MOPAR Air Intake & EGR Cleaner (Liquid - Non-Aerosol)

Mopar(FCA US LLC Service & Customer Care Division)

Chemwatch Hazard Alert Code: **3**

Part Number: **674**
Version No: **2.2**
Safety Data Sheet according to OSHA HazCom Standard (2012) requirements

Issue Date: **09/21/2022**
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L.GHS.USA.EN

SECTION 1 Identification

Product Identifier

Product name	MOPAR Air Intake & EGR Cleaner (Liquid - Non-Aerosol)
Synonyms	68628281AA, 68621326AA
Proper shipping name	Flammable liquids, n.o.s. (contains xylene, distillates, petroleum, light, hydrotreated, distillates, petroleum, middle, sweetened and acetone)
Other means of identification	Not Available

Recommended use of the chemical and restrictions on use

Relevant identified uses	Intake Cleaner

Name, address, and telephone number of the chemical manufacturer, importer, or other responsible party

Registered company name	**Mopar(FCA US LLC Service & Customer Care Division)**	**Mopar (FCA US LLC Service & Customer Care Division)**
Address	26311 Lawrence Avenue, Center Line Michigan 48015 United States	26311 Lawerence Avenue, Center Line Michigan 48015 United States
Telephone	1-800-846-6727	1-800-846-6727
Fax	Not Available	Not Available
Website	Not Available	Not Available
Email	moparsds@fcagroup.com	moparsds@fcagroup.com

Emergency phone number

Association / Organisation	**CHEMTREC**	**CHEMTREC**
Emergency telephone numbers	+1 703-741-5970	+1 703-741-5970
Other emergency telephone numbers	248-512-8002	248-512-8002

SECTION 2 Hazard(s) identification

Classification of the substance or mixture

ChemWatch Hazard Ratings

		Min	Max
Flammability	3		
Toxicity	1		
Body Contact	2		
Reactivity	0		
Chronic	3		

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

NFPA 704 diamond

Note: The hazard category numbers found in GHS classification in section 2 of this SDSs are NOT to be used to fill in the NFPA 704 diamond. Blue = Health Red = Fire Yellow = Reactivity White = Special (Oxidizer or water reactive substances)

Classification	Serious Eye Damage/Eye Irritation Category 2A, Carcinogenicity Category 1B, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Reproductive Toxicity Category 1A, Specific Target Organ Toxicity - Repeated Exposure Category 2, Flammable Liquids Category 2, Skin Corrosion/Irritation Category 2, Aspiration Hazard Category 1

Label elements

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

Hazard statement(s)

H319	Causes serious eye irritation.
H350	May cause cancer.
H336	May cause drowsiness or dizziness.
H360	May damage fertility or the unborn child.
H373	May cause damage to organs through prolonged or repeated exposure.
H225	Highly flammable liquid and vapour.
H315	Causes skin irritation.
H304	May be fatal if swallowed and enters airways.

Hazard(s) not otherwise classified

Not Applicable

Precautionary statement(s) General

P101	If medical advice is needed, have product container or label at hand.
P102	Keep out of reach of children.
P103	Read label before use.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P233	Keep container tightly closed.
P260	Do not breathe mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P240	Ground/bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use only non-sparking tools.
P243	Take precautionary measures against static discharge.
P261	Avoid breathing mist/vapours/spray.
P202	Do not handle until all safety precautions have been read and understood.
P264	Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.
P331	Do NOT induce vomiting.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam to extinguish.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P314	Get medical advice/attention if you feel unwell.
P337+P313	If eye irritation persists: Get medical advice/attention.
P302+P352	IF ON SKIN: Wash with plenty of water.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water/shower.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

P403+P235	Store in a well-ventilated place. Keep cool.
P405	Store locked up.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

Precautionary statement(s) Disposal

Continued...

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
1330-20-7*	25.6-32	xylene
100-41-4*	4.8-6.4	ethylbenzene
64742-47-8*	10-30	distillates, petroleum, light, hydrotreated
67-64-1	10-30	acetone
872-50-4*	10-30	1-Methyl-2-Pyrrolidone
64741-86-2*	2-2.495	distillates, petroleum, middle, sweetened
Not Available	1.5-1.995	Polyether Amine
64742-94-5*	<=0.245	Naphtha, Heavy Aromatic
91-57-6*	<=0.0637	2-methylnaphthalene
90-12-0*	<=0.030625	1-methylnaphthalene
91-20-3*	<=1	naphthalene
108-88-3*	0.032-0.16	toluene
67-63-0*	5-10	2-Propanol

The specific chemical identity and/or exact percentage (concentration) of composition has been withheld as a trade secret.

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, aerosols or combustion products are inhaled remove from contaminated area. ▸ Other measures are usually unnecessary.
Ingestion	▸ Immediately give a glass of water. ▸ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor. ▸ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

Most important symptoms and effects, both acute and delayed

See Section 11

Indication of any immediate medical attention and special treatment needed

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

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

[Ellenhorn and Barceloux: Medical Toxicology]
Management:
Measurement of serum and urine acetone concentrations may be useful to monitor the severity of ingestion or inhalation.
Inhalation Management:

- Maintain a clear airway, give humidified oxygen and ventilate if necessary.
- If respiratory irritation occurs, assess respiratory function and, if necessary, perform chest X-rays to check for chemical pneumonitis.
- Consider the use of steroids to reduce the inflammatory response.
- Treat pulmonary oedema with PEEP or CPAP ventilation.

Dermal Management:

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

Eye Management:

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

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

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

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

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

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

SECTION 5 Fire-fighting measures

Extinguishing media

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

Special hazards arising from the substrate or mixture

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

Special protective equipment and precautions for fire-fighters

Fire Fighting	
Fire/Explosion Hazard	‣ Liquid and vapour are highly flammable. ‣ Severe fire hazard when exposed to heat, flame and/or oxidisers. ‣ Vapour may travel a considerable distance to source of ignition. ‣ Heating may cause expansion or decomposition leading to violent rupture of containers. ‣ On combustion, may emit toxic fumes of carbon monoxide (CO). Combustion products include: carbon dioxide (CO_2) other pyrolysis products typical of burning organic material. **Contains low boiling substance:** Closed containers may rupture due to pressure buildup under fire conditions.

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	‣ Remove all ignition sources. ‣ Clean up all spills immediately. ‣ Avoid breathing vapours and contact with skin and eyes. ‣ Control personal contact with the substance, by using protective equipment. ‣ Contain and absorb small quantities with vermiculite or other absorbent material. ‣ Wipe up. ‣ Collect residues in a flammable waste container.
Major Spills	‣ Clear area of personnel and move upwind. ‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear full body protective clothing with breathing apparatus. ‣ Prevent, by all means available, spillage from entering drains or water courses. ‣ Consider evacuation (or protect in place). ‣ No smoking, naked lights or ignition sources. ‣ Increase ventilation. ‣ Stop leak if safe to do so. ‣ Water spray or fog may be used to disperse / absorb vapour. ‣ Contain or absorb spill with sand, earth or vermiculite. ‣ Collect recoverable product into labelled containers for recycling. ‣ Collect solid residues and seal in labelled drums for disposal. ‣ Wash area and prevent runoff into drains. ‣ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ‣ If contamination of drains or waterways occurs, advise emergency services. Chemical Class: ketones For release onto land: recommended sorbents listed in order of priority. SORBENT TYPE | RANK | APPLICATION | COLLECTION | LIMITATIONS

Continued...

LAND SPILL - SMALL

cross-linked polymer - particulate	1	shovel	shovel	R, W, SS
cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT
sorbent clay - particulate	2	shovel	shovel	R,I, P
wood fiber - pillow	3	throw	pitchfork	R, P, DGC, RT
treated wood fiber - pillow	3	throw	pitchfork	DGC, RT
foamed glass - pillow	4	throw	pitchfork	R, P, DGC, RT

LAND SPILL - MEDIUM

cross-linked polymer - particulate	1	blower	skiploader	R,W, SS
cross-linked polymer - pillow	2	throw	skiploader	R, DGC, RT
sorbent clay - particulate	3	blower	skiploader	R, I, P
polypropylene - particulate	3	blower	skiploader	R, SS, DGC
expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC
polypropylene - mat	4	throw	skiploader	DGC, RT

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

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling

- Containers, even those that have been emptied, may contain explosive vapours.
- Do NOT cut, drill, grind, weld or perform similar operations on or near containers.

Contains low boiling substance:
Storage in sealed containers may result in pressure buildup causing violent rupture of containers not rated appropriately.

- Check for bulging containers.
- Vent periodically
- Always release caps or seals slowly to ensure slow dissipation of vapours
- Avoid all personal contact, including inhalation.
- Wear protective clothing when risk of exposure occurs.
- Use in a well-ventilated area.
- Prevent concentration in hollows and sumps.
- **DO NOT** enter confined spaces until atmosphere has been checked.
- Avoid smoking, naked lights, heat or ignition sources.
- When handling, **DO NOT** eat, drink or smoke.
- Vapour may ignite on pumping or pouring due to static electricity.
- **DO NOT** use plastic buckets.
- Earth and secure metal containers when dispensing or pouring product.
- Use spark-free tools when handling.
- Avoid contact with incompatible materials.
- Keep containers securely sealed.
- Avoid physical damage to containers.
- Always wash hands with soap and water after handling.
- Work clothes should be laundered separately.
- Use good occupational work practice.
- Observe manufacturer's storage and handling recommendations contained within this SDS.
- Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.

Other information

Consider storage under inert gas.

- Store in original containers in approved flame-proof area.
- No smoking, naked lights, heat or ignition sources.
- **DO NOT** store in pits, depressions, basements or areas where vapours may be trapped.
- Keep containers securely sealed.
- Store away from incompatible materials in a cool, dry well ventilated area.
- Protect containers against physical damage and check regularly for leaks.
- Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container

- Packing as supplied by manufacturer.
- Plastic containers may only be used if approved for flammable liquid.
- Check that containers are clearly labelled and free from leaks.
- For low viscosity materials (i) : Drums and jerry cans must be of the non-removable head type. (ii) : Where a can is to be used as an inner package, the can must have a screwed enclosure.
- For materials with a viscosity of at least 2680 cSt. (23 deg. C)
- For manufactured product having a viscosity of at least 250 cSt. (23 deg. C)

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	▸ Manufactured product that requires stirring before use and having a viscosity of at least 20 cSt (25 deg. C): (i) Removable head packaging; (ii) Cans with friction closures and (iii) low pressure tubes and cartridges may be used. ▸ Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages ▸ In addition, where inner packagings are glass and contain liquids of packing group I there must be sufficient inert absorbent to absorb any spillage, unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
Storage incompatibility	Acetone: ▸ may react violently with chloroform, activated charcoal, aliphatic amines, bromine, bromine trifluoride, chlorotriazine, chromic(IV) acid, chromic(VI) acid, chromium trioxide, chromyl chloride, hexachloromelamine, iodine heptafluoride, iodoform, liquid oxygen, nitrosyl chloride, nitrosyl perchlorate, nitryl perchlorate, perchloromelamine, peroxomonosulfuric acid, platinum, potassium tert-butoxide, strong acids, sulfur dichloride, trichloromelamine, xenon tetrafluoride ▸ reacts violently with bromoform and chloroform in the presence of alkalies or in contact with alkaline surfaces. ▸ may form unstable and explosive peroxides in contact with strong oxidisers, fluorine, hydrogen peroxide (90%), sodium perchlorate, 2-methyl-1,3-butadiene ▸ can increase the explosive sensitivity of nitromethane on contact flow or agitation may generate electrostatic charges due to low conductivity ▸ dissolves or attacks most rubber, resins, and plastics (polyethylenes, polyester, vinyl ester, PVC, Neoprene, Viton) Ketones in this group: ▸ are reactive with many acids and bases liberating heat and flammable gases (e.g., H2). ▸ react with reducing agents such as hydrides, alkali metals, and nitrides to produce flammable gas (H2) and heat. ▸ are incompatible with isocyanates, aldehydes, cyanides, peroxides, and anhydrides. ▸ react violently with aldehydes, HNO3 (nitric acid), HNO3 + H2O2 (mixture of nitric acid and hydrogen peroxide), and HClO4 (perchloric acid). ▸ may react with hydrogen peroxide to form unstable peroxides; many are heat- and shock-sensitive explosives. A significant property of most ketones is that the hydrogen atoms on the carbons next to the carbonyl group are relatively acidic when compared to hydrogen atoms in typical hydrocarbons. Under strongly basic conditions these hydrogen atoms may be abstracted to form an enolate anion. This property allows ketones, especially methyl ketones, to participate in condensation reactions with other ketones and aldehydes. This type of condensation reaction is favoured by high substrate concentrations and high pH (greater than 1 wt% NaOH). ▸ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US OSHA Permissible Exposure Limits (PELs) Table Z-1	xylene	Xylenes (o-, m-, p-isomers)	100 ppm / 435 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	ethylbenzene	Ethyl benzene	100 ppm / 435 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	ethylbenzene	Ethyl benzene	100 ppm / 435 mg/m3	545 mg/m3 / 125 ppm	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	distillates, petroleum, light, hydrotreated	Oil mist, mineral	5 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	acetone	Acetone	1000 ppm / 2400 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	acetone	Acetone	250 ppm / 590 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	distillates, petroleum, middle, sweetened	Oil mist, mineral	5 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	2-methylnaphthalene	Particulates Not Otherwise Regulated (PNOR)- Respirable fraction	5 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	2-methylnaphthalene	Particulates Not Otherwise Regulated (PNOR)- Total dust	15 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	2-methylnaphthalene	Inert or Nuisance Dust: Total Dust	15 mg/m3 / 50 mppcf	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	2-methylnaphthalene	Inert or Nuisance Dust: Respirable fraction	5 mg/m3 / 15 mppcf	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	2-methylnaphthalene	Particulates not otherwise regulated	Not Available	Not Available	Not Available	See Appendix D
US OSHA Permissible Exposure Limits (PELs) Table Z-1	naphthalene	Naphthalene	10 ppm / 50 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	naphthalene	Inert or Nuisance Dust: Respirable fraction	5 mg/m3 / 15 mppcf	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	naphthalene	Inert or Nuisance Dust: Total Dust	15 mg/m3 / 50 mppcf	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	naphthalene	Naphthalene	10 ppm / 50 mg/m3	75 mg/m3 / 15 ppm	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-2	toluene	Toluene	200 ppm	300 ppm	500 (10 min) ppm	(Z37.12-1967)
US NIOSH Recommended Exposure Limits (RELs)	toluene	Toluene	100 ppm / 375 mg/m3	560 mg/m3 / 150 ppm	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	2-Propanol	Isopropyl alcohol	400 ppm / 980 mg/m3	Not Available	Not Available	Not Available

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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US NIOSH Recommended Exposure Limits (RELs)	2-Propanol	Isopropyl alcohol	400 ppm / 980 mg/m3	1225 mg/m3 / 500 ppm	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
xylene	Not Available	Not Available	Not Available
ethylbenzene	Not Available	Not Available	Not Available
distillates, petroleum, light, hydrotreated	140 mg/m3	1,500 mg/m3	8,900 mg/m3
acetone	Not Available	Not Available	Not Available
1-Methyl-2-Pyrrolidone	30 ppm	32 ppm	190 ppm
distillates, petroleum, middle, sweetened	140 mg/m3	1,500 mg/m3	8,900 mg/m3
2-methylnaphthalene	9 mg/m3	54 mg/m3	320 mg/m3
1-methylnaphthalene	20 mg/m3	61 mg/m3	360 mg/m3
naphthalene	15 ppm	83 ppm	500 ppm
toluene	Not Available	Not Available	Not Available
2-Propanol	400 ppm	2000* ppm	12000** ppm

Ingredient	Original IDLH	Revised IDLH
xylene	900 ppm	Not Available
ethylbenzene	800 ppm	Not Available
distillates, petroleum, light, hydrotreated	2,500 mg/m3	Not Available
acetone	2,500 ppm	Not Available
1-Methyl-2-Pyrrolidone	Not Available	Not Available
distillates, petroleum, middle, sweetened	2,500 mg/m3	Not Available
Polyether Amine	Not Available	Not Available
Naphtha, Heavy Aromatic	Not Available	Not Available
2-methylnaphthalene	Not Available	Not Available
1-methylnaphthalene	Not Available	Not Available
naphthalene	250 ppm	Not Available
toluene	500 ppm	Not Available
2-Propanol	2,000 ppm	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
1-Methyl-2-Pyrrolidone	E	≤ 0.1 ppm
Naphtha, Heavy Aromatic	C	> 1 to ≤ 10 parts per million (ppm)
1-methylnaphthalene	E	≤ 0.1 ppm
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

Odour Threshold Value: 3.6 ppm (detection), 699 ppm (recognition)
Saturation vapour concentration: 237000 ppm @ 20 C
NOTE: Detector tubes measuring in excess of 40 ppm, are available.
Exposure at or below the recommended TLV-TWA is thought to protect the worker against mild irritation associated with brief exposures and the bioaccumulation, chronic irritation of the respiratory tract and headaches associated with long-term acetone exposures. The NIOSH REL-TWA is substantially lower and has taken into account slight irritation experienced by volunteer subjects at 300 ppm. Mild irritation to acclimatised workers begins at about 750 ppm - unacclimatised subjects will experience irritation at about 350-500 ppm but acclimatisation can occur rapidly. Disagreement between the peak bodies is based largely on the view by ACGIH that widespread use of acetone, without evidence of significant adverse health effects at higher concentrations, allows acceptance of a higher limit.
Half-life of acetone in blood is 3 hours which means that no adjustment for shift-length has to be made with reference to the standard 8 hour/day, 40 hours per week because body clearance occurs within any shift with low potential for accumulation.
A STEL has been established to prevent excursions of acetone vapours that could cause depression of the central nervous system.
Odour Safety Factor(OSF)
OSF=38 (ACETONE)

NOTE N: The classification as a carcinogen need not apply if the full refining history is known and it can be shown that the substance from which it is produced is not a carcinogen. This note applies only to certain complex oil-derived substances in Annex VI.
European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically

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	"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. ▸ Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulated area. ▸ Work should be undertaken in an isolated system such as a "glove-box" . Employees should wash their hands and arms upon completion of the assigned task and before engaging in other activities not associated with the isolated system. ▸ Within regulated areas, the carcinogen should be stored in sealed containers, or enclosed in a closed system, including piping systems, with any sample ports or openings closed while the carcinogens are contained within. ▸ Open-vessel systems are prohibited. ▸ Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation. ▸ Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system. ▸ For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. ▸ Except for outdoor systems, regulated areas should be maintained under negative pressure (with respect to non-regulated areas). ▸ Local exhaust ventilation requires make-up air be supplied in equal volumes to replaced air. ▸ Laboratory hoods must be designed and maintained so as to draw air inward at an average linear face velocity of 0.76 m/sec with a minimum of 0.64 m/sec. Design and construction of the fume hood requires that insertion of any portion of the employees body, other than hands and arms, be disallowed.
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
Body protection	See Other protection below
Other protection	▸ Employees working with confirmed human carcinogens should be provided with, and be required to wear, clean, full body protective clothing (smocks, coveralls, or long-sleeved shirt and pants), shoe covers and gloves prior to entering the regulated area. [AS/NZS ISO 6529:2006 or national equivalent] ▸ Employees engaged in handling operations involving carcinogens should be provided with, and required to wear and use half-face filter-type respirators with filters for dusts, mists and fumes, or air purifying canisters or cartridges. A respirator affording higher levels of protection may be substituted. [AS/NZS 1715 or national equivalent] ▸ Emergency deluge showers and eyewash fountains, supplied with potable water, should be located near, within sight of, and on the same level with locations where direct exposure is likely. ▸ Prior to each exit from an area containing confirmed human carcinogens, employees should be required to remove and leave protective clothing and equipment at the point of exit and at the last exit of the day, to place used clothing and equipment in impervious containers at the point of exit for purposes of decontamination or disposal. The contents of such impervious containers must be identified with suitable labels. For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. ▸ Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. ▸ Overalls. ▸ PVC Apron. ▸ PVC protective suit may be required if exposure severe. ▸ Eyewash unit. ▸ Ensure there is ready access to a safety shower. ▸ Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity. ▸ For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets). ▸ Non sparking safety or conductive footwear should be considered. Conductive footwear describes a boot or shoe with a sole made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground the foot an shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.

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:
MOPAR Air Intake & EGR Cleaner (Liquid - Non-Aerosol)

Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C

Respiratory protection

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator

Continued...

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

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

up to 10 x ES	AX-AUS	-	AX-PAPR-AUS / Class 1
up to 50 x ES	-	AX-AUS / Class 1	-
up to 100 x ES	-	AX-2	AX-PAPR-2 ^

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

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

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

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Colourless to light yellow

Physical state	Liquid	**Relative density (Water = 1)**	0.844
Odour	Characteristic, strong	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	306 - 653 ppm	**Auto-ignition temperature (°C)**	465
pH (as supplied)	7	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	-95	**Viscosity (cSt)**	0.417
Initial boiling point and boiling range (°C)	56	**Molecular weight (g/mol)**	58.08
Flash point (°C)	-18	**Taste**	Not Available
Evaporation rate	6 BuAC = 1	**Explosive properties**	Not Available
Flammability	HIGHLY FLAMMABLE.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	12.8	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	2	**Volatile Component (%vol)**	50
Vapour pressure (kPa)	24.70	**Gas group**	Not Available
Solubility in water	Soluble in water. Soluble in ethanol. Soluble in ether. Soluble in dimethyl ether. Soluble in petroleum spirit. Soluble in chloroform. Soluble in dimethylformamide. Soluble in oils/fats. Water: Complete Ethanol: Complete Ether: Complete	**pH as a solution (Not Available%)**	Not Available

Continued...

Vapour density (Air = 1)	2	**VOC g/L**	50%

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	The material is not thought to produce adverse health effects or irritation of the respiratory tract (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Material is highly volatile and may quickly form a concentrated atmosphere in confined or unventilated areas. The vapour may displace and replace air in breathing zone, acting as a simple asphyxiant. This may happen with little warning of overexposure. The use of a quantity of material in an unventilated or confined space may result in increased exposure and an irritating atmosphere developing. Before starting consider control of exposure by mechanical ventilation. 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. Exposure to ketone vapours may produce nose, throat and mucous membrane irritation. High concentrations of vapour may produce central nervous system depression characterised by headache, vertigo, loss of coordination, narcosis and cardiorespiratory failure. Some ketones produce neurological disorders (polyneuropathy) characterised by bilateral symmetrical paresthesia and muscle weakness primarily in the legs and arms. Systemic effects of acetone inhalation exposure include central nervous system depression, light-headedness, incoherent speech, ataxia, stupor, hypotension, tachycardia, metabolic acidosis, hyperglycaemia and ketosis. Rarely, convulsions and tubular necrosis may be evident. Other symptoms of exposure may include restlessness, headache, vomiting, low blood-pressure and rapid and irregular pulse, eye and throat irritation, weakness of the legs and dizziness. Inhalation of high concentrations may produce dryness of the mouth and throat, nausea, uncoordinated movement, loss of coordinated speech, drowsiness and, in severe cases, coma. Inhalation of acetone vapours over long periods causes irritation of the respiratory tract, coughing and headache. Rats exposed to 52200 ppm vapour for 1 hour showed clear signs of narcosis; fatalities occurred at 126600 ppm.
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. Accidental ingestion of the material may be damaging to the health of the individual.
Skin Contact	The material may accentuate any pre-existing dermatitis condition Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected. Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. The material may produce mild skin irritation; limited evidence or practical experience suggests, that the material either: ▸ produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or ▸ produces significant, but mild, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (non allergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.
Eye	The liquid may produce eye discomfort and is capable of causing temporary impairment of vision and/or transient eye inflammation, ulceration Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.

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Chronic	On the basis, primarily, of animal experiments, the material may be regarded as carcinogenic to humans. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in cancer on the basis of: - appropriate long-term animal studies - other relevant information Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests. There is sufficient evidence to establish a causal relationship between human exposure to the material and impaired fertility

MOPAR Air Intake & EGR Cleaner (Liquid - Non-Aerosol)	
TOXICITY	**IRRITATION**
Not Available	Not Available

xylene	
TOXICITY	**IRRITATION**
Inhalation (Guinea Pig)LC: 450 ppm/4h[2]	Eye (human): 200 ppm irritant
Inhalation (Human) TCLo: 200 ppm[2]	Eye (rabbit): 5 mg/24h SEVERE
Inhalation (Human) TCLo: 200 ppm/4h[2]	Eye (rabbit): 87 mg mild
Inhalation (man) LCLo: 10000 ppm/6h[2]	Eye: adverse effect observed (irritating)[1]
Inhalation(Rat) LC50; 5000 ppm/4h[2]	Skin (rabbit):500 mg/24h moderate
Intraperitoneal (Mouse) LD50: 1548 mg/kg[2]	Skin: adverse effect observed (irritating)[1]
Intraperitoneal (Rat) LD50: 2459 mg/kg[2]	
Intravenous (Rabbit) LD: 129 mg/kg[2]	
Oral (Human)LD: 50 mg/kg[2]	
Oral (Human)LDLo: 50 mg/kg[2]	
Oral (Mouse) LD50; 2119 mg/kg[2]	
Oral (Rat) LD50; 4300 mg/kg[2]	
Subcutaneous (Rat) LD50: 1700 mg/kg[2]	

ethylbenzene	
TOXICITY	**IRRITATION**
Dermal (rabbit) LD50: 17800 mg/kg[2]	Eye (rabbit): 500 mg - SEVERE
Inhalation (Human) TCLo: 100 ppm/8h[2]	Eye: no adverse effect observed (not irritating)[1]
Inhalation (Rat)LC: 4000 ppm/4h[2]	Skin (rabbit): 15 mg/24h mild
Inhalation (Rat)LCLo: 4000 ppm/4h[2]	Skin: no adverse effect observed (not irritating)[1]
Intraperitoneal (mouse) LD50: 2642 mg/kg[2]	
Oral (Rat) LD50; 3500 mg/kg[2]	

distillates, petroleum, light, hydrotreated	
TOXICITY	**IRRITATION**
Dermal (rabbit) LD50: >2000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
Inhalation(Rat) LC50; >4.3 mg/l4h[1]	Skin: adverse effect observed (irritating)[1]
Oral (Rat) LD50; >5000 mg/kg[2]	

acetone	
TOXICITY	**IRRITATION**
Dermal (rabbit) LD50: 20000 mg/kg[2]	Eye (human): 500 ppm - irritant
Inhalation(Mouse) LC50; 44 mg/L4h[2]	Eye (rabbit): 20mg/24hr -moderate
Oral (Rat) LD50; 5800 mg/kg[2]	Eye (rabbit): 3.95 mg - SEVERE
	Eye: adverse effect observed (irritating)[1]
	Skin (rabbit): 500 mg/24hr - mild
	Skin (rabbit):395mg (open) - mild
	Skin: no adverse effect observed (not irritating)[1]

1-Methyl-2-Pyrrolidone	
TOXICITY	**IRRITATION**
Dermal (rabbit) LD50: 8000 mg/kg[2]	Eye (rabbit): 100 mg - moderate
Oral (Rat) LD50; 3914 mg/kg[2]	
Oral (Rat) LD50; 4200 mg/kg*[2]	

distillates, petroleum, middle, sweetened	
TOXICITY	**IRRITATION**
Dermal (rabbit) LD50: >2000 mg/kg[2]	Not Available
Inhalation(Rat) LC50; 1.72 mg/l4h[1]	
Oral (Rat) LD50; >5000 mg/kg[2]	

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Polyether Amine	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
Naphtha, Heavy Aromatic	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >3160 mg/kg[2]	Eye (rabbit): Irritating
	Oral (Rat) LD50; 3200 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin: adverse effect observed (irritating)[1]
2-methylnaphthalene	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50; 1630 mg/kg[2]	Not Available
1-methylnaphthalene	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50; 1840 mg/kg[2]	Not Available
naphthalene	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2500 mg/kg[2]	Eye (rabbit): 100 mg - mild
	Oral (child) LDLo: 100 mg/kg[2]	Skin (rabbit):495 mg (open) - mild
	Oral (Rat) LD50; 490 mg/kg[2]	
	Unrep. (human) LDLo: 29 mg/kg[2]	
	Unrep. (man) LDLo: 74 mg/kg[2]	
toluene	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 12124 mg/kg[2]	Eye (rabbit): 2mg/24h - SEVERE
	Inhalation (Human) TCLo: 100 ppm[2]	Eye (rabbit):0.87 mg - mild
	Inhalation (man) TCLo: 200 ppm[2]	Eye (rabbit):100 mg/30sec - mild
	Inhalation(Rat) LC50; >26700 ppm/1h[2]	Eye: adverse effect observed (irritating)[1]
	Oral (Human)LDLo: 50 mg/kg[2]	Skin (rabbit):20 mg/24h-moderate
	Oral (Rat) LD50; 636 mg/kg[2]	Skin (rabbit):500 mg - moderate
		Skin: adverse effect observed (irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
2-Propanol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 12800 mg/kg[2]	Eye (rabbit): 10 mg - moderate
	Inhalation (Human) TCLo: 150 ppm/2h[2]	Eye (rabbit): 100 mg - SEVERE
	Inhalation (Human) TCLo: 35 ppm/4h[2]	Eye (rabbit): 100mg/24hr-moderate
	Inhalation(Mouse) LC50; 53000 mg/m3/4h[2]	Skin (rabbit): 500 mg - mild
	Inhalation(Rat) LC50; 72600 mg/m3/4h[2]	
	Intraperitoneal (Guinea pig) LD50: 2560 mg/kg[2]	
	Intraperitoneal (Mouse) LD50: 4477 mg/kg[2]	
	Intraperitoneal (Rabbit) LD50: 667 mg/kg[2]	
	Intraperitoneal (Rat) LD50: 2735 mg/kg[2]	
	Intraperitoneal (Rat) TDLo: 800 mg/kg[2]	
	Intravenous (Cat) LD: 1963 mg/kg[2]	
	Intravenous (Dog) LD: 1024 mg/kg[2]	
	Intravenous (Mouse) LD50: 1509 mg/kg[2]	
	Intravenous (Rabbit) LD50: 1184 mg/kg[2]	
	Intravenous (Rat) LD50: 1088 mg/kg[2]	
	Oral (Dog) LD: 1537 mg/kg[2]	
	Oral (Human)LD: 3570 mg/kg[2]	
	Oral (Human)LD: 5272 mg/kg[2]	
	Oral (Human)LDLo: 3570 mg/kg[2]	
	Oral (Human)TDLo: 14432 mg/kg[2]	
	Oral (Human)TDLo: 223 mg/kg[2]	
	Oral (Human)TDLo: 286 mg/kg[2]	
	Oral (man) TDLo: 14432 mg/kg[2]	
	Oral (Mouse) LD50; 3600 mg/kg[2]	

Continued...

	Oral (Rabbit) LD50: 6410 mg/kg[2]	
	Oral (Rat) LD50; 5000 mg/kg[2]	
	Oral (Rat) LD50; 5045 mg/kg[2]	
Legend:	*1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*	

MOPAR Air Intake & EGR Cleaner (Liquid - Non-Aerosol)	Most people have very few pyrroles in their system at any given time; certain individuals, however, have an unusually high number of pyrroles in their bodies, resulting in a condition known as pyroluria. Pyroluria is becoming more and more prevalent both in children with ADHD and autism and in the general population. It is a little known condition; it was first given the name 'mauve factor. Pyroluria is a genetically acquired chemical imbalance in which the body produces an abnormally large number of pyrroles. A pyrrole is a chemical that is the by-product of haemoglobin synthesis and have no known function in the body; they are normally excreted in the urine. Pyroluria occurs when the pyrroles bind to pyroxidine (vitamin B6) and zinc, causing these vital nutrients to be excreted from the body in large amounts. Deficiencies of B6 and zinc are associated with a wide range of emotional and psychiatric problems. Vitamin B6 is critical for the production of neurotransmitters and deficiencies can have a profound effect on both mental and physical health. Nervousness, extreme irritability, anxiety, depression, short-term memory problems, and explosive anger have all been linked to Proluria. A large percentage of patients with psychiatric disorders such as schizophrenia exhibit high levels of pyrroles; emotionally dysregulated children and those with an excessive alcohol intake also tend to have an abnormally high pyrrole count. In addition, zinc deficiencies have been associated with a number of physiological disorders, including poor immune function, poor growth, and delayed sexual development. Because zinc and B6 are so important to both our overall physical and mental health, identifying and treating this devastating condition is critical. Included in this condition are changes to fatty acid metabolism which lead to high levels of an omega 6 fatty acid call arachidonic acid. Current research shows that neurological disorders such as ASD as well as psychiatric disorders have a high rate of remission with pyrrole treatment. Pyrrole Disorder can also be triggered by a traumatic event and high stress levels and is also often part of the picture for PDD, ODD, Tics, Tourette's Syndrome, Depression, and Bipolar Disorder. Treatment of Pyroluria consists of a replacement of zinc and vitamin B6. Because the treatment is replacing deficiencies not pharmacologic, it needs to be titrated to individual requirements. Common symptoms associated with Pyrrole Disorder in children (may not include all symptoms): · Sleep problems, particularly taking a long time to fall asleep and waking up easily at night · Sensory sensitivity i.e. to the labels on clothes, to specific fabrics – e.g. 'feels prickly', to noises. · Anxiety. (Anxiety can contribute to digestive upsets such as IBS) · Poor tolerance of stress · Depression, pessimistic outlook, mood swings, emotional lability · Anger and violence out of proportion to the situation, including rage · Poor functioning of the digestive system, including poor absorption of nutrients as well as food sensitivities · Low immunity – constantly suffering with coughs and colds · Motion sickness · Reading and learning problems · Poor memory · Slow growth · Delayed puberty · Unpleasant body odour · Stretch marks on skin · Pain in joints and extremities · Sweet fruity breath and body odour, problems with sugar metabolism, allergies
xylene	Reproductive effector in rats
ethylbenzene	Liver changes, utheral tract, effects on fertility, foetotoxicity, specific developmental abnormalities (musculoskeletal system) recorded. Ethylbenzene is readily absorbed following inhalation, oral, and dermal exposures, distributed throughout the body, and excreted primarily through urine. There are two different metabolic pathways for ethylbenzene with the primary pathway being the alpha-oxidation of ethylbenzene to 1-phenylethanol, mostly as the R-enantiomer. The pattern of urinary metabolite excretion varies with different mammalian species. In humans, ethylbenzene is excreted in the urine as mandelic acid and phenylgloxylic acids; whereas rats and rabbits excrete hippuric acid and phenaceturic acid as the main metabolites. Ethylbenzene can induce liver enzymes and hence its own metabolism as well as the metabolism of other substances. Ethylbenzene has a low order of acute toxicity by the oral, dermal or inhalation routes of exposure. Studies in rabbits indicate that ethylbenzene is irritating to the skin and eyes. There are numerous repeat dose studies available in a variety of species, these include: rats, mice, rabbits, guinea pig and rhesus monkeys. Hearing loss has been reported in rats (but not guinea pigs) exposed to relatively high exposures *(400 ppm and greater)* of ethylbenzene In chronic toxicity/carcinogenicity studies, both rats and mice were exposed via inhalation to 0, 75, 250 or 750 ppm for 104 weeks. In rats, the kidney was the target organ of toxicity, with renal tubular hyperplasia noted in both males and females at the 750 ppm level only. In mice, the liver and lung were the principal target organs of toxicity. In male mice at 750 ppm, lung toxicity was described as alveolar epithelial metaplasia, and liver toxicity was described as hepatocellular syncitial alteration, hypertrophy and mild necrosis; this was accompanied by increased follicular cell hyperplasia in the thyroid. As a result the NOAEL in male mice was determined to be 250 ppm. In female mice, the 750 ppm dose group had an increased incidence of eosinophilic foci in the liver (44% vs 10% in the controls) and an increased incidence in follicular cell hyperplasia in the thyroid gland. In studies conducted by the U.S. National Toxicology Program, inhalation of ethylbenzene at 750 ppm resulted in increased lung tumors in male mice, liver tumors in female mice, and increased kidney tumors in male and female rats. No increase in tumors was reported at 75 or 250 ppm. Ethylbenzene is considered to be an animal carcinogen, however, the relevance of these findings to humans is currently unknown. Although no reproductive toxicity studies have been conducted on ethylbenzene, repeated-dose studies indicate that the reproductive organs are not a target for ethylbenzene toxicity Ethylbenzene was negative in bacterial gene mutation tests and in a yeast assay on mitotic recombination. **NOTE:** Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.
1-Methyl-2-Pyrrolidone	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.

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	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 A substance (or part of a group of chemical substances) of very high concern (SVHC) - or product containing an SVHC: It is proposed that use within the European Union be subject to authorisation under the REACH Regulation.Indeed, listing of a substance as an SVHC by the European Chemicals Agency (ECHA) is the first step in the procedure for authorisation or restriction of use of a chemical. The criteria are given in article 57 of the REACH Regulation. A substance may be proposed as an SVHC if it meets one or more of the following criteria: ▸ it is carcinogenic *; ▸ it is mutagenic *; ▸ it is toxic for reproduction *; ▸ it is persistent, bioaccumulative and toxic (PBT substances); ▸ it is very persistent and very bioaccumulative (vPvB substances); ▸ there is "scientific evidence of probable serious effects to human health or the environment which give rise to an equivalent level of concern"; such substances are identified on a case-by-case basis. * Collectively described as CMR substances The "equivalent concern" criterion is significant because it is this classification which allows substances which are, for example, neurotoxic, endocrine-disrupting or otherwise present an unanticipated environmental health risk to be regulated under REACH] Simply because a substance meets one or more of the criteria does not necessarily mean that it will be proposed as an SVHC. Many such substances are already subject to restrictions on their use within the European Union, such as those in Annex XVII of the REACH Regulation SVHCs are substances for which the current restrictions on use (where these exist) might be insufficient. There are three priority groups for assessment: ▸ PBT substances and vPvB substances; ▸ substances which are widely dispersed during use; ▸ substances which are used in large quantities.
distillates, petroleum, middle, sweetened	For aviation fuels: Kerosene (straight-run and hydrodesulfurised) and related jet fuels (e.g., JP-5, JP-8, Jet-A, Jet-A1) were selected for characterisation of health effects considered representative of the aviation fuels. Kerosene is similar to aviation turbine fuel (CAS RN 64741-86-2) from both a process and physical-chemical perspective, but is refined to less stringent requirements and is not subject to the same additives as final aviation fuels. JP-5, JP-8 and Jet-A are military and commercial grades of aviation turbine fuel, and are therefore also relevant for consideration in the health effects assessment of aviation fuels. Acute toxicity: Overall, aviation fuels have low acute oral (median lethal dose [LD50] > 5000 mg/kg-bw) dermal toxicity (LD50 >5000 mg/kg b.w) and inhalation toxicity (LC50 > 5000 mg/m3) for exposure to mammals. They are not skin sensitizers, but can produce eye and skin irritation (mild and mild-to-severe, respectively) A one-hour nose-only exposure of female C57Bl/6 mice to 1000 mg/m3 JP-8 caused immediate immunosuppression, a significant loss of viable immune cells and significantly reduced immune organ weights Additional one-hour exposures resulted in greater immunosuppression. Skin irritation was the only effect reported after dermal exposure of male and female Sprague-Dawley (SD) rats to 678 mg/kg-bw per day of aviation gasoline fuel (CAS RN 64741-87-3) 5 days per week for 4 weeks Increased spleen weights and decreased red blood cells were observed in rabbits dermally exposed to 200 mg/kg-bw (a lowest-observed-adverse-effect level [LOAEL]) kerosene 3 times per week for 4 weeks Immunosuppression (as indicated by impaired induction of contact hypersensitivity and suppression of the delayed-type hypersensitivity response) in female mice was seen after dermal exposure to 1140 mg/kg-bw of JP-8 once per day for 5 days. In female SD rats, immunosuppression was not observed after dermal exposure to Jet-A at 495 mg/kg-bw per day for 4 weeks. Generalised sloughing of the bronchiolar epithelium and various cellular changes in alveolar type II epithelial cells, including increased number

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	and size of surfactant-producing lamellar bodies, was observed in male C57Bl/6 mice that were nose-only exposed to JP-8 vapours and aerosols at 45 mg/m3 (a LOAEC) for 1 hour per day for 7 days. In another study, groups of male B6.A.D. mice were exposed to 0, 7, 12, 26, 48 and 118 mg/m3 JP-8 for 1 hour per day for 7 days The vapour/aerosol combination in this study would have resulted in actual exposures of 0, 57, 97, 211, 390 and 960 mg/m3. Thus, exposure to 390 mg/m3 resulted in increased alveolar permeability, increased total protein in the bronchoalveolar lavage fluid, and concentration-dependent morphological lung and alveolar injury. Although these effects occurred in the absence of impaired respiratory function, they were considered by the authors as adverse because they exhibited concentration-dependency and are predictive of longer-term respiratory damage. In another study of mice exposed to JP-8, significant concentration-dependent decreases in thymic cell viability and splenic immune cell proliferation have been noted at 810 mg/m3 , the lowest concentration tested (the 100 mg/m3 exposure group was actually exposed to 810 mg/m3 combined vapour/aerosols) Mice exposed to 1000 mg/m3 for one 1 per day for 7 days exhibited reduced immune response to influenza viral infection,decreased immune cell viability, decreased immune cell proliferative response to mitogens, and a loss of T cells from the lymph nodes . Exposure of rats to 1000 mg/m3 JP-8 for 6 hours per day, 5 days per week for 6 weeks, had significant effects on neurobehavioural capacity. Inhalation exposure to JP-5 at 750 mg/m3 resulted in decreased growth rate in male rats, and statistically significant increases in blood urea nitrogen and serum creatinine levels in both sexes. Another inhalation study showed bone marrow histological changes (10% reduction in fat cells), as well as low-level cell proliferation in male rats exposed to 250 mg/m3 JP-5 . No adverse effects were reported in rats administered 3000 mg/kg JP-8 by gavage daily for 90 days In a subchronic dermal study, dose-dependent skin irritation and increased spleen weights in high-dose females were reported after male and female SD rats were exposed to 165, 330 or 495 mg/kg-bw hydrodesulfurized kerosene daily for 13 weeks Developmental toxicity: (Kerosene and Jet-A did not elicit developmental toxicity in inhalation studies conducted in rats (however, in a study conducted with JP-8 in mice, a lowest-observed-adverse effect concentration (LOAEC) of 1000 mg/m3 was established for maternal, reproductive and developmental toxicity Conversely, developmental effects were observed in C57Bl/6 mice exposed to a maternally toxic concentration of JP-8. Mouse dams were exposed, nose-only, to 1000 mg/m3 JP-8 aerosols, in a single-concentration study, 1 time per day from gestational days 7 or 15 to birth. Adverse effects occurred in dams and pups of both groups, and included statistically significant immunosuppression as measured at 6 to 8 weeks postpartum. Other statistically significant effects included decreased spleen weights and splenic cells (pups), decreased thymus weights and precursor T cells (dams and pups), and decreased litter sizes. Male pup birth and survival rates were also decreased Carcinogenity: As the predominant route of exposure to aviation fuels is determined to be inhalation, estimates of cancer potency for inhalation of benzene (a component) were used to characterize risk to the general population from evaporative emissions of aviation fuels. The International Agency for Research on Cancer (IARC) classified “jet fuel” (CAS RN not assigned) as a group 3 carcinogen (“not classifiable as to its carcinogenicity to humans” – inadequate data in humans and inadequate or limited data in animals) (IARC 1989a). In deriving this classification, IARC in part considered health effects data on kerosene (CAS RN 8008-20-6). The aviation fuels (CAS RNs 64741-86-2, 64741-87-3 and 68527-27-5) were classified as EU category 2 carcinogens (“may cause cancer”) by the European Commission (European Commission 2004; ESIS c1995-2012). The risk phrases R65 for classification and labelling (“harmful: may cause lung damage if swallowed”) and R46 (“may cause heritable genetic damage”) were also assigned by the European Commission to CAS RNs 64741-87-3 and 68527-27-5 Genotoxicity: Aviation fuels exhibited mixed results in in vitro and in vivo genotoxicity assays.Results from limited studies in laboratory animals indicated the potential for developmental health effects at high concentrations in mice but not in rats. The potential for exposure of the general population to evaporative emissions of aviation fuel at airports and in the vicinity of bulk storage facilities has been evaluated. For non-cancer effects, margins of exposure between upper-bounding estimates of exposure and critical effect levels identified in laboratory animals are considered adequate to address uncertainties in the health effects and exposure databases. For cancer, margins of exposure between upper-bounding estimates of exposure and estimates of cancer potency are considered adequate to address uncertainties related to health effects and exposure. Aviation Fuels: Final Screening Assessment Petroleum Sector Stream Approach - Environment Canada Health Canada April 2014
Naphtha, Heavy Aromatic	For petroleum: This product contains benzene, which can cause acute myeloid leukaemia, and n-hexane, which can be metabolized to compounds which are toxic to the nervous system. This product contains toluene, and animal studies suggest high concentrations of toluene lead to hearing loss. This product contains ethyl benzene and naphthalene, from which animal testing shows evidence of tumour formation. Cancer-causing potential: Animal testing shows inhaling petroleum causes tumours of the liver and kidney; these are however not considered to be relevant in humans. Mutation-causing potential: Most studies involving gasoline have returned negative results regarding the potential to cause mutations, including all recent studies in living human subjects (such as in petrol service station attendants). Reproductive toxicity: Animal studies show that high concentrations of toluene (>0.1%) can cause developmental effects such as lower birth weight and developmental toxicity to the nervous system of the foetus. Other studies show no adverse effects on the foetus. Human effects: Prolonged or repeated contact may cause defatting of the skin which can lead to skin inflammation and may make the skin more susceptible to irritation and penetration by other materials. Animal testing shows that exposure to gasoline over a lifetime can cause kidney cancer, but the relevance in humans is questionable.
1-methylnaphthalene	Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens). Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis. Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure.
naphthalene	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
toluene	For toluene: **Acute Toxicity** Humans exposed to intermediate to high levels of toluene for short periods of time experience adverse central nervous system effects ranging from headaches to intoxication, convulsions, narcosis, and death. Similar effects are observed in short-term animal studies. **Humans -** Toluene ingestion or inhalation can result in severe central nervous system depression, and in large doses, can act as a narcotic. The ingestion of about 60 mL resulted in fatal nervous system depression within 30 minutes in one reported case. Constriction and necrosis of myocardial fibers, markedly swollen liver, congestion and haemorrhage of the lungs and acute tubular necrosis were found on autopsy. Central nervous system effects (headaches, dizziness, intoxication) and eye irritation occurred following inhalation exposure to 100 ppm toluene 6 hours/day for 4 days. Exposure to 600 ppm for 8 hours resulted in the same and more serious symptoms including euphoria, dilated pupils, convulsions, and nausea . Exposure to 10,000-30,000 ppm has been reported to cause narcosis and death Toluene can also strip the skin of lipids causing dermatitis **Animals -** The initial effects are instability and incoordination, lachrymation and sniffles (respiratory exposure), followed by narcosis. Animals die of respiratory failure from severe nervous system depression. Cloudy swelling of the kidneys was reported in rats following inhalation exposure to 1600 ppm, 18-20 hours/day for 3 days **Subchronic/Chronic Effects:**

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	Repeat doses of toluene cause adverse central nervous system effects and can damage the upper respiratory system, the liver, and the kidney. Adverse effects occur as a result from both oral and the inhalation exposures. A reported lowest-observed-effect level in humans for adverse neurobehavioral effects is 88 ppm. **Humans -** Chronic occupational exposure and incidences of toluene abuse have resulted in hepatomegaly and liver function changes. It has also resulted in nephrotoxicity and, in one case, was a cardiac sensitiser and fatal cardiotoxin. Neural and cerebellar dystrophy were reported in several cases of habitual "glue sniffing." An epidemiological study in France on workers chronically exposed to toluene fumes reported leukopenia and neutropenia. Exposure levels were not given in the secondary reference; however, the average urinary excretion of hippuric acid, a metabolite of toluene, was given as 4 g/L compared to a normal level of 0.6 g/L **Animals -** The major target organs for the subchronic/chronic toxicity of toluene are the nervous system, liver, and kidney. Depressed immune response has been reported in male mice given doses of 105 mg/kg/day for 28 days. Toluene in corn oil administered to F344 male and female rats by gavage 5 days/week for 13 weeks, induced prostration, hypoactivity, ataxia, piloerection, lachrymation, excess salivation, and body tremors at doses 2500 mg/kg. Liver, kidney, and heart weights were also increased at this dose and histopathologic lesions were seen in the liver, kidneys, brain and urinary bladder. The no-observed-adverse effect level (NOAEL) for the study was 312 mg/kg (223 mg/kg/day) and the lowest-observed-adverse effect level (LOAEL) for the study was 625 mg/kg (446 mg/kg/day) . **Developmental/Reproductive Toxicity** Exposures to high levels of toluene can result in adverse effects in the developing human foetus. Several studies have indicated that high levels of toluene can also adversely effect the developing offspring in laboratory animals. **Humans -** Variable growth, microcephaly, CNS dysfunction, attentional deficits, minor craniofacial and limb abnormalities, and developmental delay were seen in three children exposed to toluene in utero as a result of maternal solvent abuse before and during pregnancy **Animals** - Sternebral alterations, extra ribs, and missing tails were reported following treatment of rats with 1500 mg/m3 toluene 24 hours/day during days 9-14 of gestation. Two of the dams died during the exposure. Another group of rats received 1000 mg/m3 8 hours/day during days 1-21 of gestation. No maternal deaths or toxicity occurred, however, minor skeletal retardation was present in the exposed fetuses. CFLP Mice were exposed to 500 or 1500 mg/m3 toluene continuously during days 6-13 of pregnancy. All dams died at the high dose during the first 24 hours of exposure, however none died at 500 mg/m3. Decreased foetal weight was reported, but there were no differences in the incidences of skeletal malformations or anomalies between the treated and control offspring. **Absorption** - Studies in humans and animals have demonstrated that toluene is readily absorbed via the lungs and the gastrointestinal tract. Absorption through the skin is estimated at about 1% of that absorbed by the lungs when exposed to toluene vapor. Dermal absorption is expected to be higher upon exposure to the liquid; however, exposure is limited by the rapid evaporation of toluene . **Distribution** - In studies with mice exposed to radiolabeled toluene by inhalation, high levels of radioactivity were present in body fat, bone marrow, spinal nerves, spinal cord, and brain white matter. Lower levels of radioactivity were present in blood, kidney, and liver. Accumulation of toluene has generally been found in adipose tissue, other tissues with high fat content, and in highly vascularised tissues . **Metabolism** - The metabolites of inhaled or ingested toluene include benzyl alcohol resulting from the hydroxylation of the methyl group. Further oxidation results in the formation of benzaldehyde and benzoic acid. The latter is conjugated with glycine to yield hippuric acid or reacted with glucuronic acid to form benzoyl glucuronide. o-cresol and p-cresol formed by ring hydroxylation are considered minor metabolites **Excretion -** Toluene is primarily (60-70%) excreted through the urine as hippuric acid. The excretion of benzoyl glucuronide accounts for 10-20%, and excretion of unchanged toluene through the lungs also accounts for 10-20%. Excretion of hippuric acid is usually complete within 24 hours after exposure.
2-Propanol	For isopropanol (IPA): **Acute toxicity:** Isopropanol has a low order of acute toxicity. It is irritating to the eyes, but not to the skin. Very high vapor concentrations are irritating to the eyes, nose, and throat, and prolonged exposure may produce central nervous system depression and narcosis. Human volunteers reported that exposure to 400 ppm isopropanol vapors for 3 to 5 min. caused mild irritation of the eyes, nose and throat. Although isopropanol produced little irritation when tested on the skin of human volunteers, there have been reports of isolated cases of dermal irritation and/or sensitization. The use of isopropanol as a sponge treatment for the control of fever has resulted in cases of intoxication, probably the result of both dermal absorption and inhalation. There have been a number of cases of poisoning reported due to the intentional ingestion of isopropanol, particularly among alcoholics or suicide victims. These ingestions typically result in a comatose condition. Pulmonary difficulty, nausea, vomiting, and headache accompanied by various degrees of central nervous system depression are typical. In the absence of shock, recovery usually occurred. **Repeat dose studies**: The systemic (non-cancer) toxicity of repeated exposure to isopropanol has been evaluated in rats and mice by the inhalation and oral routes. The only adverse effects-in addition to clinical signs identified from these studies were to the kidney. **Reproductive toxicity**: A recent two-generation reproductive study characterised the reproductive hazard for isopropanol associated with oral gavage exposure. This study found that the only reproductive parameter apparently affected by isopropanol exposure was a statistically significant decrease in male mating index of the F1 males. It is possible that the change in this reproductive parameter was treatment related and significant, although the mechanism of this effect could not be discerned from the results of the study. However, the lack of a significant effect of the female mating index in either generation, the absence of any adverse effect on litter size, and the lack of histopathological findings of the testes of the high-dose males suggest that the observed reduction in male mating index may not be biologically meaningful. **Developmental toxicity:** The developmental toxicity of isopropanol has been characterized in rat and rabbit developmental toxicity studies. These studies indicate that isopropanol is not a selective developmental hazard. Isopropanol produced developmental toxicity in rats, but not in rabbits. In the rat, the developmental toxicity occurred only at maternally toxic doses and consisted of decreased foetal body weights, but no teratogenicity **Genotoxicity:** All genotoxicity assays reported for isopropanol have been negative **Carcinogenicity:** rodent inhalation studies were conduct to evaluate isopropanol for cancer potential. The only tumor rate increase seen was for interstitial (Leydig) cell tumors in the male rats. Interstitial cell tumors of the testis is typically the most frequently observed spontaneous tumor in aged male Fischer 344 rats. These studies demonstrate that isopropanol does not exhibit carcinogenic potential relevant to humans. Furthermore, there was no evidence from this study to indicate the development of carcinomas of the testes in the male rat, nor has isopropanol been found to be genotoxic. Thus, the testicular tumors seen in the isopropanol exposed male rats are considered of no significance in terms of human cancer risk assessment
MOPAR Air Intake & EGR Cleaner (Liquid - Non-Aerosol) & ACETONE	for acetone: The acute toxicity of acetone is low. Acetone is not a skin irritant or sensitiser but is a defatting agent to the skin. Acetone is an eye irritant. The subchronic toxicity of acetone has been examined in mice and rats that were administered acetone in the drinking water and again in rats treated by oral gavage. Acetone-induced increases in relative kidney weight changes were observed in male and female rats used in the oral 13-week study. Acetone treatment caused increases in the relative liver weight in male and female rats that were not associated with histopathologic effects and the effects may have been associated with microsomal enzyme induction. Haematologic effects consistent with macrocytic anaemia were also noted in male rats along with hyperpigmentation in the spleen. The most notable findings in the mice were increased liver and decreased spleen weights. Overall, the no-observed-effect-levels in the drinking water study were 1% for male rats (900 mg/kg/d) and male mice (2258 mg/kg/d), 2% for female mice (5945 mg/kg/d), and 5% for female rats (3100 mg/kg/d). For developmental effects, a statistically significant reduction in foetal weight, and a slight, but statistically significant increase in the percent incidence of later resorptions were seen in mice at 15,665 mg/m3 and in rats at 26,100 mg/m3. The no-observable-effect level for developmental toxicity was determined to be 5220 mg/m3 for both rats and mice. Teratogenic effects were not observed in rats and mice tested at 26,110 and 15,665 mg/m3, respectively. Lifetime dermal carcinogenicity studies in mice treated with up to 0.2 mL of acetone did not reveal any increase in organ tumor incidence relative to untreated control animals. The scientific literature contains many different studies that have measured either the neurobehavioural performance or neurophysiological response of humans exposed to acetone. Effect levels ranging from about 600 to greater than 2375 mg/m3 have been reported. Neurobehavioral studies with acetone-exposed employees have recently shown that 8-hr exposures in excess of 2375 mg/m3 were not associated with any dose-related changes in response time, vigilance, or digit span scores. Clinical case studies, controlled human volunteer studies, animal research, and occupational field evaluations all indicate that the NOAEL for this effect is 2375 mg/m3 or greater.

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xylene & ethylbenzene	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
xylene & toluene	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.
xylene & 2-Propanol	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.
ethylbenzene & ACETONE & naphthalene & 2-Propanol	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
ethylbenzene & naphthalene	**WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
distillates, petroleum, light, hydrotreated & distillates, petroleum, middle, sweetened	No significant acute toxicological data identified in literature search. For "kerosenes" **Acute toxicity:** Oral LD50s for three kerosenes (Jet A, CAS No. 8008-20-6 and CAS No. 64742-81-0) ranged from > 2 to >20 g/kg The dermal LD50s of the same three kerosenes were all >2.0 g//kg. Inhalation LC50 values in Sprague-Dawley rats for straight run kerosene (CAS No. 8008-20-6) and hydrodesulfurised kerosene (CAS No. 64742-81-0) were reported to be > 5 and > 5.2 mg/l, respectively. No mortalities in rats were reported in rats when exposed for eight hours to saturated vapor of deodorised kerosene (probably a desulfurised kerosene). Six hour exposures of cats to the same material produced an LC50 of >6.4 mg/l When tested in rabbits for skin irritation, straight run kerosene (CAS No. 8008-20-6) produced “moderate” to “severe” irritation. Six additional skin irritation studies on a range of kerosenes produced “mild” to “severe” irritation. An eye irritation in rabbits of straight run kerosene (CAS No. 8008-20-6) produced Draize scores of 0.7 and 2.0 (unwashed and washed eyes) at 1 hour. By 24 hours, the Draize scores had returned to zero. Eye irritation studies have also been reported for hydrodesulfurized kerosene and jet fuel. These materials produced more irritation in the unwashed eyes at 1 hour than had the straight run kerosene. The eye irritation persisted longer than that seen with straight run kerosene, but by day 7 had resolved. Straight run kerosene (CAS No. 8008-20-6), Jet A, and hydrodesulfurized kerosene (CAS No. 64742-81-0) have not produced sensitisation when tested in guinea pigs **Repeat-Dose toxicity:** Multiple repeat-dose toxicity studies have been reported on a variety of kerosenes or jet fuels. When applied dermally, kerosenes and jet fuels have been shown to produce dermal and systemic effects Dose levels of 200, 1000 and 2000 mg/kg of a straight run kerosene (CAS No. 8008-20-6) were applied undiluted to the skin of male and female New Zealand white rabbits The test material was applied 3x/week for 28 days. One male and one female in the 2000 mg/kg dose group found dead on days 10 and 24 respectively were thought to be treatment-related. Clinical signs that were considered to be treatment-related included: thinness, nasal discharge, lethargy, soiled anal area, anal discharge, wheezing. The high dose group appeared to have a treatment related mean body weight loss when compared to controls. Dose-related skin irritation was observed, ranging from “slight” to “moderate” in the low and high dose groups, respectively. Other treatment-related dermal findings included cracked, flaky and/or leathery skin, crusts and/or hair loss. Reductions in RBC, haemoglobin and haematocrit were seen in the male dose groups. There were no treatment related effects on a variety of clinical chemistry values. Absolute and relative weights for a number of organs were normal, with the following exceptions that were judged to be treatment-related: • increased relative heart weights for the mid- and high- dose males and females, • increased absolute and relative spleen weights in treated females, and • differences in absolute and relative adrenal weights in both male and female treated animals (considered to be stress-related and therefore, indirectly related to treatment). Gross necropsy findings were confined largely to the skin. Enlarged spleens were seen in the female groups. Microscopic examination of tissues taken at necropsy found proliferative inflammatory changes in the treated skin of all male and female animals in the high dose group. These changes were, in the majority of animals, accompanied by an increase in granulopoiesis of the bone marrow. Four of six high dose males had testicular changes (multifocal or diffuse tubular hypoplasia) that were considered by the study authors to be secondary to the skin and/or weight changes. In a different study, hydrodesulfurised kerosene was tested in a thirteen-week dermal study using Sprague-Dawley rats. Test material was applied 5x/week to the skin of male and female rats at dose levels of 165, 330 and 495 mg/kg. Aside from skin irritation at the site of application, there were no treatment-related clinical signs during the study. Screening of all animals using a functional observation battery (FOB) did not find any substance-related effects. Opthalomological examination of all animals also found no treatment-related effects. There were no treatment-related effects on growth rates, hematological or clinical chemical values, or absolute or relative organ weights. Microscopic examination of tissues from animals surviving to termination found no treatment-related changes, with the exception of a minimal degree of a proliferative and inflammatory changes in the skin. A hydrodesulfurised middle distillate (CAS no. 64742-80-9) has also been tested in a four week inhalation study . In the study, Sprague-Dawley rats were exposed to a nominal concentration of 25mg/m3 kerosene. Exposures were for approximately 6 hr/day, five days each week for four consecutive weeks. There were no treatment-related effects on clinical condition, growth rate, absolute or relative organ weights, or any of the hematological or clinical chemistry determinations. Microscopic examination found no treatment-related changes observed in any tissues. **Carcinogenicity:** In addition to the repeat-dose studies discussed above, a number of dermal carcinogenicity studies have been performed on kerosenes or jet fuels. .Following the discovery that hydrodesulfurised (HDS) kerosene caused skin tumors in lifetime mouse skin painting studies, the role of dermal irritation in tumor formation was extensively studied. HDS kerosene proved to be a mouse skin tumor promoter rather than initiator, and this promotion required prolonged dermal irritation . If the equivalent dose of kerosene was applied to the skin in manner that did not cause significant skin irritation (eg, dilution with a mineral oil) no skin tumors occurred . Dermal bioavailability studies in mice confirmed that the reduced irritation seen with samples in mineral oil was not due to decreased skin penetration . The effect of chronic acanthosis on the dermal tumorigenicity of a hydrodesulfurised kerosene was studied and the author concluded that hyperplasia was essential for tumor promotion. However, the author also concluded that subacute inflammation did not appear to be a significant factor A sample of a hydrodesulfurised kerosene has been tested in an initiation-promotion assay in male CD-1 mice . Animal survivals were not effected by exposure to the kerosene. The study's authors concluded that the kerosene was not an initiator but it did show tumor promoting activity. ***In-Vitro* (Genotoxicity):** The potential *in vitro* genotoxicities of kerosene and jet fuel have been evaluated in a variety of studies. Standard Ames assays on two kerosene samples and a sample of Jet A produced negative results with/without activation . Modified Ames assays on four kerosenes also produced negative results (with/without activation) except for one positive assay that occurred with activation . The testing of five kerosene and jet fuel samples in mouse lymphoma assays produced a mixture of negative and positive results . Hydrodesulfurized kerosene tested in a sister chromatid exchange assay produced negative results (with/without activation) ***In-Vivo* Genotoxicity:** Multiple *in vivo* genotoxicity studies have been done on a variety of kerosene-based materials. Four samples of kerosene were negative and a sample of Jet A was positive in *in vivo* bone marrow cytogenetic tests in Sprague-Dawley rats . One of the kerosene samples produced a positive response in male mice and negative results in females when tested in a sister chromatid exchange assay . Both deodorised kerosene and Jet A samples produced negative results in dominant lethal assays. The kerosene was administered to both mice and rats intraperitoneally, while the jet fuel was administered only to mice via inhalation. **Reproductive/Developmental Toxicity** Either 0, 20, 40 or 60% (v/v) kerosene in mineral oil was applied to the skin of the rats. The dose per body weight equivalents were 0, 165, 330 and 494 mg/kg. Test material was applied daily, 7 days/week from 14 days premating through 20 days of gestation. There were no treatment-related effects on mortality and no clinical signs of toxicity were observed. There were no compound-related effects on any of the reproductive/developmental parameters. The authors concluded that the no observable effect level (NOEL) for

Continued...

	reproductive/developmental toxicity of HDS kerosene under the treatment conditions of the study was 494 mg/kg/day. Developmental toxicity screening studies on a kerosene and a sample of Jet A have been reported . There were no compound-related deaths in either study. While kerosene produced no clinical signs, the jet fuel produced a dose-related eye irritation (or infection). The signs of irritation lasted from 2 to 8 days with most animals showing signs for 3 days. Neither of the test materials had an effect on body weights or food consumption. Examination of offspring at delivery did not reveal any treatment-related abnormalities, soft tissue changes or skeletal abnormalities. The sex ratio of the fetuses was also unaffected by treatment with either of the compounds.
distillates, petroleum, light, hydrotreated & distillates, petroleum, middle, sweetened & Naphtha, Heavy Aromatic	Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length,with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent that iso- or cyclo-paraffins. The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption,is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.
1-Methyl-2-Pyrrolidone & 2-methylnaphthalene & 1-methylnaphthalene & 2-Propanol	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. 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. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.
2-methylnaphthalene & 1-methylnaphthalene	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. Data demonstrate that during inhalation exposure,aromatic hydrocarbons undergo substantial partitioning into adipose tissues. Following cessation of exposure, the level of aromatic hydrocarbons in body fats rapidly declines. Thus, the aromatic hydrocarbons are unlikely to bioaccumulate in the body. Selective partitioning of the aromatic hydrocarbons into the non-adipose tissues is unlikely. No data is available regarding distribution following dermal absorption. However, distribution following this route of exposure is likely to resemble the pattern occurring with inhalation exposure. Aromatics hydrocarbons may undergo several different Phase I dealkylation, hydroxylation and oxidation reactions which may or may not be followed by Phase II conjugation to glycine, sulfation or glucuronidation. However, the major predominant biotransformation pathway is typical of that of the alkylbenzenes and consists of: (1) oxidation of one of the alkyl groups to an alcohol moiety; (2) oxidation of the hydroxyl group to a carboxylic acid; (3) the carboxylic acid is then conjugated with glycine to form a hippuric acid. The minor metabolites can be expected to consist of a complex mixture of isomeric triphenols, the sulfate and glucuronide conjugates of dimethylbenzyl alcohols, dimethylbenzoic acids and dimethylhippuric acids. Consistent with the low propensity for bioaccumulation of aromatic hydrocarbons, these substances are likely to be significant inducers of their own metabolism. The predominant route of excretion of aromatic hydrocarbons following inhalation exposure involves either exhalation of the unmetabolized parent compound, or urinary excretion of its metabolites. When oral administration occurs, there is little exhalation of unmetabolized these hydrocarbons, presumably due to the first pass effect in the liver. Under these circumstances, urinary excretion of metabolites is the dominant route of excretion.

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

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

SECTION 12 Ecological information

Toxicity

MOPAR Air Intake & EGR Cleaner (Liquid - Non-Aerosol)

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

xylene

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	4.6mg/l	2
EC50	48h	Crustacea	1.8mg/l	2
NOEC(ECx)	73h	Algae or other aquatic plants	0.44mg/l	2
LC50	96h	Fish	2.6mg/l	2

ethylbenzene

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	4.6mg/l	1
EC50	48h	Crustacea	1.37-4.4mg/l	4
NOEC(ECx)	720h	Fish	0.381mg/L	4

Continued...

	LC50	96h	Fish	3.381-4.075mg/L	4
	EC50	96h	Algae or other aquatic plants	3.6mg/l	2

distillates, petroleum, light, hydrotreated

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	3072h	Fish	1mg/l	1

acetone

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

1-Methyl-2-Pyrrolidone

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	504h	Crustacea	12.5mg/l	2
EC50	72h	Algae or other aquatic plants	>500mg/l	1
EC50	48h	Crustacea	ca.4897mg/l	1
LC50	96h	Fish	464mg/l	1

distillates, petroleum, middle, sweetened

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	288h	Algae or other aquatic plants	20mg/l	1

Polyether Amine

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

Naphtha, Heavy Aromatic

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	48h	Crustacea	0.95mg/l	1
EC50	72h	Algae or other aquatic plants	<1mg/l	1
EC50	48h	Crustacea	0.95mg/l	1
LC50	96h	Fish	2-5mg/l	Not Available
EC50	96h	Algae or other aquatic plants	1mg/l	2

2-methylnaphthalene

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	96h	Crustacea	1.3mg/L	5
EC50	48h	Crustacea	5mg/L	5
LC50	96h	Fish	9mg/l	Not Available

1-methylnaphthalene

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	24h	Crustacea	1.61mg/L	5
EC50	48h	Crustacea	8.2mg/L	5

naphthalene

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	1.09-3.4mg/l	4
LC50	96h	Fish	0.51mg/l	4
BCF	1344h	Fish	23-146	7
NOEC(ECx)	48h	Fish	0.013mg/L	4
EC50	72h	Algae or other aquatic plants	~0.4~0.5mg/l	2

toluene

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	3.78mg/L	5
NOEC(ECx)	168h	Crustacea	0.74mg/L	5
LC50	96h	Fish	5-35mg/l	4
EC50	96h	Algae or other aquatic plants	>376.71mg/L	4

2-Propanol

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

Legend: *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA,*

Continued...

	Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data

For Ketones: Ketones, unless they are alpha, beta--unsaturated ketones, can be considered as narcosis or baseline toxicity compounds.
Aquatic Fate: Hydrolysis of ketones in water is thermodynamically favourable only for low molecular weight ketones. Reactions with water are reversible with no permanent change in the structure of the ketone substrate. Ketones are stable to water under ambient environmental conditions. When pH levels are greater than 10, condensation reactions can occur which produce higher molecular weight products. Under ambient conditions of temperature, pH, and low concentration, these condensation reactions are unfavourable. Based on its reactions in air, it seems likely that ketones undergo photolysis in water.
Terrestrial Fate: It is probable that ketones will be biodegraded by micro-organisms in soil and water.
Ecotoxicity: Ketones are unlikely to bioconcentrate or biomagnify.
for acetone:
log Kow: -0.24
Half-life (hr) air: 312-1896
Half-life (hr) H2O surface water: 20
Henry's atm m3 /mol: 3.67E-05
BOD 5: 0.31-1.76,46-55%
COD: 1.12-2.07
ThOD: 2.2
BCF: 0.69
Environmental fate:
Acetone preferentially locates in the air compartment when released to the environment. A substantial amount of acetone can also be found in water, which is consistent with the high water to air partition coefficient and its small, but detectable, presence in rain water, sea water, and lake water samples. Very little acetone is expected to reside in soil, biota, or suspended solids. This is entirely consistent with the physical and chemical properties of acetone and with measurements showing a low propensity for soil absorption and a high preference for moving through the soil and into the ground water
In air, acetone is lost by photolysis and reaction with photochemically produced hydroxyl radicals; the estimated half-life of these combined processes is about 22 days. The relatively long half-life allows acetone to be transported long distances from its emission source.
Acetone is highly soluble and slightly persistent in water, with a half-life of about 20 hours; it is minimally toxic to aquatic life.
Acetone released to soil volatilises although some may leach into the ground where it rapidly biodegrades.
Acetone does not concentrate in the food chain.
Acetone meets the OECD definition of readily biodegradable which requires that the biological oxygen demand (BOD) is at least 70% of the theoretical oxygen demand (THOD) within the 28-day test period
Drinking Water Standard: none available.
Soil Guidelines: none available.
Air Quality Standards: none available.
Ecotoxicity:
Testing shows that acetone exhibits a low order of toxicity
Fish LC50: brook trout 6070 mg/l; fathead minnow 15000 mg/l
Bird LC0 (5 day): Japanese quail, ring-neck pheasant 40,000 mg/l
Daphnia magna LC50 (48 h): 15800 mg/l; NOEC 8500 mg/l
Aquatic invertebrate 2100 - 16700 mg/l
Aquatic plant NOEC: 5400-7500 mg/l
Daphnia magna chronic NOEC 1660 mg/l
Acetone vapors were shown to be relatively toxic to two types insects and their eggs. The time to 50% lethality (LT50) was found to be 51.2 hr and 67.9 hr when the flour beetle (*Tribolium confusum*) and the flour moth (*Ephestia kuehniella*) were exposed to an airborne acetone concentration of 61.5 mg/m3. The LT50 values for the eggs were 30-50% lower than for the adult. The direct application of acetone liquid to the body of the insects or surface of the eggs did not, however, cause any mortality.
The ability of acetone to inhibit cell multiplication has been examined in a wide variety of microorganisms. The results have generally indicated mild to minimal toxicity with NOECs greater than 1700 mg/L for exposures lasting from 6 hr to 4 days. Longer exposure periods of 7 to 8 days with bacteria produced mixed results; but overall the data indicate a low degree of toxicity for acetone. The only exception to these findings were the results obtained with the flagellated protozoa (*Entosiphon sulcatum*) which yielded a 3-day NOEC of 28 mg/L.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
ethylbenzene	HIGH (Half-life = 228 days)	LOW (Half-life = 3.57 days)
acetone	LOW (Half-life = 14 days)	MEDIUM (Half-life = 116.25 days)
1-Methyl-2-Pyrrolidone	LOW	LOW
2-methylnaphthalene	HIGH	HIGH
1-methylnaphthalene	HIGH	HIGH
naphthalene	HIGH (Half-life = 258 days)	LOW (Half-life = 1.23 days)
toluene	LOW (Half-life = 28 days)	LOW (Half-life = 4.33 days)
2-Propanol	LOW (Half-life = 14 days)	LOW (Half-life = 3 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
xylene	MEDIUM (BCF = 740)
ethylbenzene	LOW (BCF = 79.43)
distillates, petroleum, light, hydrotreated	LOW (BCF = 159)
acetone	LOW (BCF = 0.69)
1-Methyl-2-Pyrrolidone	LOW (BCF = 0.16)
Naphtha, Heavy Aromatic	LOW (BCF = 159)
2-methylnaphthalene	MEDIUM (LogKOW = 3.86)
1-methylnaphthalene	MEDIUM (LogKOW = 3.87)
naphthalene	HIGH (BCF = 18000)
toluene	LOW (BCF = 90)

Continued...

Ingredient	Bioaccumulation
2-Propanol	LOW (LogKOW = 0.05)

Mobility in soil

Ingredient	Mobility
ethylbenzene	LOW (KOC = 517.8)
acetone	HIGH (KOC = 1.981)
1-Methyl-2-Pyrrolidone	LOW (KOC = 20.94)
2-methylnaphthalene	LOW (KOC = 2976)
1-methylnaphthalene	LOW (KOC = 3038)
naphthalene	LOW (KOC = 1837)
toluene	LOW (KOC = 268)
2-Propanol	HIGH (KOC = 1.06)

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. ▸ Recycle wherever possible. ▸ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▸ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material). ▸ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	NO

Land transport (DOT)

UN number	1993
UN proper shipping name	Flammable liquids, n.o.s. (contains xylene, distillates, petroleum, light, hydrotreated, distillates, petroleum, middle, sweetened and acetone)
Transport hazard class(es)	Class: 3 Subrisk: Not Applicable
Packing group	II
Environmental hazard	Not Applicable
Special precautions for user	Hazard Label: 3 Special provisions: IB2, T7, TP1, TP8, TP28

Air transport (ICAO-IATA / DGR)

UN number	1993
UN proper shipping name	Flammable liquid, n.o.s. * (contains xylene, distillates, petroleum, light, hydrotreated, distillates, petroleum, middle, sweetened and acetone)
Transport hazard class(es)	ICAO/IATA Class: 3 ICAO / IATA Subrisk: Not Applicable ERG Code: 3H
Packing group	II
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: A3 Cargo Only Packing Instructions: 364 Cargo Only Maximum Qty / Pack: 60 L

Continued...

Passenger and Cargo Packing Instructions	353
Passenger and Cargo Maximum Qty / Pack	5 L
Passenger and Cargo Limited Quantity Packing Instructions	Y341
Passenger and Cargo Limited Maximum Qty / Pack	1 L

Sea transport (IMDG-Code / GGVSee)

UN number	1993
UN proper shipping name	FLAMMABLE LIQUID, N.O.S. (contains xylene, distillates, petroleum, light, hydrotreated, distillates, petroleum, middle, sweetened and acetone)
Transport hazard class(es)	IMDG Class: 3 IMDG Subrisk: Not Applicable
Packing group	II
Environmental hazard	Not Applicable
Special precautions for user	EMS Number: F-E, S-E Special provisions: 274 Limited Quantities: 1 L

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

Not Applicable

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

Product name	Group
xylene	Not Available
ethylbenzene	Not Available
distillates, petroleum, light, hydrotreated	Not Available
acetone	Not Available
1-Methyl-2-Pyrrolidone	Not Available
distillates, petroleum, middle, sweetened	Not Available
Polyether Amine	Not Available
Naphtha, Heavy Aromatic	Not Available
2-methylnaphthalene	Not Available
1-methylnaphthalene	Not Available
naphthalene	Not Available
toluene	Not Available
2-Propanol	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
xylene	Not Available
ethylbenzene	Not Available
distillates, petroleum, light, hydrotreated	Not Available
acetone	Not Available
1-Methyl-2-Pyrrolidone	Not Available
distillates, petroleum, middle, sweetened	Not Available
Polyether Amine	Not Available
Naphtha, Heavy Aromatic	Not Available
2-methylnaphthalene	Not Available
1-methylnaphthalene	Not Available
naphthalene	Not Available
toluene	Not Available
2-Propanol	Not Available

SECTION 15 Regulatory information

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

xylene is found on the following regulatory lists

Continued...

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
US - California Hazardous Air Pollutants Identified as Toxic Air Contaminants
US - Massachusetts - Right To Know Listed Chemicals
US ATSDR Minimal Risk Levels for Hazardous Substances (MRLs)
US Clean Air Act - Hazardous Air Pollutants
US CWA (Clean Water Act) - List of Hazardous Substances
US DOE Temporary Emergency Exposure Limits (TEELs)
US EPA Integrated Risk Information System (IRIS)
US EPCRA Section 313 Chemical List
US OSHA Permissible Exposure Limits (PELs) Table Z-1
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

ethylbenzene is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
US - California Hazardous Air Pollutants Identified as Toxic Air Contaminants
US - California Proposition 65 - Carcinogens
US - California Proposition 65 - No Significant Risk Levels (NSRLs) for Carcinogens
US - California Safe Drinking Water and Toxic Enforcement Act of 1986 - Proposition 65 List
US - Massachusetts - Right To Know Listed Chemicals
US ATSDR Minimal Risk Levels for Hazardous Substances (MRLs)
US Clean Air Act - Hazardous Air Pollutants
US CWA (Clean Water Act) - List of Hazardous Substances
US CWA (Clean Water Act) - Priority Pollutants
US CWA (Clean Water Act) - Toxic Pollutants
US DOE Temporary Emergency Exposure Limits (TEELs)
US EPA Integrated Risk Information System (IRIS)
US EPCRA Section 313 Chemical List
US NIOSH Recommended Exposure Limits (RELs)
US OSHA Permissible Exposure Limits (PELs) Table Z-1
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

distillates, petroleum, light, hydrotreated is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 1: Carcinogenic to humans
US - California Proposition 65 - Carcinogens
US - California Safe Drinking Water and Toxic Enforcement Act of 1986 - Proposition 65 List
US DOE Temporary Emergency Exposure Limits (TEELs)
US National Toxicology Program (NTP) 15th Report Part A Known to be Human Carcinogens
US OSHA Permissible Exposure Limits (PELs) Table Z-1
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

acetone is found on the following regulatory lists

US - Massachusetts - Right To Know Listed Chemicals
US ATSDR Minimal Risk Levels for Hazardous Substances (MRLs)
US DOE Temporary Emergency Exposure Limits (TEELs)
US Drug Enforcement Administration (DEA) List I and II Regulated Chemicals
US EPA Integrated Risk Information System (IRIS)
US NIOSH Recommended Exposure Limits (RELs)
US OSHA Permissible Exposure Limits (PELs) Table Z-1
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances
US TSCA Section 4/12 (b) - Sunset Dates/Status

1-Methyl-2-Pyrrolidone is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
US - California Proposition 65 - Maximum Allowable Dose Levels (MADLs) for Chemicals Causing Reproductive Toxicity
US - California Proposition 65 - Reproductive Toxicity
US - California Safe Drinking Water and Toxic Enforcement Act of 1986 - Proposition 65 List
US - Massachusetts - Right To Know Listed Chemicals
US AIHA Workplace Environmental Exposure Levels (WEELs)
US DOE Temporary Emergency Exposure Limits (TEELs)
US EPCRA Section 313 Chemical List
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US Toxicology Excellence for Risk Assessment (TERA) Workplace Environmental Exposure Levels (WEEL)
US TSCA Chemical Substance Inventory - Interim List of Active Substances
US TSCA Section 12(b) - List of Chemical Substances Subject to Export Notification Requirements
US TSCA Section 4/12 (b) - Sunset Dates/Status

distillates, petroleum, middle, sweetened is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 1: Carcinogenic to humans
US - California Proposition 65 - Carcinogens
US - California Safe Drinking Water and Toxic Enforcement Act of 1986 - Proposition 65 List
US DOE Temporary Emergency Exposure Limits (TEELs)
US National Toxicology Program (NTP) 15th Report Part A Known to be Human Carcinogens
US OSHA Permissible Exposure Limits (PELs) Table Z-1
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

Polyether Amine is found on the following regulatory lists

Not Applicable

Naphtha, Heavy Aromatic is found on the following regulatory lists

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

2-methylnaphthalene is found on the following regulatory lists

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
US - Alaska Air Quality Control - Concentrations Triggering an Air Quality Episode for Air Pollutants Other Than PM-2.5
US ATSDR Minimal Risk Levels for Hazardous Substances (MRLs)
US Clean Air Act - Hazardous Air Pollutants
US DOE Temporary Emergency Exposure Limits (TEELs)
US EPA Integrated Risk Information System (IRIS)
US NIOSH Recommended Exposure Limits (RELs)
US OSHA Permissible Exposure Limits (PELs) Table Z-1
US OSHA Permissible Exposure Limits (PELs) Table Z-3
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

1-methylnaphthalene is found on the following regulatory lists

Continued...

US - Massachusetts - Right To Know Listed Chemicals
US ATSDR Minimal Risk Levels for Hazardous Substances (MRLs)
US Clean Air Act - Hazardous Air Pollutants
US DOE Temporary Emergency Exposure Limits (TEELs)
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

naphthalene is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
US - Alaska Air Quality Control - Concentrations Triggering an Air Quality Episode for Air Pollutants Other Than PM-2.5
US - California Hazardous Air Pollutants Identified as Toxic Air Contaminants
US - California Proposition 65 - Carcinogens
US - California Proposition 65 - No Significant Risk Levels (NSRLs) for Carcinogens
US - California Safe Drinking Water and Toxic Enforcement Act of 1986 - Proposition 65 List
US - Massachusetts - Right To Know Listed Chemicals
US ATSDR Minimal Risk Levels for Hazardous Substances (MRLs)
US Clean Air Act - Hazardous Air Pollutants
US CWA (Clean Water Act) - List of Hazardous Substances
US CWA (Clean Water Act) - Priority Pollutants
US CWA (Clean Water Act) - Toxic Pollutants
US DOE Temporary Emergency Exposure Limits (TEELs)
US EPA Integrated Risk Information System (IRIS)
US EPCRA Section 313 Chemical List
US National Toxicology Program (NTP) 15th Report Part B. Reasonably Anticipated to be a Human Carcinogen
US NIOSH Recommended Exposure Limits (RELs)
US OSHA Permissible Exposure Limits (PELs) Table Z-1
US OSHA Permissible Exposure Limits (PELs) Table Z-3
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances
US TSCA Section 4/12 (b) - Sunset Dates/Status

toluene is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
US - California Hazardous Air Pollutants Identified as Toxic Air Contaminants
US - California Proposition 65 - Maximum Allowable Dose Levels (MADLs) for Chemicals Causing Reproductive Toxicity
US - California Proposition 65 - Reproductive Toxicity
US - California Safe Drinking Water and Toxic Enforcement Act of 1986 - Proposition 65 List
US - Massachusetts - Right To Know Listed Chemicals
US ATSDR Minimal Risk Levels for Hazardous Substances (MRLs)
US Clean Air Act - Hazardous Air Pollutants
US CWA (Clean Water Act) - List of Hazardous Substances
US CWA (Clean Water Act) - Priority Pollutants
US CWA (Clean Water Act) - Toxic Pollutants
US DOE Temporary Emergency Exposure Limits (TEELs)
US Drug Enforcement Administration (DEA) List I and II Regulated Chemicals
US EPA Integrated Risk Information System (IRIS)
US EPCRA Section 313 Chemical List
US NIOSH Recommended Exposure Limits (RELs)
US OSHA Permissible Exposure Limits (PELs) Table Z-2
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

2-Propanol is found on the following regulatory lists

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
US - Massachusetts - Right To Know Listed Chemicals
US DOE Temporary Emergency Exposure Limits (TEELs)
US EPCRA Section 313 Chemical List
US NIOSH Recommended Exposure Limits (RELs)
US OSHA Permissible Exposure Limits (PELs) Table Z-1
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances
US TSCA Section 4/12 (b) - Sunset Dates/Status

Federal Regulations

Superfund Amendments and Reauthorization Act of 1986 (SARA)

Section 311/312 hazard categories

Flammable (Gases, Aerosols, Liquids, or Solids)	Yes
Gas under pressure	No
Explosive	No
Self-heating	No
Pyrophoric (Liquid or Solid)	No
Pyrophoric Gas	No
Corrosive to metal	No
Oxidizer (Liquid, Solid or Gas)	No
Organic Peroxide	No
Self-reactive	No
In contact with water emits flammable gas	No
Combustible Dust	No
Carcinogenicity	Yes
Acute toxicity (any route of exposure)	No
Reproductive toxicity	Yes
Skin Corrosion or Irritation	Yes
Respiratory or Skin Sensitization	No
Serious eye damage or eye irritation	Yes
Specific target organ toxicity (single or repeated exposure)	Yes
Aspiration Hazard	Yes
Germ cell mutagenicity	No

Continued...

Simple Asphyxiant	No
Hazards Not Otherwise Classified	Yes

US. EPA CERCLA Hazardous Substances and Reportable Quantities (40 CFR 302.4)

Name	Reportable Quantity in Pounds (lb)	Reportable Quantity in kg
xylene	100	45.4
ethylbenzene	1000	454
acetone	5000	2270
naphthalene	100	45.4
toluene	1000	454

State Regulations

US. California Proposition 65

WARNING: This product can expose you to chemicals including **ethylbenzene, distillates, petroleum, light, hydrotreated, distillates, petroleum, middle, sweetened, naphthalene**, which are known to the State of California to cause cancer, and **1-Methyl-2-Pyrrolidone, toluene**, which are known to the State of California to cause birth defects or other reproductive harm. For more information, go to www.P65Warnings.ca.gov.

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (xylene; ethylbenzene; distillates, petroleum, light, hydrotreated; acetone; 1-Methyl-2-Pyrrolidone; distillates, petroleum, middle, sweetened; Naphtha, Heavy Aromatic; naphthalene; toluene; 2-Propanol)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	No (2-methylnaphthalene; 1-methylnaphthalene)
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (distillates, petroleum, middle, sweetened; 1-methylnaphthalene)
Vietnam - NCI	Yes
Russia - FBEPH	No (distillates, petroleum, middle, sweetened)
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.*

SECTION 16 Other information

Revision Date	09/21/2022
Initial Date	08/05/2022

SDS Version Summary

Version	Date of Update	Sections Updated
1.2	09/21/2022	Classification, Ingredients, Name

Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.
The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
PC—STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists
STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit。
IDLH: Immediately Dangerous to Life or Health Concentrations
ES: Exposure Standard
OSF: Odour Safety Factor
NOAEL :No Observed Adverse Effect Level
LOAEL: Lowest Observed Adverse Effect Level
TLV: Threshold Limit Value
LOD: Limit Of Detection
OTV: Odour Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index
AIIC: Australian Inventory of Industrial Chemicals

Continued...

DSL: Domestic Substances List
NDSL: Non-Domestic Substances List
IECSC: Inventory of Existing Chemical Substance in China
EINECS: European INventory of Existing Commercial chemical Substances
ELINCS: European List of Notified Chemical Substances
NLP: No-Longer Polymers
ENCS: Existing and New Chemical Substances Inventory
KECI: Korea Existing Chemicals Inventory
NZIoC: New Zealand Inventory of Chemicals
PICCS: Philippine Inventory of Chemicals and Chemical Substances
TSCA: Toxic Substances Control Act
TCSI: Taiwan Chemical Substance Inventory
INSQ: Inventario Nacional de Sustancias Químicas
NCI: National Chemical Inventory
FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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end of SDS