycol toxicity 24-72 hours after acute exposure. The hallmark of renal toxicity is the presence of birefringent calcium oxalate monohydrate crystals deposited in renal tubules and their presence in urine after ingestion of relatively high amounts of ethylene glycol. Other signs of nephrotoxicity can include tubular cell degeneration and necrosis and tubular interstitial inflammation. If untreated, the degree of renal damage caused by high doses of ethylene glycol progresses and leads to haematuria, proteinuria, decreased renal function, oliguria, anuria , and ultimately renal failure. These changes in the kidney are linked to acute tubular necrosis but normal or near normal renal function can return with adequate supportive therapy.

Metabolic Effects. One of the major adverse effects following acute oral exposure of humans to ethylene glycol involves metabolic changes. These changes occur as early as 12 hours after ethylene glycol exposure. Ethylene glycol intoxication is accompanied by metabolic acidosis which is manifested by decreased pH and bicarbonate content of serum and other bodily fluids caused by accumulation of excess glycolic acid. Other characteristic metabolic effects of ethylene glycol poisoning are increased serum anion gap, increased osmolal gap, and hypocalcaemia. Serum anion gap is calculated from concentrations of sodium, chloride, and bicarbonate, is normally 12-16 mM, and is typically elevated after ethylene glycol ingestion due to increases in unmeasured metabolite anions (mainly glycolate).
Neurological Effects: Adverse neurological reactions are among the first symptoms to appear in humans after ethylene glycol ingestion. These early neurotoxic effects are also the only symptoms attributed to unmetabolised ethylene glycol. Together with metabolic changes, they occur during the period of 30 minutes to 12 hours after exposure and are considered to be part of the first stage in ethylene glycol intoxication. In cases of acute intoxication, in which a large amount of ethylene glycol is ingested over a very short time period, there is a progression of neurological manifestations which, if not treated, may lead to generalized seizures and coma. Ataxia, slurred speech, confusion, and somnolence are common during the initial phase of ethylene glycol intoxication as are irritation, restlessness, and disorientation. Cerebral edema and crystalline deposits of calcium oxalate in the walls of small blood vessels in the brain were found at autopsy in people who died after acute ethylene glycol ingestion.

Effects on cranial nerves appear late (generally 5-20 days post-ingestion), are relatively rare, and

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	according to some investigators constitute a fourth, late cerebral phase in ethylene glycol intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar nerves and are reversible over many months. **Reproductive Effects:** Reproductive function after intermediate-duration oral exposure to ethylene glycol has been tested in three multi-generation studies (one in rats and two in mice) and several shorter studies (15-20 days in rats and mice). In these studies, effects on fertility, foetal viability, and male reproductive organs were observed in mice, while the only effect in rats was an increase in gestational duration. **Developmental Effects:** The developmental toxicity of ethylene glycol has been assessed in several acute-duration studies using mice, rats, and rabbits. Available studies indicate that malformations, especially skeletal malformations occur in both mice and rats exposed during gestation; mice are apparently more sensitive to the developmental effects of ethylene glycol. Other evidence of embyrotoxicity in laboratory animals exposed to ethylene glycol exposure includes reduction in foetal body weight. **Cancer:** No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol. **Genotoxic Effects**: Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available *in vivo* and *in vitro* laboratory studies provide consistently negative genotoxicity results for ethylene glycol. NOTE: Changes in kidney, liver, spleen and lungs are observed in animals exposed to high concentrations of this substance by all routes. ** ASCC (NZ) SDS
ISOPROPANOL	For isopropanol (IPA): **Acute toxicity:** Isopropanol has a low order of acute toxicity. It is irritating to the eyes, but not to the skin. Very high vapor concentrations are irritating to the eyes, nose, and throat, and prolonged exposure may produce central nervous system depression and narcosis. Human volunteers reported that exposure to 400 ppm isopropanol vapors for 3 to 5 min. caused mild irritation of the eyes, nose and throat. Although isopropanol produced little irritation when tested on the skin of human volunteers, there have been reports of isolated cases of dermal irritation and/or sensitization. The use of isopropanol as a sponge treatment for the control of fever has resulted in cases of intoxication, probably the result of both dermal absorption and inhalation. There have been a number of cases of poisoning reported due to the intentional ingestion of isopropanol, particularly among alcoholics or suicide victims. These ingestions typically result in a comatose condition. Pulmonary difficulty, nausea, vomiting, and headache accompanied by various degrees of central nervous system depression are typical. In the absence of shock, recovery usually occurred. **Repeat dose studies**: The systemic (non-cancer) toxicity of repeated exposure to isopropanol has been evaluated in rats and mice by the inhalation and oral routes. The only adverse effects-in addition to clinical signs identified from these studies were to the kidney. **Reproductive toxicity**: A recent two-generation reproductive study characterised the reproductive hazard for isopropanol associated with oral gavage exposure. This study found that the only reproductive parameter apparently affected by isopropanol exposure was a statistically significant decrease in male mating index of the F1 males. It is possible that the change in this reproductive parameter was treatment related and significant, although the mechanism of this effect could not be discerned from the results of the study. However, the lack of a significant effect of the female mating index in either generation, the absence of any adverse effect on litter size, and the lack of histopathological findings of the testes of the high-dose males suggest that the observed reduction in male mating index may not be biologically meaningful. **Developmental toxicity:** The developmental toxicity of isopropanol has been characterized in rat and rabbit developmental toxicity studies. These studies indicate that isopropanol is not a selective developmental hazard. Isopropanol produced developmental toxicity in rats, but not in rabbits. In the rat, the developmental toxicity occurred only at maternally toxic doses and consisted of decreased foetal body weights, but no teratogenicity **Genotoxicity:** All genotoxicity assays reported for isopropanol have been negative **Carcinogenicity:** rodent inhalation studies were conduct to evaluate isopropanol for cancer potential. The only tumor rate increase seen was for interstitial (Leydig) cell tumors in the male rats. Interstitial cell tumors of the testis is typically the most frequently observed spontaneous tumor in aged male Fischer 344 rats. These studies demonstrate that isopropanol does not exhibit carcinogenic potential relevant to humans. Furthermore, there was no evidence from this study to indicate the development of carcinomas of the testes in the male rat, nor has isopropanol been found to be genotoxic. Thus, the testicular tumors seen in the isopropanol exposed male rats are considered of no significance in terms of human cancer risk assessment 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. The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.

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GAMMA-GLYCIDOXYPROPYLTRIMETHOXYSILANE	For alkoxysilanes: Low molecular weight alkoxysilanes (including alkyl orthosilicates) are a known concern for lung toxicity, due to inhalation of vapours or aerosols causing irreversible lung damage at low doses. Alkoxysilane groups that rapidly hydrolyse when in contact with water, result in metabolites that may only cause mild skin irritation. Although there appears to be signs of irritation under different test conditions, based on the available information, the alkoxysilanes cannot be readily classified as a skin irritant. The trimethoxysilane group of chemicals have previously been associated with occupational eye irritation in exposed workers who experienced severe inflammation of the cornea . Based on the collective information, these substances are likely to be severe irritants to the eyes. Methoxysilanes are generally reported to possess higher reactivity and toxicity compared to ethoxysilanes; some methoxysilanes appear to be carcinogenic .In the US, alkoxysilanes with alkoxy groups greater than C2 are classified as moderate concern. Based on available information on methoxysilanes, the possibility that this family causes skin sensitisation cannot be ruled out. Amine-functional methoxysilanes have previously been implicated as a cause of occupational contact dermatitis, often as a result of repeated skin exposure with workers involved in the manufacture or use of the resins containing the chemical during fibreglass production. For gamma-glycidopropyltrimethoxysilane (GPTMS) GPTMS is subject to rapid hydrolysis, and the observed toxicity is expected to be due primarily to methanol and silanetriols. GPTMS has been tested for acute toxicity by the oral, dermal, and inhalation routes of exposure. Reported acute oral LD50s in rats range from 7010 to 16900 mg/kg bw and > 5 ml/kg bw to 22.6 ml/kg bw. The dermal LD50s are 6800 mg/kg bw and 4.0 ml/kg bw. The 4-hour inhalation LC50 was greater than 2.7 mg/L in one study and greater than 5.3 mg/L in another study. GPTMS is mildly irritating to the skin and eyes and is not a known skin sensitiser in humans or in animals. Following inhalation exposures of rats to target aerosol concentrations of 0, 75, 225 and 750 mg/m3 (actual concentrations were 0, 77, 226, 707 mg/m3 (males) and 0, 73, 226, 734 mg/m3 (females)), GPTMS in 9 repeated exposures administered over two weeks, 6 animals in the high dose group died or were sacrificed from three to five days after initiation of the study. These animals had signs of inanition but no acute tissue toxicity. At both the mid and high doses, rats exhibited some clinical signs including a dose-related decrease in body weight. Under the conditions of this study, the No Observed Adverse Effect Concentration is 225 mg/m3. Repeated exposure of rats by gavage to GPTMS doses of 40, 400 and 1000 mg/kg bw/day for 5 days/week for 4 weeks resulted in no test substance-related organ weights effects or gross or microscopic pathological changes. Under the conditions of this study, the NOAEL for the test substance was found to be 1000 mg/kg bw/day. **Genotoxicity:** GPTMS did not induce chromosomal damage in mouse bone marrow cells by gavage at doses of 500, 1670 and 5000 mg/kg bw/day, or when administered by intraperitoneal (i.p.) injection at 1600 mg/kg bw/day. However, chromosomal damage was induced in mouse bone marrow cells when administered by i.p. in water at doses of 500, 1000 and 2000 mg/kg bw/day. GPTMS induced gene mutations in bacteria. GPTMS induced gene mutations in mouse lymphoma L1578Y TK cells but did not induce forward mutations in CHO cells. GPTMS induced SCE in vitro. There are no in vivo gene mutation data. **Carcinogenicity:** GPTMS was not considered tumourigenic when applied to the clipped skin of mice (25 ul dose of 25% GPTMS in acetone) three times per week for approximately 78 weeks. Note that there was only one dose level, and this dose was relatively low. **Reproductive toxicity:** In a one-generation reproduction toxicity study in rats, no reproductive effects were observed at any of the doses tested (250, 500, or 1000 mg/kg bw/day). At 1000 mg/kg bw/day, treatment with GPTMS resulted in the following signs in parental animals: discomfort after dosing (noted for females from early/mid gestation onwards), decreased body weight gain (males), increased mean relative liver and kidney weights (noted for males and females), and histopathological effects on livers and kidneys (males). Based on these data, a NOAEL for parental animals was established at 500 mg/kg bw/day. A NOAEL for reproductive effects was established at 1000 mg/kg bw/day. **Developmental toxicity:** Three developmental studies have been conducted using GPTMS. In a rabbit study, the maternal NOAEL was 200 mg/kg bw/day and the developmental NOAEL was 400 mg/kg bw/day (the highest dose tested). In a rat study, the NOAELs for both maternal and developmental toxicity were also at the highest dose tested (1000 mg/kg bw/day). In another rat study, developmental effects were observed at the maternally toxic dose of 3000 mg/kg bw/day (again, the highest dose tested). The material may be irritating to the eye, with prolonged contact causing 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 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**	⊘

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Serious Eye Damage/Irritation	✔	**STOT - Single Exposure**	⊘
Respiratory or Skin sensitisation	⊘	**STOT - Repeated Exposure**	⊘
Mutagenicity	⊘	**Aspiration Hazard**	✔

Legend: ✔ *– Data required to make classification available*
✘ *– Data available but does not fill the criteria for classification*
⊘ *– Data Not Available to make classification*

CMR STATUS

REPROTOXIN	methyl ethyl ketone	ILO Chemicals in the electronics industry that have toxic effects on reproduction
	xylene	ILO Chemicals in the electronics industry that have toxic effects on reproduction

SKIN	ethylene glycol monobutyl ether	Australia Exposure Standards - Skin	Sk
	gamma-glycidoxypropyltrimethoxysilane	Australia Exposure Standards - Skin	Sk

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

NOT AVAILABLE

Ingredient	Endpoint	Test Duration	Effect	Value	Species	BCF
methyl isobutyl ketone	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
methyl ethyl ketone	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
xylene	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
aromatic hydrocarbons, C9-11	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
ethylene glycol monobutyl ether	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
isopropanol	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
gamma-glycidoxypropyltrimethoxysilane	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available

Within an aromatic series, acute toxicity increases with increasing alkyl substitution on the aromatic nucleus. For example, there is an increase in toxicity as alkylation of the naphthalene structure increases. The order of most toxic to least in a study using grass shrimp (Palaemonetes pugio) and brown shrimp (Penaeus aztecus) was dimethylnaphthalenes > methylnaphthalenes >naphthalenes.
Studies conclude that the toxicity of an oil appears to be a function of its di-aromatic and tri-aromatic hydrocarbons, which includes three-ring hydrocarbons such as phenanthrene.
The heavier (4-, 5-, and 6-ring) PAHs are more persistent than the lighter (2- and 3-ring) PAHs and tend to have greater carcinogenic and other chronic impact potential. PAHs in general are more frequently associated with chronic risks. These risks include cancer and often are the result of exposures to complex mixtures of chronic-risk aromatics (such as PAHs, alkyl PAHs, benzenes, and alkyl benzenes), rather than exposures to low levels of a single compound.

Anthrcene is a phototoxic PAH . UV light greatly increases the toxicity of anthracene to bluegill sunfish. . Benchmarks developed in the absence of UV light may be under-protective, and biological resources in strong sunlight are at more risk than those that are not.
For ethylene glycol monoalkyl ethers and their acetates:
Members of this category include ethylene glycol propyl ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE)
Environmental fate:
The ethers, like other simple glycol ethers possess no functional groups that are readily subject to hydrolysis in the presence of waters. The acetates possess an ester group that hydrolyses in neutral ambient water under abiotic conditions.
Level III fugacity modeling indicates that category members, when released to air and water, will partition predominately to water and, to a lesser extent, to air and soil. Estimates of soil and sediment partition coefficients (Kocs ranging from 1- 10) suggest that category members would exhibit high soil mobility. Estimated bioconcentration factors (log BCF) range from 0.463 to 0.732. Biodegradation studies indicate that all category members are readily biodegradable. The physical chemistry and environmental fate properties indicate that category members will not persist or bioconcentrate in the environment.

Ecotoxicity:
Glycol ether acetates do not hydrolyse rapidly into their corresponding glycol ethers in water under environmental conditions. The LC50 or EC50 values for EGHE are lower than those for EGPE and EGBE (which have shorter chain lengths and lower log Kow values). Overall, the LC50 values for the glycol ethers in aquatic species range from 94 to > 5000 mg/L. For EGHE, the 96-hour LC50 for *Brachydanio rerio* (zebra fish) is between 94 and mg/L, the 48-hour EC50 for *Daphnia magna* was 145 mg/L and the 72-hour EC50 values for biomass and growth rate of algae (*Scenedesmus subspicatus*) were 98 and 198 mg/L, respectively. LC50/EC50 values for EGPE and EGBE in aquatic species are 835 mg/l or greater.
Aquatic toxicity data for EGBEA show a 96-hour LC50 of 28.3 mg/L for rainbow trout (*Oncorhynchus mykiss)*, a 48-hour LC50 of 37-143 mg/L for *Daphnia magna,* a 72-hour EC50 of greater than 500 mg/L for biomass or growth rate of algae (*Scenedesmus subspicatus* and *Pseudokirchneriella subcapitata,* respectively), and a 7-day EC10 of 30.4 mg/L and a NOEC of 16.4 mg/L for reproduction in *Ceriodaphnia dubia*. For gamma-glycidopropyltrimethoxysilane (GPTMS)
Environmental fate;
The melting point of GPTMS is < -70 C, the boiling point is 290 C at 1013 hPa, and the vapor pressure is 0.003 hPa at 20 C. Because GPTMS is hydrolytically unstable, the water solubility was not measured.

Continued...

Estimated values for water solubility (1 x 10+6 mg/L) and partition coefficient (log Kow = -0.9), may also not be accurate because of the chemical's rapid hydrolysis. From photodegradation modeling, the half-life in the atmosphere due to reaction with photochemically-induced OH radicals is estimated to be 5.8 hours. However, the overall half-life may be even shorter, as concurrent hydrolysis will also occur.
The measured hydrolysis half-life for GPTMS at 25 C ranges from 3 minutes to 6.5 hours over the pH range of 5 to 9. At pH 7 and 25 C, the half-life of the parent compound is 6.5 hours and the conversion of GPTMS to methanol and 3-glycidoxypropylsilanetriol is expected to reach 99.9% in <2.8 days. The epoxy group slowly reacts (over a period of months) to form diols in water. The Si-C bond will not undergo hydrolysis. The transient silanol groups will condense with other silanols to yield an epoxy-functional silicone resin (oligomer resin). The measured (and calculated) hydrolysis half-lives demonstrate that GPTMS is hydrolytically unstable over a range of environmentally relevant pH and temperature conditions.
Ecotoxicity:
Fish LC50 (96 h):juvenile rainbow trout (Oncorhynchus mykiss) 237 mg/l (semi-static); carp (Cyprinus carpio) 55 mg/l Daphnia
magna EC50 (48 h): 473-710 mg/l
Algae EbC50 (72 h): Selenastrum capricornutum 250 mg/l; ErC50 350 mg/l
Since GPTMS is subject to hydrolysis, which may occur during preparation of the dosing solutions and/or during testing, the observed toxicity is likely due to the hydrolysis products methanol and silanetriols.
for methyl isobutyl ketone (MIBK)
log Kow : 1.19-1.31
Koc : 19-106
Half-life (hr) air : 15-17
Half-life (hr) H2O surface water : 15-33
Henry's atm m3 /mol: 9.40E-05
BOD 5: 0.12-2.14,4. 4%
COD : 2.16, 79% ThOD :
2.72
BCF : 2-5
Environmental fate:
MIBK has a short half-life in the atmosphere and is also biodegraded in water. It is not expected to bioaccumulate. The toxicity of MIBK for microorganisms and aquatic organisms is low
MIBK is not expected to be retarded by absorption to soils rich in organic matter; therefore it is expected to be mobile in soil and subject to leaching. MIBK may contribute to the formation of photochemical smog.
The relatively high vapour pressure (14.5 mm Hg at 20 C) and estimated Henry's Law Constant (9.4 x 10-5 atm-m3/mol, 20 C) indicate that it volatilise from moist and dry soil When released to water it does not adsorb significantly to suspended solids, and will volatilise to the atmosphere.
Transformation and Persistence:
Air: The main degradation pathway for MIBK in the atmosphere is reaction with photochemically produced hydroxyl radicals. The half-life of MIBK from the reaction with hydroxyl radicals has been estimated to be 16-17 hours. based on its UV light absorption spectrum, direct photolysis of MIBK is expected to occur with a half-life of about 15 hours; acetone is a photo-oxidation product.
Smog chamber studies indicate MIBK is moderately reactive with nitrogen oxides producing acetone, peroxyacetylnitrate and methyl nitrate. As a volatile organic chemical (VOC) MIBK can contribute to photochemical smog in the presence of other VOCs
Soil: In wet or dry soil, MIBK may undergo volatilisation to air and photolysis on the soil surface. It is highly mobile and may be leached from the soil by water, and is susceptible to aerobic degradation by mixed populations of microorganisms.
Water: MIBK is not expected to bioconcentrate in fish and other aquatic organisms; its estimated bioconcentration factor is 2.
Ecotoxicity:
The toxicity of MIBK in aquatic organisms is low; toxicity values are greater than 100 mg/l.
MIBK also has low toxicity in terrestrial rodents for oral and inhalation exposure. It is moderately toxic to birds, based on oral LD50 values between 50 to 500 mg/kg (redwinged blackbirds)
Fish LC50 (96 h): fathead minnow (Pimephales promelas) 505 mg/l; (24 h): goldfish (Carassius auratus) 460 mg/l Daphnia
magna LC50 (24 h): 4280 mg/l
Brine shrimp LC50 (24 h):1230 mg/l
Significant environmental findings are limited. Oxiranes (including glycidyl ethers and alkyl oxides, and epoxides) exhibit common characteristics with respect to environmental fate and ecotoxicology. One such oxirane is ethyloxirane and data presented here may be taken as representative.
for 1,2-butylene oxide (ethyloxirane):
Environmental fate: Ethyloxirane is highly soluble in water and has a very low soil-adsorption coefficient, which suggests that if released to water, adsorption of ethyloxirane to sediment and suspended solids is not expected. Volatilisation of ethyloxirane from water surfaces would be expected based on the moderate estimated Henry's Law constant. If ethyloxirane is released to soil, it is expected to have low adsorption and thus very high mobility. Volatilisation from moist soil and dry soil surfaces is expected, based on its vapour pressure. It is expected that ethyloxirane exists solely as a vapour in ambient atmosphere, based on its very high vapour pressure. Ethyloxirane may also be removed from the atmosphere by wet deposition processes, considering its relatively high water solubility.

Persistence: The half-life in air is about 5.6 days from the reaction of ethyloxirane with photochemically produced hydroxyl radicals which indicates that this chemical meets the persistence criterion in air (half-life of = 2 days)*.
Ethyloxirane is hydrolysable, with a half-life of 6.5 days, and biodegradable up to 100% degradation and is not expected to persist in water. A further model-predicted biodegradation half-life of 15 days in water was obtained and used to predict the half-life of this chemical in soil and sediment by applying Boethling's extrapolation factors (t1/2water : t1/2 soil : t1/2sediment = 1: 1: 4) (Boethling 1995). According to these values, it can be concluded that ethyloxirane does not meet the persistence criteria in water and soil (half-lives = 182 days) and sediments (half-life = 365 days).
Experimental and modelled log Kow values of 0.68 and 0.86, respectively, indicate that the potential for bioaccumulation of ethyloxirane in organisms is likely to be low. Modelled bioaccumulation -factor (BAF) and bioconcentration -factor (BCF) values of 1 to 17 L/kg indicate that ethyloxirane does not meet the bioaccumulation criteria (BCF/BAF = 5000)*
Ecotoxicity:
Experimental ecotoxicological data for ethyloxirane (OECD 2001) indicate low to moderate toxicity to aquatic organisms. For fish and water flea, acute LC50/EC50 values vary within a narrow range of 70-215 mg/L; for algae, toxicity values exceed 500 mg/L, while for bacteria they are close to 5000 mg/L

* *Persistence and Bioaccumulation Regulations* (Canada 2000).
For xylenes :
log Koc : 2.05-3.08

Continued...

Koc : 25.4-204
Half-life (hr) air : 0.24-42
Half-life (hr) H2O surface water : 24-672
Half-life (hr) H2O ground : 336-8640
Half-life (hr) soil : 52-672
Henry's Pa m3 /mol: 637-879
Henry's atm m3 /mol: 7.68E-03
BOD 5 if unstated: 1.4,1%
COD : 2.56,13%
ThOD : 3.125
BCF : 23
log BCF : 1.17-2.41

Environmental Fate

Terrestrial fate:: Measured Koc values of 166 and 182, indicate that 3-xylene is expected to have moderate mobility in soil. Volatilisation of p-xylene is expected to be important from moist soil surfaces given a measured Henry's Law constant of 7.18x10-3 atm-cu m/mole. The potential for volatilisation of 3-xylene from dry soil surfaces may exist based on a measured vapor pressure of 8.29 mm Hg. p-Xylene may be degraded during its passage through soil). The extent of the degradation is expected to depend on its concentration, residence time in the soil, the nature of the soil, and whether resident microbial populations have been acclimated. p-Xylene, present in soil samples contaminated with jet fuel, was completely degraded aerobically within 5 days. In aquifer studies under anaerobic conditions, p-xylene was degraded, usually within several weeks, with the production of 3-methylbenzylfumaric acid, 3-methylbenzylsuccinic acid, 3-methylbenzoate, and 3-methylbenzaldehyde as metabolites.

Aquatic fate: Koc values indicate that p-xylene may adsorb to suspended solids and sediment in water. p-Xylene is expected to volatilise from water surfaces based on the measured Henry's Law constant. Estimated volatilisation half-lives for a model river and model lake are 3 hours and 4 days, respectively. BCF values of 14.8, 23.4, and 6, measured in goldfish, eels, and clams, respectively, indicate that bioconcentration in aquatic organisms is low. p-Xylene in water with added humic substances was 50% degraded following 3 hours irradiation suggesting that indirect photooxidation in the presence of humic acids may play an important role in the abiotic degradation of p-xylene. Although p-xylene is biodegradable and has been observed to degrade in pond water, there are insufficient data to assess the rate of this process in surface waters. p-Xylene has been observed to degrade in anaerobic and aerobic groundwater in several studies; however, it is known to persist for many years in groundwater, at least at sites where the concentration might have been quite high.

Atmospheric fate:

Most xylenes released to the environment will occur in the atmosphere and volatilisation is the dominant environmental fate process. In the ambient atmosphere, xylenes are expected to exist solely in the vapour phase. Xylenes are degraded in the atmosphere primarily by reaction with photochemically-produced hydroxyl radicals, with an estimated atmospheric lifetime of about 0.5 to 2 days. Xylenes' susceptibility to photochemical oxidation in the troposphere is to the extent that they may contribute to photochemical smog formation.

According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere and from its vapour pressure, p-xylene, is expected to exist solely as a vapour in the ambient atmosphere. Vapour-phase p-xylene 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 16 hours. A half-life of 1.0 hr in summer and 10 hr in winter was measured for the reaction of p-xylene with photochemically-produced hydroxyl radicals. p-Xylene has a moderately high photochemical reactivity under smog conditions, higher than the other xylene isomers, with loss rates varying from 9-42% per hr. The photooxidation of p-xylene results in the production of carbon monoxide, formaldehyde, glyoxal, methylglyoxal, 3-methylbenzylnitrate, m-tolualdehyde, 4-nitro-3-xylene, 5-nitro-3-xylene, 2,6-dimethyl-p-benzoquinone, 2,4-dimethylphenol, 6-nitro-2,4-dimethylphenol, 2,6-dimethylphenol, and 4-nitro-2,6-dimethylphenol.

Ecotoxicity:

for xylenes
Fish LC50 (96 h) Pimephales promelas 13.4 mg/l; Oncorhyncus mykiss 8.05 mg/l; Lepomis macrochirus 16.1 mg/l (all flow through values); Pimephales promelas 26.7 (static)
Daphnia EC50 948 h): 3.83 mg/l
Photobacterium phosphoreum EC50 (24 h): 0.0084 mg/l
Gammarus lacustris LC50 (48 h): 0.6 mg/l

For glycol ethers:

Environmental fate:

Ether groups are generally stable to hydrolysis in water under neutral conditions and ambient temperatures. OECD guideline studies indicate ready biodegradability for several glycol ethers although higher molecular weight species seem to biodegrade at a slower rate. No glycol ethers that have been tested demonstrate marked resistance to biodegradative processes. Upon release to the atmosphere by evaporation, high boiling glycol ethers are estimated to undergo photodegradation (atmospheric half lives = 2.4-2.5 hr). When released to water, glycol ethers undergo biodegradation (typically 47-92% after 8-21 days) and have a low potential for bioaccumulation (log Kow ranges from -1.73 to +0.51).

Ecotoxicity:

Aquatic toxicity data indicate that the tri- and tetra ethylene glycol ethers are "practically non-toxic" to aquatic species. No major differences are observed in the order of toxicity going from the methyl- to the butyl ethers.
Glycols exert a high oxygen demand for decomposition and once released to the environments cause the death of aquatic organisms if dissolved oxygen is depleted.

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.

Continued...

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
methyl isobutyl ketone	HIGH (Half-life = 7001 days)	LOW (Half-life = 1.9 days)
methyl ethyl ketone	LOW (Half-life = 14 days)	LOW (Half-life = 26.75 days)
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
ethylene glycol monobutyl ether	LOW (Half-life = 56 days)	LOW (Half-life = 1.37 days)
isopropanol	LOW (Half-life = 14 days)	LOW (Half-life = 3 days)
gamma-glycidoxypropyltrimethoxysilane	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
methyl isobutyl ketone	LOW (LogKOW = 1.31)
methyl ethyl ketone	LOW (LogKOW = 0.29)
xylene	MEDIUM (BCF = 740)
ethylene glycol monobutyl ether	LOW (BCF = 2.51)
isopropanol	LOW (LogKOW = 0.05)
gamma-glycidoxypropyltrimethoxysilane	LOW (LogKOW = -0.9152)

Mobility in soil

Ingredient	Mobility
methyl isobutyl ketone	LOW (KOC = 10.91)
methyl ethyl ketone	MEDIUM (KOC = 3.827)
ethylene glycol monobutyl ether	HIGH (KOC = 1)
isopropanol	HIGH (KOC = 1.06)
gamma-glycidoxypropyltrimethoxysilane	LOW (KOC = 90.22)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal

- Containers may still present a chemical hazard/ danger when empty.
- Return to supplier for reuse/ recycling if possible.

Otherwise:

- If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.
- Where possible retain label warnings and MSDS and observe all notices pertaining to the product.

Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.

A Hierarchy of Controls seems to be common - the user should investigate:

- Reduction
- Reuse
- Recycling
- Disposal (if all else fails)

This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use.

If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.

- **DO NOT** allow wash water from cleaning or process equipment to enter drains.
- It may be necessary to collect all wash water for treatment before disposal.
- In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.
- Where in doubt contact the responsible authority.
- Recycle wherever possible.
- Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified.
- Dispose of by: burial in a land-fill specifically licenced to accept chemical and / or pharmaceutical wastes or Incineration in a licenced apparatus (after admixture with suitable combustible material).
- Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

Continued...

SECTION 14 TRANSPORT INFORMATION

Labels Required

	
Marine Pollutant	NO
HAZCHEM	•3YE

Land transport (ADG)

UN number	1263
Packing group	II
UN proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound) (contains mibk and mek and xylene (mixed isomers) and ipa)
Environmental hazard	No relevant data
Transport hazard class(es)	Class: 3 Subrisk: Not Applicable
Special precautions for user	Special provisions: 163 * Limited quantity: 5 L

Air transport (ICAO-IATA / DGR)

UN number	1263
Packing group	II
UN proper shipping name	Paint (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base); Paint related material (including paint thinning or reducing compounds) (contains mibk and mek and xylene (mixed isomers) and ipa)
Environmental hazard	No relevant data
Transport hazard class(es)	ICAO/IATA Class: 3 ICAO / IATA Subrisk: Not Applicable ERG Code: 3L
Special precautions for user	Special provisions: A3 A72 A192 Cargo Only Packing Instructions: 364 Cargo Only Maximum Qty / Pack: 60 L Passenger and Cargo Packing Instructions: 353 Passenger and Cargo Maximum Qty / Pack: 5 L Passenger and Cargo Limited Quantity Packing Instructions: Y341 Passenger and Cargo Limited Maximum Qty / Pack: 1 L

Sea transport (IMDG-Code / GGVSee)

UN number	1263
Packing group	II
UN proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound) (contains mibk and mek and xylene (mixed isomers) and ipa)
Environmental hazard	Not Applicable
Transport hazard class(es)	IMDG Class: 3 IMDG Subrisk: Not Applicable
Special precautions for user	EMS Number: F-E , S-E Special provisions: 163 Limited Quantities: 5 L

Continued...

Transport in bulk according to Annex II of MARPOL 73 / 78 and the IBC code

Source	Ingredient	Pollution Category
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	methyl isobutyl ketone	Z
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	methyl ethyl ketone	Z
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	xylene	Y

SECTION 15 REGULATORY INFORMATION

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

methyl isobutyl ketone(108-10-1) is found on the following regulatory lists	"Australia Exposure Standards","International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs","Australia Inventory of Chemical Substances (AICS)","Australia Hazardous Substances Information System - Consolidated Lists"
methyl ethyl ketone(78-93-3) is found on the following regulatory lists	"Australia Exposure Standards","Australia Inventory of Chemical Substances (AICS)","Australia Hazardous Substances Information System - Consolidated Lists"
xylene(1330-20-7) is found on the following regulatory lists	"Australia Exposure Standards","International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs","Australia Inventory of Chemical Substances (AICS)","Australia Hazardous Substances Information System - Consolidated Lists"
aromatic hydrocarbons, C9-11(70693-06-0) is found on the following regulatory lists	"Australia Inventory of Chemical Substances (AICS)"
ethylene glycol monobutyl ether(111-76-2) is found on the following regulatory lists	"Australia Exposure Standards","International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs","Australia Inventory of Chemical Substances (AICS)","Australia Hazardous Substances Information System - Consolidated Lists"
isopropanol(67-63-0) is found on the following regulatory lists	"Australia Exposure Standards","International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs","Australia Inventory of Chemical Substances (AICS)","Australia Hazardous Substances Information System - Consolidated Lists"
gamma-glycidoxypropyltrimethoxysilane(2530-83-8) is found on the following regulatory lists	"Australia Exposure Standards","Australia Inventory of Chemical Substances (AICS)","Australia Hazardous Substances Information System - Consolidated Lists"

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	N (aromatic hydrocarbons, C9-11)
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	Y
Legend:	*Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 OTHER INFORMATION

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.
A list of reference resources used to assist the committee may be found at:

Continued...

www.chemwatch.net/references

The (M)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.

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