8310 Conformal Coating Stripper

Mektronics

Chemwatch Hazard Alert Code: **3**

Version No: **5.8**
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	8310 Conformal Coating Stripper
Synonyms	Part #: 8310-100ML, 8310-55ML,SDS Code: 8310–Liquid
Other means of identification	Not Available

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

Relevant identified uses	Removing conformal coatings and protective coatings

Details of the supplier of the safety data sheet

Registered company name	Mektronics	MG Chemicals (Head office)
Address	Unit 3 8 Bonz Place, Seven Hills NSW 2147 Australia	9347 - 193 Street Surrey V4N 4E7 British Columbia Canada
Telephone	1300 788 701	+(1) 800-201-8822
Fax	1300 722 004	+(1) 800-708-9888
Website	www.mektronics.com.au	www.mgchemicals.com
Email	sales@mektronics.com.au	Info@mgchemicals.com

Emergency telephone number

Association / Organisation	CHEMTREC Australia	Not Available
Emergency telephone numbers	+(61) 2-9037-2994	Not Available
Other emergency telephone numbers	+(1) 703-527-3887	Not Available

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

HAZARDOUS CHEMICAL. NON-DANGEROUS GOODS. According to the WHS Regulations and the ADG Code.
COMBUSTIBLE LIQUID, regulated for storage purposes only

Poisons Schedule	Not Applicable
Classification [1]	Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Specific target organ toxicity - single exposure Category 3, Skin Sensitizer Category 1, Acute Aquatic Hazard Category 2, Chronic Aquatic Hazard Category 3, Flammable Liquid Category 4, Reproductive Toxicity Category 1A
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)

H315	Causes skin irritation.
H319	Causes serious eye irritation.
H335	May cause respiratory irritation.
H317	May cause an allergic skin reaction.
H412	Harmful to aquatic life with long lasting effects.

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H227	Combustible liquid
H360	May damage fertility or the unborn child.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat/sparks/open flames/hot surfaces. - No smoking.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/attention.
P362	Take off contaminated clothing and wash before reuse.
P363	Wash contaminated clothing before reuse.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam for extinction.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

P501	Dispose of contents/container in accordance with local regulations.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
872-50-4	30-60	N-methyl-2-pyrrolidone
1119-40-0	10-20	dimethyl glutarate
106-65-0	3-7	dimethyl succinate
627-93-0	1-5	dimethyl adipate
5989-27-5	1-5	d-limonene

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	▸ Immediately give a glass of water. ▸ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

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

Continued...

difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.
Treat symptomatically.
for simple esters:

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 (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.
- Give activated charcoal.

ADVANCED TREATMENT

- Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- Positive-pressure ventilation using a bag-valve mask might be of use.
- Monitor and treat, where necessary, for arrhythmias.
- Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- 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.

NS: Non-specific determinant - observed following exposure to other materials.

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

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

Special hazards arising from the substrate or mixture

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

Advice for firefighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Use water delivered as a fine spray to control fire and cool adjacent area. ▸ Avoid spraying water onto liquid pools. ▸ **DO NOT** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	▸ Combustible. ▸ Slight fire hazard when exposed to heat or flame. ▸ Heating may cause expansion or decomposition leading to violent rupture of containers. ▸ On combustion, may emit toxic fumes of carbon monoxide (CO). ▸ May emit acrid smoke.

Continued...

- Mists containing combustible materials may be explosive.

Combustion products include; carbon dioxide (CO_2) aldehydes nitrogen oxides (NOx) other pyrolysis products typical of burning organic materialMay emit poisonous fumes.May emit corrosive fumes. **WARNING**: Long standing in contact with air and light may result in the formationof potentially explosive peroxides.

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Minor Spills

Environmental hazard - contain spillage.
Slippery when spilt.

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

Major Spills

Environmental hazard - contain spillage.
Chemical Class: ester and ethers
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 - particulate	3	shovel	shovel	R, W, P, DGC
wood fiber - pillow	3	throw	pitchfork	R, P, DGC, RT
treated wood fiber - pillow	3	throw	pitchfork	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	W, SS, DGC
expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC
wood fiber - particulate	4	blower	skiploader	R, W, P, DGC

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

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

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling

- Avoid all personal contact, including inhalation.
- Wear protective clothing when risk of exposure occurs.
- Use in a well-ventilated area.
- Prevent concentration in hollows and sumps.
- **DO NOT** enter confined spaces until atmosphere has been checked.
- Avoid smoking, naked lights or ignition sources.

Continued...

	▸ Avoid contact with incompatible materials. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Keep containers securely sealed when not in use. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions. ▸ **DO NOT** allow clothing wet with material to stay in contact with skin
Other information	▸ Store in original containers. ▸ Keep containers securely sealed. ▸ No smoking, naked lights or ignition sources. ▸ Store in a cool, dry, well-ventilated area. ▸ Store away from incompatible materials and foodstuff containers. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Metal can or drum ▸ Packaging as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	Dibasic esters: ▸ react with strong oxidisers with risk of fire and/ or explosion ▸ are incompatible with strong acids, nitrates d-Limonene: ▸ forms unstable peroxides in storage, unless inhibited; may polymerise ▸ reacts with strong oxidisers and may explode or combust ▸ is incompatible with strong acids, including acidic clays, peroxides, halogens, vinyl chloride and iodine pentafluoride ▸ flow or agitation may generate electrostatic charges due to low conductivity ▸ Esters react with acids to liberate heat along with alcohols and acids. ▸ Strong oxidising acids may cause a vigorous reaction with esters that is sufficiently exothermic to ignite the reaction products. ▸ Heat is also generated by the interaction of esters with caustic solutions. ▸ Flammable hydrogen is generated by mixing esters with alkali metals and hydrides. ▸ Esters may be incompatible with aliphatic amines and nitrates.

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	N-methyl-2-pyrrolidone	1-Methyl-2-pyrrolidone	103 mg/m3 / 25 ppm	309 mg/m3 / 75 ppm	Not Available	Sk

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
N-methyl-2-pyrrolidone	Methyl 2-pyrrolidinone, 1-; (N-Methylpyrrolidone)	10 ppm	10 ppm	10 ppm
N-methyl-2-pyrrolidone	Petroleum 50 thinner; (Paint thinner)	5.5 ppm	61 ppm	370 ppm
dimethyl succinate	Butanedioic acid, dimethyl ester; (Succinic acid, dimethyl ester)	2.5 ppm	28 ppm	170 ppm
d-limonene	Limonene, d-	20 ppm	20 ppm	160 ppm

Ingredient	Original IDLH	Revised IDLH
N-methyl-2-pyrrolidone	Not Available	Not Available
dimethyl glutarate	Not Available	Not Available
dimethyl succinate	Not Available	Not Available
dimethyl adipate	Not Available	Not Available
d-limonene	Not Available	Not Available

MATERIAL DATA

for N-methyl-2-pyrrolidone (NMP):
Reports of skin and eye irritation and chronic headaches have been reported in workers exposed to 1-methyl-2-pyrrolidone. The Australian ES is based on a 10-fold uncertainty factor of the no-observable-adverse-effect level (NOAEL) of 24 ppm where adverse respiratory effects were observed in a 4-week inhalation study in rats.
for d-Limonene:
CEL TWA: 30 ppm, 165.6 mg/m3 (compare WEEL-TWA*)
(CEL = Chemwatch Exposure Limit)
A Workplace Environmental Exposure Level* has been established by AIHA (American Industrial Hygiene Association) who have produced the following rationale:
d-Limonene is not acutely toxic. In its pure form it is not a sensitiser but is irritating to the skin. Although there is clear evidence of carcinogenicity in male rats, the effect has been attributed to an alpha-2u-globin (a2u-G) renal toxicity which is both species and gender specific. Humans do not synthesise a2u-G, and metabolism studies indicate that 75% to 95% of d-limonene is excreted in 2-3 days with different metabolites identified between humans and rats. In a 2-year study, liver effects were noted in male mice at 500 mg/kg and reduced survival was noted in female rats at 600 mg/kg. The no observable effect levels (NOELs) were 250 and 300 mg/kg, respectively. A WEEL of 30 ppm is recommended to protect against these effects.

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.

Continued...

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.

Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection.
Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.
An approved self contained breathing apparatus (SCBA) may be required in some situations.
Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying 'escape' velocities which, in turn, determine the 'capture velocities' of fresh circulating air required to effectively remove the contaminant.

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

Within each range the appropriate value depends on:

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

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

Personal protection

Eye and face protection

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

Skin protection

See Hand protection below

Hands/feet protection

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

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.
- P.V.C. apron.
- Barrier cream.
- Skin cleansing cream.

Continued...

	▸ Eye wash unit.
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:
8310 Conformal Coating Stripper

Material	CPI
PVA	B
BUTYL	C
NATURAL RUBBER	C
NITRILE	C
PE/EVAL/PE	C
VITON	C

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

Respiratory protection

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 5 x ES	AK-AUS / Class 1 P2	-	AK-PAPR-AUS / Class 1 P2
up to 25 x ES	Air-line*	AK-2 P2	AK-PAPR-2 P2
up to 50 x ES	-	AK-3 P2	-
50+ x ES	-	Air-line**	-

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

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Clear Amber

Physical state	Liquid	**Relative density (Water = 1)**	1.02
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	93	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	67	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Combustible.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	10	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	1	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water (g/L)	Partly miscible	**pH as a solution (1%)**	6-7
Vapour density (Air = 1)	>2	**VOC g/L**	Not Available

SECTION 10 STABILITY AND REACTIVITY

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

SECTION 11 TOXICOLOGICAL INFORMATION

Continued...

Information on toxicological effects

Inhaled	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. Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.
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 has **NOT** been classified by EC Directives or other classification systems as 'harmful by ingestion'. This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. The material may accentuate any pre-existing dermatitis condition Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	Evidence exists, or practical experience predicts, that the material may cause 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. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur. Direct contact with the liquid N-methyl-2-pyrrolidone (NMP) may produce painful burning or stinging of the eyes and lids, watering and inflammation of the conjunctiva and temporary corneal clouding.
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in impaired fertility on the basis of: - clear evidence in animal studies of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects but which is not a secondary non-specific consequence of other toxic effects. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of: - clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. In the presence of air, a number of common flavour and fragrance chemicals can form peroxides surprisingly fast. Antioxidants can in most cases minimise the oxidation. Fragrance terpenes are generally easily oxidised in air. Non-oxidised limonene, linalool and caryophyllene turned out to be very weak sensitizers, however after oxidation limonene hydroperoxide and linalool hydroperoxide are strong sensitizers. Of the patients tested 2.6% showed positive reaction to oxidised limonene, 1.3% to oxidised linalool, 1.1% to linalool hydroperoxide, 0.5% to oxidised caryophyllene, while testing with caryophyllene oxide and oxidised myrcene resulted in few positive patch tests. 2/3 of the patients reacting positive to oxidised terpenes had fragrance related contact allergy and/or positive history for adverse reactions to fragrances. As well as the hydroperoxides produced by linalol, limonene and delta-3-carene other oxidation and resinification effects progressively causes other fairly major changes in essential oil quality over time. Autoxidation of fragrance terpenes contributes greatly to fragrance allergy, which emphasizes the need of testing with compounds that patients are actually exposed to and not only with the ingredients originally applied in commercial formulations. Hydroperoxides of d-limonene are potent contact allergens when studied in guinea pigs. They may result when d-limonene is unstabilised against oxidation, or upon prolonged standing at room temperature and/ or upon exposure to light, or when stabiliser levels diminish. The two major hydroperoxides in auto-oxidised d-limonene, are cis- and trans- limonene-2-hydroperoxide (2-hydroperoxy-p-mentha-6,8-diene). In photo-oxidised d-limonene, they represent a minor fraction. Hydroperoxides may bind to proteins of the skin to make antigens either via a radical mechanism or after reactions to give epoxides. The cross-reactivity between the epoxide limonene-1,2-oxide, a potent contact allergen, and the hydroperoxides is NOT significant, indicating different mechanisms of sensitisation. d-Limonene was considered to be weakly carcinogenic for the mouse fore-stomach epithelium, but not tumour producing. In 13-week and 2-year gavage-studies, male rats showed a range of compound-related kidney lesions including exacerbation of age-related nephropathy, mineralisation in the renal medulla, hyperplasia of the transitional epithelium overlying the renal papilla and proliferation of the renal tubular epithelium. Neoplasms were believed to be caused by progression to tubular cell hyperplasia to tubular cell adenomas and, with increasing size, to adenocarcinomas or carcinomas. The similarity of the nephrotoxicity caused by trichloroethylene and N-(4'-fluoro-4-biphenyl)acetamide, tris(2,3-dibromopropyl)phosphate in rats and the species specific nature of the response suggests that degeneration and necrosis of convoluted tubules may be associated with the accumulation of alpha-2u-globin (a2u-G). Since a2u-G is a species and gender-specific protein that is causal for both the cytotoxic and carcinogenic response in male rats, extrapolation of d-limonene carcinogenicity data from rat studies to other species (including humans) is probably not warranted. Humans do not synthesise a2u-G; they do however produce other related low molecular weight proteins capable of binding chemicals that cause a2u-G nephropathy in rats but this does not necessarily connote human risk. The Risk Assessment Forum of the USA EPA concluded; ▸ Male renal rat tumours arising as a result of a process involving a2u-G accumulation do not contribute to the qualitative weight-of-evidence that the chemical poses a human carcinogenic hazard. Such tumours are included in dose-response extrapolations for the estimation of human carcinogenic risk. ▸ If the chemical induces a2u-G accumulation in male rats, the associated nephropathy is not to be used as an end-point for determining non-carcinogenic hazard. Peroxidisable terpenes and terpenoids should only be used when the level of peroxides is kept to the lowest practicable level, for instance by adding antioxidants at the time of production. Such products should have a peroxide value of less than 10 millimoles peroxide per liter. This requirement is based on the published

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literature mentioning sensitising properties when containing peroxides.
Chronic solvent inhalation exposures may result in nervous system impairment and liver and blood changes. [PATTYS]
Long-term exposure to methanol vapour, at concentrations exceeding 3000 ppm, may produce cumulative effects characterised by gastrointestinal disturbances (nausea, vomiting), headache, ringing in the ears, insomnia, trembling, unsteady gait, vertigo, conjunctivitis and clouded or double vision. Liver and/or kidney injury may also result. Some individuals show severe eye damage following prolonged exposure to 800 ppm of the vapour.

8310 Conformal Coating Stripper

TOXICITY	IRRITATION
Not Available	Not Available

N-methyl-2-pyrrolidone

TOXICITY	IRRITATION
dermal (rat) LD50: >5000 mg/kg[1]	*[Manufacturer]
Inhalation (rat) LC50: 8300 ppm/4H[2]	Eye (rabbit): 100 mg - moderate
Oral (rat) LD50: 3914 mg/kg][2]	

dimethyl glutarate

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	[Manuf. DU]
Oral (rat) LD50: >2000 mg/kg[1]	Eye (rabbit): Irritant
	Skin (human): Irritant

dimethyl succinate

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	Nil reported
Oral (rat) LD50: >5000 mg/kgd[2]	

dimethyl adipate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >2500 mg/kg[2]	Eye (rabbit): Irritant
Inhalation (rat) LC50: 10.7 mg/l/1h[2]	Skin (human): SEVERE
Inhalation (rat) LC50: 11 mg/l/4h[2]	
Oral (rat) LD50: 8191 mg/kgt[2]	

d-limonene

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg[2]	Nil reported
Oral (rat) LD50: >2000 mg/kg[1]	Skin (rabbit): 500mg/24h moderate

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

8310 Conformal Coating Stripper

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.
The following information refers to contact allergens as a group and may not be specific to this product.
Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.
d-Limonene is readily absorbed by inhalation and ingestion. Dermal absorption is reported to be lower than by the inhalation route. d-Limonene is rapidly distributed to different tissues in the body, readily metabolised and eliminated primarily through the urine.
Limonene exhibits low acute toxicity by all three routes in animals. Limonene is a skin irritant in both experimental animals and humans. Limited data are available on the potential to cause eye and respiratory irritation. Autooxidised products of d-limonene have the potential to be skin sensitisers. Limited data are available in humans on the potential to cause respiratory sensitisation. Autooxidation of limonene occurs readily in the presence of light and air forming a variety of oxygenated monocyclic terpenes. Risk of skin sensitisation is high in situations where contact with oxidation products of limonene occurs.
Renal tumours induced by limonene in male rats is though to be sex and species specific and are not considered relevant to humans. Repeated exposure affects the amount and activity of liver enzymes, liver weight, blood cholesterol levels and bile flow in animals. Increase in liver weight is considered a physiological adaption as no toxic effects on the liver have been reported. From available data it is not possible to identify an NOAEL for these effects. Limonene is neither genotoxic or teratogenic nor toxic to the reproductive system.

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N-METHYL-2-PYRROLIDONE	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. for N-methyl-2-pyrrolidone (NMP): **Acute toxicity:** In rats, NMP is absorbed rapidly after inhalation, oral, and dermal administration, distributed throughout the organism, and eliminated mainly by hydroxylation to polar compounds, which are excreted via urine. About 80% of the administered dose is excreted as NMP and NMP metabolites within 24 h. A probably dose-dependent yellow coloration of the urine in rodents is observed. The major metabolite is 5-hydroxy-*N*-methyl-2-pyrrolidone. Studies in humans show comparable results. Dermal penetration through human skin has been shown to be very rapid. NMP is rapidly biotransformed by hydroxylation to 5-hydroxy-*N*-methyl-2-pyrrolidone, which is further oxidized to *N*-methylsuccinimide; this intermediate is further hydroxylated to 2-hydroxy-*N*-methylsuccinimide. These metabolites are all colourless. The excreted amounts of NMP metabolites in the urine after inhalation or oral intake represented about 100% and 65% of the administered doses, respectively. NMP has a low potential for skin irritation and a moderate potential for eye irritation in rabbits. Repeated daily doses of 450 mg/kg body weight administered to the skin caused painful and severe haemorrhage and eschar formation in rabbits. These adverse effects have not been seen in workers occupationally exposed to pure NMP, but they have been observed after dermal exposure to NMP used in cleaning processes. No sensitisation potential has been observed. In acute toxicity studies in rodents, NMP showed low toxicity. Uptake of oral, dermal, or inhaled acutely toxic doses causes functional disturbances and depressions in the central nervous system. Local irritation effects were observed in the respiratory tract when NMP was inhaled and in the pyloric and gastrointestinal tracts after oral administration. In humans, there was no irritative effect in the respiratory system after an 8-h exposure to 50 mg/m3. **Repeat dose toxicity:** There is no clear toxicity profile of NMP after multiple administration. In a 28-day dietary study in rats, a compound-related decrease in body weight gain was observed in males at 1234 mg/kg body weight and in females at 2268 mg/kg body weight. Testicular degeneration and atrophy in males and thymic atrophy in females were observed at these dose levels. The no-observed-adverse-effect level (NOAEL) was 429 mg/kg body weight in males and 1548 mg/kg body weight in females. In a 28-day intubation study in rats, a dose-dependent increase in relative liver and kidney weights and a decrease in lymphocyte count in both sexes were observed at 1028 mg/kg body weight. The NOAEL in this study was 514 mg/kg body weight. In another rat study, daily dietary intake for 90 days caused decreased body weights at doses of 433 and 565 mg/kg body weight in males and females, respectively. There were also neurobehavioural effects at these dose levels. The NOAELs in males and females were 169 and 217 mg/kg body weight, respectively. The toxicity profile after exposure to airborne NMP depends strongly on the ratio of vapour to aerosol and on the area of exposure (i.e., head-only or whole-body exposure). Because of higher skin absorption for the aerosol, uptake is higher in animals exposed to aerosol than in those exposed to vapour at similar concentrations. Studies in female rats exposed head only to 1000 mg/m3 showed only minor nasal irritation, but massive mortality and severe effects on major organs were observed when the females were whole-body exposed to the same concentration of coarse droplets at high relative humidity. Several studies in rats following repeated exposure to NMP at concentrations between 100 and 1000 mg/m3 have shown systemic toxicity effects at the lower dose levels. In most of the studies, the effects were not observed after a 4-week observation period. In rats, exposure to 3000 mg NMP/m3 (head only) for 6 h/day, 5 days/week, for 13 weeks caused a decrease in body weight gain, an increase in erythrocytes, haemoglobin, haematocrit, and mean corpuscular volume, decreased absolute testis weight, and cell loss in the germinal epithelium of the testes. The NOAEL was 500 mg/m3. There are no data in humans after repeated-dose exposure. **Carcinogenicity:** NMP did not show any clear evidence for carcinogenicity in rats exposed to concentrations up to 400 mg/m3 in a long-term inhalation study. **Genotoxicity:** The mutagenic potential of NMP is weak. Only a slight increase in the number of revertants was observed when tested in a *Salmonella* assay with base-pair substitution strains. NMP has been shown to induce aneuploidy in yeast *Saccharomyces cerevisiae* cells. No investigations regarding mutagenicity in humans were available. **Reproductive toxicity:** In a two-generation reproduction study in rats, whole-body exposure of both males and females to 478 mg/m3 of NMP vapour for 6 h/day, 7 days/week, for a minimum of 100 days (pre-mating, mating, gestation, and lactation periods) resulted in a 7% decrease in fetal weight in the F1 offspring. A 4-11% transient, non-dose-dependent decrease was observed in the average pup weight at all exposure levels tested (41, 206, and 478 mg/m3). **Developmental toxicity:** When NMP was administered dermally, developmental toxicity was registered in rats at 750 mg/kg body weight. The observed effects were increased preimplantation losses, decreased fetal weights, and delayed ossification. The NOAEL for both developmental effects and maternal toxicity (decreased body weight gain) was 237 mg/kg body weight. Inhalation studies in rats (whole-body exposure) demonstrated developmental toxicity as increased preimplantation loss without significant effect on implantation rate or number of live fetuses at 680 mg/m3 and behavioural developmental toxicity at 622 mg/m3. In an inhalation study (whole-body exposure), the NOAEL for maternal effects was 100 mg/m3, and the NOAEL for developmental effects was 360 mg/m3. A tolerable inhalation concentration, 0.3 mg/m3, based on mortality and organ damage, is expected to be protective against any possible reproductive toxicity. Similarly, an oral tolerable intake of 0.6 mg/kg body weight per day, based on a 90-day study, is expected to provide adequate protection against possible reproductive effects. Because of non-existent data on the exposure of the general population and very limited information on occupational exposure, no meaningful risk characterisation can be performed
DIMETHYL GLUTARATE	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 family of dibasic (methyl) esters (DBEs) comprise dimethyl succinate (DMS, CAS No. 106-65-0), dimethyl glutarate (DMG, CASNo. 1119-40-0), and dimethyl adipate (DMA, CAS No. 627-93-0), and their mixtureDBE (CAS No. 95481-62-2). A crude dibasic ester mixture is distilled to produceDMS, DMG, and DMA and three other fractions that are mixtures of these estersgenerally composed of 10-25, 55-65, and 15-25% DMA, DMG, and DMS, respectively. The three discrete compounds are all short four-to six-carbonstraight-chain dicarboxylic acid dimethyl esters differing incrementally by onecarbon atom. The four members of the category produce similar levels of acuteand repeated-dose toxicity in experimental animals DBEs have very low acute oral toxicities with LD50 s in ratsgenerally > 5,000 mg/kg (with two exceptions reported as >500 and<5,000 mg/kg b.wt. for DBE (the mixture) and DMS By skin absorption, DBEs have a low order of acute toxicity torabbits with dermal LD50s of 3,000 mg/kg . Based upon the most recent GLPstudies DBEs are not considered to produce primary dermal irritation as definedin EPA Guidelines . Earlier studies did show moderate irritation in one of sixrabbits, but these results were not repeated in later studies. All four DBEmaterials are considered to produce eye irritation as defined by EPAGuidelines. Mild to moderate irritation involving the cornea was observed inrabbits with recovery by 7 days. DBEs are not skin sensitisers, and arenot harmful via skin or inhalation exposures. DBE is slightly toxic byinhalation with 1-and 4-hour LC50s in rats of > 10.7 and > 11 mg/L,respectively. In subchronic inhalation studies with all four DBEs, degenerationof the olfactory epithelium of the nose was observed. This change in the nasaltissues is related to enzymatic hydrolysis of DBE within the nasal cavity.However, risk to human nasal tissue due to DBE toxicity is likely to be reducedwhen compared to rats since DBEs are hydrolysed more slowly in humans. Noinformation is available on the carcinogenic potential of DBEs. A range ofstudies with DMS, DMG, DMA and DBE did not produce genetic damage in animals orbacterial cell cultures. DBE was positive in one study with cultured mammaliancells, but the positive findings were not apparent when the assay was repeated.Testing in rats indicates DBEs are not developmental or reproductive toxicants.
DIMETHYL SUCCINATE	The family of dibasic (methyl) esters (DBEs) comprise dimethyl succinate (DMS, CAS No. 106-65-0), dimethyl glutarate (DMG, CASNo. 1119-40-0), and dimethyl adipate (DMA, CAS No. 627-93-0), and their mixtureDBE (CAS No. 95481-62-2). A crude dibasic ester mixture is distilled to produceDMS, DMG, and DMA and three other fractions that are mixtures of these estersgenerally composed of 10-25, 55-65, and 15-25% DMA, DMG, and DMS, respectively. The three discrete compounds are all short four-to six-carbonstraight-chain dicarboxylic acid dimethyl esters differing incrementally by onecarbon atom. The four members of the category produce similar levels of acuteand repeated-dose toxicity in experimental animals DBEs have very low acute oral toxicities with LD50 s in ratsgenerally > 5,000 mg/kg (with two exceptions reported as >500 and<5,000 mg/kg b.wt. for DBE (the mixture) and DMS By skin absorption, DBEs have a low order of acute toxicity torabbits with dermal LD50s of 3,000 mg/kg . Based upon the most recent GLPstudies DBEs are not

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DIMETHYL ADIPATE	The family of dibasic (methyl) esters (DBEs) comprise dimethyl succinate (DMS, CAS No. 106-65-0), dimethyl glutarate (DMG, CASNo. 1119-40-0), and dimethyl adipate (DMA, CAS No. 627-93-0), and their mixtureDBE (CAS No. 95481-62-2). A crude dibasic ester mixture is distilled to produceDMS, DMG, and DMA and three other fractions that are mixtures of these estersgenerally composed of 10-25, 55-65, and 15-25% DMA, DMG, and DMS, respectively. The three discrete compounds are all short four-to six-carbonstraight-chain dicarboxylic acid dimethyl esters differing incrementally by onecarbon atom. The four members of the category produce similar levels of acuteand repeated-dose toxicity in experimental animals DBEs have very low acute oral toxicities with LD50 s in ratsgenerally > 5,000 mg/kg (with two exceptions reported as >500 and<5,000 mg/kg b.wt. for DBE (the mixture) and DMS By skin absorption, DBEs have a low order of acute toxicity torabbits with dermal LD50s of 3,000 mg/kg . Based upon the most recent GLPstudies DBEs are not considered to produce primary dermal irritation as definedin EPA Guidelines . Earlier studies did show moderate irritation in one of sixrabbits, but these results were not repeated in later studies. All four DBEmaterials are considered to produce eye irritation as defined by EPAGuidelines. Mild to moderate irritation involving the cornea was observed inrabbits with recovery by 7 days. DBEs are not skin sensitisers, and arenot harmful via skin or inhalation exposures. DBE is slightly toxic byinhalation with 1-and 4-hour LC50s in rats of > 10.7 and > 11 mg/L,respectively. In subchronic inhalation studies with all four DBEs, degenerationof the olfactory epithelium of the nose was observed. This change in the nasaltissues is related to enzymatic hydrolysis of DBE within the nasal cavity.However, risk to human nasal tissue due to DBE toxicity is likely to be reducedwhen compared to rats since DBEs are hydrolysed more slowly in humans. Noinformation is available on the carcinogenic potential of DBEs. A range ofstudies with DMS, DMG, DMA and DBE did not produce genetic damage in animals orbacterial cell cultures. DBE was positive in one study with cultured mammaliancells, but the positive findings were not apparent when the assay was repeated.Testing in rats indicates DBEs are not developmental or reproductive toxicants. 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 produce severe 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) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.
D-LIMONENE	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested. d-Limonene is readily absorbed by inhalation and ingestion. Dermal absorption is reported to be lower than by the inhalation route. d-Limonene is rapidly distributed to different tissues in the body, readily metabolised and eliminated primarily through the urine. Limonene exhibits low acute toxicity by all three routes in animals. Limonene is a skin irritant in both experimental animals and humans. Limited data are available on the potential to cause eye and respiratory irritation. Autooxidised products of d-limonene have the potential to be skin sensitisers. Limited data are available in humans on the potential to cause respiratory sensitisation. Autooxidation of limonene occurs readily in the presence of light and air forming a variety of oxygenated monocyclic terpenes. Risk of skin sensitisation is high in situations where contact with oxidation products of limonene occurs. Renal tumours induced by limonene in male rats is though to be sex and species specific and are not considered relevant to humans. Repeated exposure affects the amount and activity of liver enzymes, liver weight, blood cholesterol levels and bile flow in animals. Increase in liver weight is considered a physiological adaption as no toxic effects on the liver have been reported. From available data it is not possible to identify an NOAEL for these effects. Limonene is neither genotoxic or teratogenic nor toxic to the reproductive system. Adverse reactions to fragrances in perfumes and in fragrancedcosmetic products include allergic contact dermatitis, irritant contactdermatitis, photosensitivity, immediate contact reactions (contact urticaria),and pigmented contact dermatitis. Airborne and connubial contact dermatitisoccur. Intolerance to perfumes, by inhalation, may occur if the perfumecontains a sensitising principal. Symptoms may vary from general illness,coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea,acute respiratory illness, hayfever, and other respiratory diseases (includingasthma). Perfumes can induce hyper-reactivity of the respiratory tract withoutproducing an IgE-mediated allergy or demonstrable respiratory obstruction. Thiswas shown by placebo-controlled challenges of nine patients to 'perfumemix'. The same patients were also subject to perfume provocation, with orwithout a carbon filter mask, to ascertain whether breathing through a filterwith active carbon would prevent symptoms. The patients breathed through themouth, during the provocations, as a nose clamp was used to prevent nasalinhalation. The patient's earlier symptoms were verified; breathing through thecarbon filter had no protective effect. The symptoms were not transmitted viathe olfactory nerve but they may have been induced by trigeminal reflex via therespiratory tract or by the eyes. Cases of occupational asthma induced by perfume substances such asisoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to givepersistent symptoms even though the exposure is below occupational exposurelimits. Inhalation intolerance has also been produced in animals. Theemissions of five fragrance products, for one hour, produced various combinationsof sensory irritation, pulmonary irritation, decreases in expiratory airflowvelocity as well as alterations of the functional observational batteryindicative of neurotoxicity in mice. Neurotoxicity was found to be more severeafter mice were repeatedly exposed to the fragrance products, being four brandsof cologne and one brand of toilet water. Contact allergy to fragrances is relatively common, affecting 1 to3% of the general population, based on limited testing with eight commonfragrance allergens and about 16 % of patients patch tested for suspectedallergic contact dermatitis. Contact allergy to fragrance ingredients occurs when an individualhas been exposed, on the skin, to a suffcient degree of fragrance contactallergens. Contact allergy is a life-long, specifically altered reactivity inthe immune system. This means that once contact allergy is developed, cells inthe immune system will be present which can recognise and react towards theallergen. As a consequence, symptoms, i.e. allergic contact dermatitis, mayoccur upon re-exposure to the fragrance allergen(s) in question. Allergiccontact dermatitis is an inflammatory skin disease characterised by erythema,swelling and vesicles in the acute phase. If exposure continues it may developinto a chronic condition with scaling and painful fissures of the skin.Allergic contact dermatitis to fragrance ingredients is most often caused bycosmetic products and usually involves the face and/or hands. It may affectfitness for work and the quality of life of the individual. Fragrance contactallergy has long been recognised as a frequent and potentially disablingproblem. Prevention is possible as it is an environmental disease and if theenvironment is modified (e.g. by reduced use concentrations of allergens), thedisease frequency and severity will decrease Fragrance contact allergy ismostly non-occupational and related to the personal use of cosmetic products.Allergic contact dermatitis can be severe and widespread, with a significant impairmentof quality of life and potential consequences for fitness for work. Thus,prevention of contact sensitisation to fragrances, both in terms of primaryprevention (avoiding sensitisation) and secondary prevention (avoiding relapsesof allergic contact dermatitis in those already sensitised), is an importantobjective of public health risk management measure. **Hands:** Contact sensitisation may be the primary cause of hand eczema, ormay be a complication of irritant or atopic hand eczema. The number of positivepatch tests has been reported to correlate with the duration of hand eczema,indicating that long-standing hand eczema may often be complicated bysensitisation .Fragrance allergy may be a relevant problem in patients withhand eczema; perfumes are present in consumer products to which their hands areexposed. A significant relationship between hand eczema and fragrance contactallergy has been found in some studies based on patients investigated forcontact allergy. However, hand eczema is a multi-factorial disease and theclinical significance of fragrance contact allergy in (severe) chronic handeczema may not be clear. **Axillae Bilateral axillary** (underarm) dermatitismay be caused by perfume in deodorants and, if the reaction is severe, it may spreaddown the arms and to other areas of the body. In individuals who consulted adermatologist, a history of such first-time symptoms was significantly relatedto the later diagnosis of perfume allergy. **Face** Facial eczema is an important manifestation of fragrance allergyfrom the use of cosmetic products (16). In men, after-shave products can causean

Continued...

eczematous eruption of the beard area and the adjacent part of the neck andmen using wet shaving as opposed to dry have been shown to have an increased riskof being fragrance allergic.
Irritant reactions (including contact urticaria): Irritanteffects of some individual fragrance ingredients, e.g. citral are known.Irritant contact dermatitis from perfumes is believed to be common, but thereare no existing investigations to substantiate this, Many more people complainabout intolerance or rashes to perfumes/perfumed products than are shown to beallergic by testing. This may be due to irritant effects or inadequatediagnostic procedures. Fragrances may cause a dose-related contact urticaria ofthe non-immunological type (irritant contact urticaria). Cinnamal, cinnamicalcohol, and Myroxylon pereirae are well recognised causes of contacturticaria, but others, including menthol, vanillin and benzaldehyde have alsobeen reported . The reactions to Myroxylon pereirae may be due to cinnamates. Arelationship to delayed contact hypersensitivity was suggested , but nosignificant difference was found between a fragrance-allergic group and acontrol group in the frequency of immediate reactions to fragrance ingredientsin keeping with a nonimmunological basis for the reactions seen.
Pigmentary anomalies: The term "pigmentedcosmetic dermatitis" was introduced in 1973 for what had previously been knownas melanosis faciei feminae when the mechanism (type IV allergy) and causativeallergens were clarified.. It refers to increased pigmentation, usually on theface/neck, often following sub-clinical contact dermatitis. Many cosmeticingredients were patch tested at non-irritant concentrations and statisticalevaluation showed that a number of fragrance ingredients were associated:jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate,hydroxycitronellal, sandalwood oil, geraniol, geranium oil.
Photo-reactions Musk ambretteproduced a considerable number of allergic photocontact reactions (in whichUV-light is required) in the 1970s and was later banned from use in the EU.Nowadays, photoallergic contact dermatitis is uncommon . Furocoumarins(psoralens) in some plant-derived fragrance ingredients caused phototoxicreactions with erythema followed by hyperpigmentation resulting in Berloquedermatitis. There are now limits for the amount of furocoumarins in fragranceproducts. Phototoxic reactions still occur but are rare.
General/respiratory: Fragrances arevolatile and therefore, in addition to skin exposure, a perfume also exposesthe eyes and naso-respiratory tract. It is estimated that 2-4% of the adultpopulation is affected by respiratory or eye symptoms by such an exposure. Itis known that exposure to fragrances may exacerbate pre-existing asthma .Asthma-like symptoms can be provoked by sensory mechanisms. In anepidemiological investigation, a significant association was found betweenrespiratory complaints related to fragrances and contact allergy to fragranceingredients, in addition to hand eczema, which were independent risk factors ina multivariate analysis.
Fragrance allergens act as haptens, i.e. low molecular weightchemicals that are immunogenic only when attached to a carrier protein.However, not all sensitising fragrance chemicals are directly reactive, butrequire previous activation. A **prehapten** is a chemical that itself isnon- or low-sensitising, but that is transformed into a hapten outside the skinby simple chemical transformation (air oxidation, photoactivation) and withoutthe requirement of specific enzymatic systems.
In the case of prehaptens, it is possible to prevent activationoutside the body to a certain extent by different measures, e.g. prevention ofair exposure during handling and storage of the ingredients and the finalproduct, and by the addition of suitable antioxidants. When antioxidants areused, care should be taken that they will not be activated themselves andthereby form new sensitisers.
Prehaptens
Most terpenes with oxidisable allylic positions can be expected toautoxidise on air exposure due to their inherent properties. Depending on thestability of the oxidation products that are formed, a difference in thesensitisation potency of the oxidised terpenes can be seen
Autoxidation is a free radical chain reaction in which hydrogenatom abstraction in combination with addition of oxygen forms peroxyl radicals.The reaction shows selectivity for positions where stable radicals can beformed. So far, all fragrance substances that have been investigated withregard to the influence of autoxidation on the allergenic potential, includingidentification of formed oxidation products, have oxidisable allylic positionsthat are able to form hydroperoxides and/or hydrogen peroxide as primaryoxidation products upon air exposure. Once the hydroperoxides have been formedoutside the skin they form specific antigens and act as skin sensitisers.Secondary oxidation products such as aldehydes and epoxides can also beallergenic, thus further increasing the sensitisation potency of theautoxidation mixture. The process of photoactivation may also play a role, butfurther research is required to establish whether this activation route iscurrently underestimated in importance due to insufficient knowledge of thetrue haptens in this context.
It should be noted that activation of substances via air oxidationresults in various haptens that might be the same or cross-reacting with otherhaptens (allergens). The main allergens after air oxidation of linalool andlinalyl acetate are the hydroperoxides. If linalyl acetate is chemicallyhydrolysed outside the skin it can thereafter be oxidised to the same haptensas seen for linalool. A corresponding example is citronellol and citronellylacetate. In clincal studies, concomitant reactions to oxidised linalool andoxidised linalyl acetate have been observed. Whether these reactions depend oncross-reactivity or are due to exposure to both fragrance substances cannot beelucidated as both have an allergenic effect themselves. Linalool and linalylacetate are the main components of lavender oil. They autoxidise on airexposure also when present in the essential oil, and form the same oxidationproducts found in previous studies of the pure synthetic terpenes. Experimentalsensitisation studies showed that air exposure of lavender oil increased thesensitisation potency. Patch test results in dermatitis patients showed aconnection between positive reactions to oxidised linalool, linalyl acetate andlavender oil.
Prohaptens
Compounds that are bioactivated in the skin and thereby formhaptens are referred to as prohaptens.
In the case of prohaptens, the possibility to become activated isinherent to the molecule and activation cannot be avoided by extrinsicmeasures. Activation processes increase the risk for cross-reactivity betweenfragrance substances. Crossreactivity has been shown for certain alcohols andtheir corresponding aldehydes, i.e. between geraniol and geranial (citral) andbetween cinnamyl alcohol and cinnamal.
The human skin expresses enzyme systems that are able tometabolise xenobiotics, modifying their chemical structure to increasehydrophilicity and allow elimination from the body. Xenobiotic metabolism canbe divided into two phases: phase I and phase II. Phase I transformations areknown as activation or functionalisation reactions, which normally introduce orunmask hydrophilic functional groups. If the metabolites are sufficiently polarat this point they will be eliminated. However, many phase I products have toundergo subsequent phase II transformations, i.e. conjugation to make themsufficiently water soluble to be eliminated. Although the purpose of xenobioticmetabolism is detoxification, it can also convert relatively harmless compoundsinto reactive species. Cutaneous enzymes that catalyse phase I transformationsinclude the cytochrome P450 mixed-function oxidase system, alcohol and aldehydedehydrogenases, monoamine oxidases, flavin-containing monooxygenases andhydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferasesand sulfotransferases are examples of phase II enzymes that have been shown tobe present in human skin . These enzymes are known to catalyse both activatingand deactivating biotransformations, but the influence of the reactions on theallergenic activity of skin sensitisers has not been studied in detail. Skinsensitising prohaptens can be recognised and grouped into chemical classesbased on knowledge of xenobiotic bioactivation reactions, clinical observationsand/or in vivo and in vitro studies of sensitisation potential and chemicalreactivity.
QSAR prediction: The relationshipsbetween molecular structure and reactivity that form the basis for structuralalerts are based on well established principles of mechanistic organicchemistry. Examples of structural alerts are aliphatic aldehydes (alerting tothe possibility of sensitisation via a Schiff base reaction with protein aminogroups), and alpha,beta-unsaturated carbonyl groups, C=C-CO- (alerting to thepossibility of sensitisation via Michael addition of protein thiol groups).Prediction of the sensitisation potential of compounds that can act via abioticor metabolic activation (pre- or prohaptens) is more complex compared to thatof compounds that act as direct haptens without any activation. Theautoxidation patterns can differ due to differences in the stability of theintermediates formed, e.g. it has been shown that autoxidation of thestructural isomers linalool and geraniol results in different majorhaptens/allergens. Moreover, the complexity of the prediction increases furtherfor those compounds that can act both as pre- and prohaptens. In such cases,the impact on the sensitisation potency depends on the degree of abioticactivation (e.g. autoxidation) in relation to the metabolic activation.
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.
Tumorigenic by RTECS criteria

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

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

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
N-methyl-2-pyrrolidone	EC50	48	Crustacea	ca.4897mg/L	1
N-methyl-2-pyrrolidone	EC50	384	Crustacea	133.481mg/L	3
N-methyl-2-pyrrolidone	LC50	96	Fish	464mg/L	1
N-methyl-2-pyrrolidone	NOEC	504	Crustacea	12.5mg/L	2
N-methyl-2-pyrrolidone	EC50	72	Algae or other aquatic plants	>500mg/L	2
dimethyl glutarate	EC50	96	Algae or other aquatic plants	7.186mg/L	3
dimethyl glutarate	LC50	96	Fish	93.991mg/L	3
dimethyl glutarate	NOEC	72	Algae or other aquatic plants	36mg/L	2
dimethyl succinate	EC50	96	Algae or other aquatic plants	11.917mg/L	3
dimethyl succinate	LC50	96	Fish	>50- <100mg/L	2
dimethyl succinate	EC50	48	Crustacea	>100mg/L	2
dimethyl succinate	EC50	72	Algae or other aquatic plants	>100mg/L	2
dimethyl succinate	NOEC	72	Algae or other aquatic plants	100mg/L	2
dimethyl adipate	EC50	96	Algae or other aquatic plants	4.351mg/L	3
dimethyl adipate	LC50	96	Fish	55.898mg/L	3
dimethyl adipate	EC50	48	Crustacea	72mg/L	2
dimethyl adipate	EC50	72	Algae or other aquatic plants	>100mg/L	2
dimethyl adipate	NOEC	72	Algae or other aquatic plants	12.5mg/L	2
d-limonene	EC50	384	Crustacea	0.051mg/L	3
d-limonene	EC50	96	Algae or other aquatic plants	0.212mg/L	3
d-limonene	LC50	96	Fish	0.199mg/L	3
d-limonene	EC50	48	Crustacea	0.36mg/L	2
d-limonene	NOEC	48	Crustacea	0.074mg/L	2
Legend:	*Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*				

Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
Terpenes such as limonene and isoprene contribute to aerosol and photochemical smog formation. Emissions of biogenic hydrocarbons, such as the terpenes, to the atmosphere may either decrease ozone concentrations when oxides of nitrogen are low or, if emissions take place in polluted air (i.e containing high concentrations of nitrogen oxides), leads to an increase in ozone concentrations. Lower terpenoids can react with unstable reactive gases and may act as precursors of photochemical smog therefore indirectly influencing community and ecosystem properties.
Complex chlorinated terpenes such as toxaphene (a persistent, mobile and toxic insecticide) and its degradation products, were produced by photoinitiated reactions in an aqueous system, initially containing limonene and other monoterpenes, simulating pulp bleach conditions
The reactions of ozone with larger unsaturated compounds, such as the terpenes can give rise to oxygenated species with low vapour pressures that subsequently condense to form secondary organic aerosol.
Substances containing unsaturated carbons are ubiquitous in indoor environments. They result from many sources (see below). Most are reactive with environmental ozone and many produce stable products which are thought to adversely affect human health.The potential for surfaces in an enclosed space to facilitate reactions should be considered.

Source of unsaturated substances	Unsaturated substances (Reactive Emissions)	Major Stable Products produced following reaction with ozone.
Occupants (exhaled breath, ski oils, personal care products)	Isoprene, nitric oxide, squalene, unsaturated sterols, oleic acid and other unsaturated fatty acids, unsaturated oxidation products	Methacrolein, methyl vinyl ketone, nitrogen dioxide, acetone, 6MHQ, geranyl acetone, 4OPA, formaldehyde, nonanol, decanal, 9-oxo-nonanoic acid, azelaic acid, nonanoic acid.
Soft woods, wood flooring, including cypress, cedar and silver fir boards, houseplants	Isoprene, limonene, alpha-pinene, other terpenes and sesquiterpenes	Formaldehyde, 4-AMC, pinoaldehyde, pinic acid, pinonic acid, formic acid, methacrolein, methyl vinyl ketone, SOAs including ultrafine particles
Carpets and carpet backing	4-Phenylcyclohexene, 4-vinylcyclohexene, styrene, 2-ethylhexyl acrylate, unsaturated fatty acids and esters	Formaldehyde, acetaldehyde, benzaldehyde, hexanal, nonanal, 2-nonenal
Linoleum and paints/polishes containing linseed oil	Linoleic acid, linolenic acid	Propanal, hexanal, nonanal, 2-heptenal, 2-nonenal, 2-decenal, 1-pentene-3-one, propionic acid, n-butyric acid
Latex paint	Residual monomers	Formaldehyde
Certain cleaning products, polishes, waxes, air fresheners	Limonene, alpha-pinene, terpinolene, alpha-terpineol, linalool, linalyl acetate and other terpenoids, longifolene and other sesquiterpenes	Formaldehyde, acetaldehyde, glycoaldehyde, formic acid, acetic acid, hydrogen and organic peroxides, acetone, benzaldehyde, 4-hydroxy-4-methyl-5-hexen-1-al, 5-ethenyl-dihydro-5-methyl-2(3H)-furanone, 4-AMC, SOAs including ultrafine particles
Natural rubber adhesive	Isoprene, terpenes	Formaldehyde, methacrolein, methyl vinyl ketone
Photocopier toner, printed paper, styrene polymers	Styrene	Formaldehyde, benzaldehyde
Environmental tobacco smoke	Styrene, acrolein, nicotine	Formaldehyde, benzaldehyde, hexanal, glyoxal, N-methylformamide, nicotinaldehyde, cotinine
Soiled clothing, fabrics, bedding	Squalene, unsaturated sterols, oleic acid and other saturated fatty acids	Acetone, geranyl acetone, 6MHO, 40PA, formaldehyde, nonanal, decanal, 9-oxo-nonanoic acid, azelaic acid, nonanoic acid
Soiled particle filters	Unsaturated fatty acids from plant waxes, leaf litter, and other vegetative debris; soot; diesel particles	Formaldehyde, nonanal, and other aldehydes; azelaic acid; nonanoic acid; 9-oxo-nonanoic acid and other oxo-acids; compounds with mixed functional groups (=O, -OH, and -COOH)
Ventilation ducts and duct liners	Unsaturated fatty acids and esters, unsaturated oils, neoprene	C5 to C10 aldehydes
'Urban grime'	Polycyclic aromatic hydrocarbons	Oxidized polycyclic aromatic hydrocarbons

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Perfumes, colognes, essential oils (e.g. lavender, eucalyptus, tea tree)	Limonene, alpha-pinene, linalool, linalyl acetate, terpinene-4-ol, gamma-terpinene	Formaldehyde, 4-AMC, acetone, 4-hydroxy-4-methyl-5-hexen-1-al, 5-ethenyl-dihydro-5-methyl-2(3H) furanone, SOAs including ultrafine particles
Overall home emissions	Limonene, alpha-pinene, styrene	Formaldehyde, 4-AMC, pinonaldehyde, acetone, pinic acid, pinonic acid, formic acid, benzaldehyde, SOAs including ultrafine particles

Abbreviations: 4-AMC, 4-acetyl-1-methylcyclohexene; 6MHQ, 6-methyl-5-heptene-2-one, 4OPA, 4-oxopentanal, SOA, Secondary Organic Aerosols
Reference: Charles J Weschler; Environmental Helath Perspectives, Vol 114, October 2006

The alkali metal cyanides (and other metal cyanides) are verysoluble in water. As a result, they readily dissociate into their respectiveanions and cations when released into water. Depending on the pH of the water,the resulting cyanide ion may then form hydrogen cyanide or react with variousmetals in natural water. The proportion of hydrogen cyanide formed from solublecyanides increases as the water pH decreases. At pH <7, >99% of thecyanide ions in water are converted to hydrogen cyanide. As the pH increases,cyanide ions in the water may form complex metallocyanides in the presence ofexcess cyanides; however, if metals are prevalent, simple metal cyanides areformed. Volatilization is the dominant mechanism for the removal of freecyanide. At pH >9.2, most of the free cyanide should exist as HCN, avolatile form of cyanide. Wide variations in the rate of volatilization areexpected since this process is affected by a number of parameters such astemperature, pH, wind speed, and cyanide concentration. Volatilization of freecyanide from concentrated solutions is most effective under conditions of hightemperatures, high dissolved oxygen levels, and at increased concentrations ofatmospheric carbon dioxide

Unlike water-soluble alkali metal cyanides, insoluble metalcyanides are not expected to degrade to hydrogen cyanide. Cyanide occurs mostcommonly as hydrogen cyanide in water, although it can also occur as thecyanide ion, alkali and alkaline earth metal cyanides (potassium cyanide,sodium cyanide, calcium cyanide), relatively stable metallocyanide complexes(ferricyanide complex [Fe(CN)6]-3), moderately stable metallocyanide complexes(complex nickel and copper cyanide), or easily decomposable metallocyanidecomplexes (zinc cyanide [Zan(CN)2], cadmium cyanide [Cd(CN)2]). Oxidation, hydrolysis, and photolysis (photodegradation) are the threepredominant chemical processes that may cause loss of simple cyanides inaquatic media.

Certain cyanides are oxidised to isocyanates by strong oxidisingagents; the isocyanates may be further hydrolysed to ammonia and carbondioxide. However, it has not yet been determined whether such oxidation andsubsequent hydrolysis of isocyanate is a significant fate process in naturalwaters known to contain peroxy radicals. In water, hydrogen cyanide andcyanide ion exist in equilibrium with their relative concentrations primarilydependent on pH and temperature. At pH <8, >93% of the free cyanide inwater will exist as undissociated hydrogen cyanide. Hydrogen cyanide can behydrolysed to formamide, which is subsequently hydrolysed to ammonium andformate ions. However, the relatively slow rates of hydrolysis reportedfor hydrogen cyanide in acidic solution and of cyanides under alkalineconditions indicate that hydrolysis is not competitive with volatilisation andbiodegradation for removal of free cyanide from ambient waters At pH<9.2, most of the free cyanide in solution should exist as hydrogen cyanide,a volatile cyanide form. On the basis of Henry's law constant and thevolatility characteristics associated with various ranges of Henry's lawconstant, volatilization is a significant and probably dominant fate processfor hydrogen cyanide in surface water. The most common alkali metal cyanides(e.g., sodium and potassium cyanide) may also be lost from surface waterprimarily through volatilization; whereas, the sparingly soluble metal cyanidessuch as copper (I) cyanide are removed from water predominantly bysedimentation and biodegradation. Because volatilisation is not an importantfate process for cyanide in groundwater, cyanide would be expected to persistfor considerably longer periods of time in underground aquifers than in surfacewater.

The significance of photolysis in the fate of cyanides in waterhas not been fully investigated. Hydrogen cyanide and cyanide ions in aqueoussolution have been found to be very resistant to photolysis by naturalsunlight, except under heterogeneous photocatalytic conditions. Photocatalyticoxidation may not be significant in natural waters, however, because ofsignificant light reduction at increasingly greater depths. In clear water orat water surfaces, some metallocyanides, such as ferrocyanides andferricyanides, may decompose to the cyanide ion by photodissociation and subsequentlyform hydrogen cyanide.

Biodegradation is an important transformation process for cyanidein natural surface waters, and is dependent on such factors as cyanideconcentrations, pH, temperature, availability of nutrients, and acclimation ofmicrobes. Although the cyanide ion is toxic to microorganisms at concentrationsas low as 5-10 mg/L, acclimation increases tolerance to this compound. Mixed microorganisms in sewage sludge or activated sludge acclimated tocyanide also significantly biodegrade concentrations <=100 mg/L of mostsimple and complex cyanides. It is known that there is a naturalattentuation of the cyanide ion and thiocyanide concentrations in waste waters,for example those obtained gold mill tails, that is due the acclimation of indigenousmicroflora in the tailings. A number of microorganisms have been identifiedthat are capable of uptake, conversion, sorption, and/or precipitation of thecyanide ion, cyanate, and thiocyanate, including species of the genera, *Actinomyces,Alcaligenes, Arthrobacter, Bacillus, Micrococcus, Neisseria, Paracoccus,Pseudomonas*, and *Thiobacillus*. Some of these species, forexample *Pseudomonas*, are capable of using the cyanide ion andthiocyanate as the sole source of carbon and nitrogen and therefore, areparticularly effective at cyanide degradation. Infact, *Pseudomonas* is the basis of commercial applications fordegrading the cyanide ion to ammonia and carbonate in waste waters generated inmining operations that use the cyanide ion to leach gold and other preciousmetals for low-grade ores. Sulfur transferases such as rhodanese areinvolved in substitution reactions that result in the conversion of the cyanideion to the less toxic thiocyanate, whereas pyridoxal phosphate enzymes areinvolved in substitution/addition reactions that result in production ofnitrile derivatives of a-amino acids. These organic nitriles may then beultimately degraded via enzyme catalysed hydrolysis to either the correspondingamino acid and ammonia or the carboxylic acid and ammonia. The cyanidehydratase and cyanidase enzymes catalyse the hydrolysis of the cyanide ion toformamide or formic acid and ammonia, respectively In soil, cyanidepresent at low concentrations would biodegrade under aerobic conditions withthe initial formation of ammonia, which would be converted to nitrite and nitratein the presence of nitrifying bacteria. Under anaerobic conditions, thecyanides ion will denitrify to gaseous nitrogen. Upper limits of 200 and 2 ppm(mg/kg CN–), respectively, have been reported for uninhibited aerobic andanaerobic biodegradation of cyanide in soil; however, these limits have notbeen confirmed in other studies. Cyanide ions in soil are not involved inoxidation-reduction reactions but may undergo complexation reactions with metalions in soil

Cyanides are sorbed by various natural media, including clays,biological solids and sediments Hydrogen cyanide and the alkali metalcyanides are not likely to be strongly sorbed onto sediments and suspendedsolids because of their high water solubilities. Soluble metal cyanides may showsomewhat stronger sorption than hydrogen cyanide, with the extent of sorptionincreasing with decreasing pH and increasing iron oxide, clay, and organicmaterial contents of sediment and suspended solids. However, sorption isprobably insignificant even for metal cyanides when compared to volatilisationand biodegradation. Cyanides are fairly mobile in soil. Mobility is lowest insoils with low pH and high concentrations of free iron oxides, positivelycharged particles, and clays (e.g., chlorite, kaolin, gibbsite), and highest insoils with high pH, high concentrations of free CaCO3 and negatively chargedparticles, and low clay content. Although cyanide has a low soil sorptioncapability, it is usually not detected in groundwater, probably because offixation by trace metals through complexation or transformation by soilmicroorganisms. In soils where cyanide levels are high enough to be toxic tomicroorganisms (i.e., landfills, spills), this compound may leach intogroundwater. Leaching of cyanide into a shallow aquifer has been demonstrated.Volatilisation of hydrogen cyanide would be a significant loss mechanism forcyanides from soil surfaces at a pH <9.2.

Most cyanide in the atmosphere exists almost entirely as hydrogencyanide gas, although small amounts of metal cyanides may be present asparticulate matter in the air. Hydrogen cyanide is very resistant to photolysisat wavelengths of normal sunlight. The most important reaction of hydrogencyanide in air is the reaction with photochemically-generated hydroxyl radicalsand subsequent rapid oxidation to carbon monoxide (CO) and nitric oxide (NO);photolysis and reaction with ozone are not important transformation processes,and reaction with singlet oxygen is not a significant transformationprocess except at stratospheric altitudes where singlet oxygen is present insignificant concentrations. The rate of hydroxyl radical reaction with hydrogencyanide in the atmosphere depends on the altitude, and the rate of the reactionis at least an order of magnitude faster at lower tropospheric altitudes (0–8km) than at upper tropospheric altitudes (10–12 km). Based on a reaction rateconstant of 3x10-14 cm3/(molecule-sec) at 25 °C and assuming an averagehydroxyl radical concentration of 5x105 molecules/cm3, the residence time forthe reaction of hydrogen cyanide vapor with hydroxyl radicals in the atmosphereis approximately 2 years

There is some evidence that certain metal cyanide complexesbioaccumulate in aquatic organisms. Fish from water with soluble silver andcopper cyanide complexes were found to have metal cyanides in their tissues atconcentrations ranging up to 168 and 304 µg/g, respectively (wet or dry weightnot specified). It is difficult to evaluate the toxicologic significance ofbioaccumulation of metal cyanide complexes because these compounds are muchless toxic than soluble hydrogen cyanide, sodium cyanide, or potassium cyanide.There is no evidence of biomagnification of cyanides in the food chain.Accumulation of cyanide in food webs is not expected, considering the rapiddetoxification of cyanide by most species and the lethal effects of large dosesof cyanide

For limonenes

Atmospheric fate: Due to the high volatility of limonene the atmosphere is expected to be the major environmental sink for this chemical where it is expected to undergo gas-phase reactions with photochemically produced hydroxyl radicals, ozone and nitrate radicals. Calculated lifetimes for the reaction of d-limonene with photochemically produced hydroxyl radicals range from 0.3-2 h based on experimentally determined rate constants. The oxidation of limonene may contribute to aerosol and photochemical smog formation.

Calculated lifetimes for the night-time reaction of d-limonene with nitrate radicals range form 0.9 to 9 minutes. The daytime atmospheric lifetime of d-limonene is estimated to range from 12 to 48 min. depending upon local hydroxyl rate and ozone concentrations. Products produced from hydroxy radical reaction with limonene are 4-acetyl-1-methylcyclohexene, a keto-aldehyde, formaldehyde, 3-oxobutanal, glyoxal and a C10 dicarbonyl. The same carbonyls, along with formic acid and C8 and C9 carboxylic acids, may form in reactions with ozone. Ozonolysis of limonene may also lead to the formation of hydrogen peroxide and organic peroxides, which have various toxic effects on plant cells and may damage forests.

Products of ozonolysis include bis(hydroxmethyl)peroxide, a precursor to hydroxymethyl hydroperoxide and hydrogen peroxide. The reaction of d-limonene with ozone in the dark results in the formation of 4-acetyl-1-methylcyclohexene and formaldehyde. Reactions with nitrogen oxides produce aerosol formation as well as lower molecular weight products such as formaldehyde, acetaldehyde, formic acid, acetone and peroxyacetyl nitrate.

Terrestrial fate: When released to the ground limonene is expected to have low to very low mobility in soil based on its physicochemical properties. The soil adsorption coefficient (Koc) calculated on the basis of solubility (13.8 mg/l, 25 C) and the log octanol/ water partition coefficient (4.23) ranges from 1030 and 4780. The Henry's law constant indicates that limonene will rapidly volatilise from both dry and moist soil; however its absorption to soil may slow the process.

Aquatic fate: In the aquatic environment, limonene is expected to evaporate to a significant extent owing to its high volatility. The estimated half-life for volatilisation of limonene from a model river (1 m deep, flow 1 m/s and wind speed 3 m/s) is 3.4 h. Some limonene is expected to absorb to sediment and suspended organic matter.

Biodegradation and bioaccumulation: Limonene does not have functional groups for hydrolysis and its cyclohexene ring and ethylene group are known to resist hydrolysis. Therefore, hydrolysis of limonene is not expected in terrestrial or in aquatic environments. The hydrolytic half-life of d-limonene is estimated to be >1000 days. Biotic degradation of limonene has been shown with some species of microorganisms such as *Penicillium digitatum, Corynespora cassiicola, Diplodia gossyppina* and a soil strain of *Pseudomonans sp (SL strain).* Limonene is readily biodegradable (41-98% degradation by biological oxygen demand in 14 d) under aerobic conditions in a standard test (OECD 301 C 'Modified MITI Test (1)', OECD, 1981a; MITI, 1992). Also in a test simulating aerobic sewage treatment (OECD 303 A 'Simulation Test - Aerobic Sewage Treatment: Coupled Units Test'; OECD, 1981b), limonene disappeared almost completely (>93.8%) during 14 days of incubation.

Biodegradation has been assessed under anaerobic conditions; there was no indication of any metabolisms, possibly because of the toxicity to micro-organisms.

Continued...

The bioconcentration factor, calculated on the basis of water solubility and the log octanol/ water partition coefficient (log Kow) is 246-262, suggesting that limonene may bioaccumulate in fish and other aquatic species.
Ecotoxicity: Technical limonene is practically nontoxic to birds on a subacute dietary basis, and is slightly toxic to freshwater fish and invertebrates on an acute basis.
for d-limonene:
LD50 Colinus virginianus (Bobwhite quail, 16 weeks old) oral >2000 mg/kg
LC50 Colinus virginianus (Bobwhite quail, 10 day old) dietary >5620 ppm/8 days
LC50 Colinus virginianus (Bobwhite quail, 14 day old) dietary >5000 ppm/8 days
LC50 Anas platyrhynchos (Mallard duck, 14 day old) dietary >5000 ppm/8 days
LC50 Oncorhynchus mykiss (Rainbow trout) 80 ppm/96 hr (95% confidence limit: 71.4-88.7 ppm); static /92% AI formulated product
LC50 Oncorhynchus mykiss (Rainbow trout) 568 ppm/96 hr (95% confidence limit: 437-852 ppm); static /4.0% AI formulated product
EC50 Daphnia magna (Water flea, <24 hr old; intoxication, immobilization) 17 ppm/48 hr (95% confidence limit: 11-33 ppm); static /4.0% AI formulated product
LC50 Pimephales promelas (Fathead minnow) 966 ppm/96 hr (95% confidence limit: 740-1652 ppm); static /4.0% AI formulated product
LC50 Pimephales promelas (Fathead minnow) 38.5 mg/L/96 hr; flow through /from table/ LC50
Leuciscus idus (Golden orfe) 32 mg/L/48 hr /Conditions of bioassay not specified in source examined
The acute toxicity of d-limonene ranges from slight to high for aquatic organisms. The lowest acute toxicity values (EC50 or LC50) identified were approximately 0.4 mg/litre for Daphnia (US EPA, 1990b) and 0.7 mg/litre for fish (US EPA, 1990a,b). The no-observed-effect concentration (NOEC) for
green algae is approximately 4 mg/litre (US EPA, 1990a). The acute toxicity (EC50 or LC50) of dipentene to Daphnia and fish is about 50-70 times lower than that for d-limonene (US EPA, 1990b).
No studies were identified on the chronic toxicity of limonene to aquatic organisms.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
N-methyl-2-pyrrolidone	LOW	LOW
dimethyl glutarate	LOW	LOW
dimethyl succinate	LOW	LOW
dimethyl adipate	LOW	LOW
d-limonene	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
N-methyl-2-pyrrolidone	LOW (BCF = 0.16)
dimethyl glutarate	LOW (LogKOW = 0.62)
dimethyl succinate	LOW (LogKOW = 0.35)
dimethyl adipate	LOW (LogKOW = 1.03)
d-limonene	HIGH (LogKOW = 4.8275)

Mobility in soil

Ingredient	Mobility
N-methyl-2-pyrrolidone	LOW (KOC = 20.94)
dimethyl glutarate	LOW (KOC = 10)
dimethyl succinate	LOW (KOC = 10)
dimethyl adipate	LOW (KOC = 10.9)
d-limonene	LOW (KOC = 1324)

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 SDS and observe all notices pertaining to the product. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 TRANSPORT INFORMATION

Continued...

Labels Required

COMBUSTIBLE LIQUID	COMBUSTIBLE LIQUID, regulated for storage purposes only
Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

N-METHYL-2-PYRROLIDONE(872-50-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards | Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists

DIMETHYL GLUTARATE(1119-40-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

DIMETHYL SUCCINATE(106-65-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

DIMETHYL ADIPATE(627-93-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

D-LIMONENE(5989-27-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists | International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
Australia Inventory of Chemical Substances (AICS)

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (dimethyl glutarate; d-limonene; dimethyl succinate; N-methyl-2-pyrrolidone; dimethyl adipate)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	Y
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

Ingredients with multiple cas numbers

Name	CAS No
N-methyl-2-pyrrolidone	26138-58-9, 872-50-4
d-limonene	138-86-3, 5989-27-5

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:
www.chemwatch.net

The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
PC—STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists

Continued...

STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit。
IDLH: Immediately Dangerous to Life or Health Concentrations
OSF: Odour Safety Factor
NOAEL :No Observed Adverse Effect Level
LOAEL: Lowest Observed Adverse Effect Level
TLV: Threshold Limit Value
LOD: Limit Of Detection
OTV: Odour Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index