MOPAR Premium Diesel Fuel Treatment

Mopar(FCA US LLC Service & Customer Care Division)

Chemwatch Hazard Alert Code: **3**

Part Number: **677**
Version No: **3.11**
Safety Data Sheet according to OSHA HazCom Standard (2012) requirements

Issue Date: **09/21/2022**
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SECTION 1 Identification

Product Identifier

Product name	MOPAR Premium Diesel Fuel Treatment
Synonyms	68621344AA, 68629555AA
Other means of identification	Not Available

Recommended use of the chemical and restrictions on use

Relevant identified uses	Fuel additive.

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	1	
Toxicity	1	
Body Contact	1	
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	Carcinogenicity Category 1B, Flammable Liquids Category 4, Aspiration Hazard Category 1

Label elements

Hazard pictogram(s)	

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Signal word	**Danger**

Hazard statement(s)

H350	May cause cancer.
H227	Combustible liquid.
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.
P280	Wear protective gloves and protective clothing.
P202	Do not handle until all safety precautions have been read and understood.

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.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

P501	Dispose of contents/container 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
64742-47-8*	50-70	distillates, petroleum, light, hydrotreated
27247-96-7*	10.5-21	2-ethylhexyl nitrate
64742-94-5*	3.5-7	solvent naphtha petroleum, heavy aromatic
64742-95-6.*	1.75-5.25	naphtha petroleum, light aromatic solvent
104-76-7*	1.75-5.25	2-ethylhexanol
64742-95-6.*	2-5	Heavy Aromatic Naptha
25551-13-7*	0.35-1.75	trimethylbenzenes
1330-20-7*	<0.7	Xylene (mixed isomers)
100-41-4*	<0.07	ethylbenzene
95-63-6*	<1.75	1,2,4-trimethyl benzene
98-82-8	<0.2	cumene
91-20-3*	0.35-1.75	naphthalene
108-67-8*	<0.07	Mesitylene

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 eyes: ▸ Wash out immediately with water. ▸ If irritation continues, seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.

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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.

SECTION 5 Fire-fighting measures

Extinguishing media

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

Special hazards arising from the substrate or mixture

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

Special protective equipment and precautions for fire-fighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Use water delivered as a fine spray to control fire and cool adjacent area. ▸ Avoid spraying water onto liquid pools. ▸ **DO NOT** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	▸ Combustible. ▸ Slight fire hazard when exposed to heat or flame. ▸ Heating may cause expansion or decomposition leading to violent rupture of containers. ▸ On combustion, may emit toxic fumes of carbon monoxide (CO). ▸ May emit acrid smoke. ▸ Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO_2) other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.

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 spill with sand, earth, inert material or vermiculite. ▸ Wipe up. ▸ Place in a suitable, labelled container for waste disposal.
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).

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	▸ 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.

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

SECTION 7 Handling and storage

Precautions for safe handling

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

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Metal can or drum ▸ Packaging as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▸ 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	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	Xylene (mixed isomers)	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 NIOSH Recommended Exposure Limits (RELs)	1,2,4-trimethyl benzene	1,2,4-Trimethylbenzene	25 ppm / 125 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	cumene	Cumene	50 ppm / 245 mg/m3	Not Available	Not Available	Skin designation
US NIOSH Recommended Exposure Limits (RELs)	cumene	Cumene	50 ppm / 245 mg/m3	Not Available	Not Available	[skin]
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: Total Dust	15 mg/m3 / 50 mppcf	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

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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US NIOSH Recommended Exposure Limits (RELs)	naphthalene	Naphthalene	10 ppm / 50 mg/m3	75 mg/m3 / 15 ppm	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	Mesitylene	1,3,5-Trimethylbenzene	25 ppm / 125 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
distillates, petroleum, light, hydrotreated	140 mg/m3	1,500 mg/m3	8,900 mg/m3
naphtha petroleum, light aromatic solvent	1,200 mg/m3	6,700 mg/m3	40,000 mg/m3
2-ethylhexanol	Not Available	Not Available	Not Available
Heavy Aromatic Naptha	1,200 mg/m3	6,700 mg/m3	40,000 mg/m3
Xylene (mixed isomers)	Not Available	Not Available	Not Available
ethylbenzene	Not Available	Not Available	Not Available
1,2,4-trimethyl benzene	140 mg/m3	360 mg/m3	2,200 mg/m3
1,2,4-trimethyl benzene	Not Available	Not Available	480 ppm
cumene	Not Available	Not Available	Not Available
naphthalene	15 ppm	83 ppm	500 ppm
Mesitylene	Not Available	Not Available	480 ppm

Ingredient	Original IDLH	Revised IDLH
distillates, petroleum, light, hydrotreated	2,500 mg/m3	Not Available
2-ethylhexyl nitrate	Not Available	Not Available
solvent naphtha petroleum, heavy aromatic	Not Available	Not Available
naphtha petroleum, light aromatic solvent	Not Available	Not Available
2-ethylhexanol	Not Available	Not Available
Heavy Aromatic Naptha	Not Available	Not Available
trimethylbenzenes	Not Available	Not Available
Xylene (mixed isomers)	900 ppm	Not Available
ethylbenzene	800 ppm	Not Available
1,2,4-trimethyl benzene	Not Available	Not Available
cumene	900 ppm	Not Available
naphthalene	250 ppm	Not Available
Mesitylene	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
naphtha petroleum, light aromatic solvent	C	> 1 to ≤ 10 parts per million (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

For cumene:
Odour Threshold Value: 0.008-0.132 ppm (detection), 0.047 ppm (recognition)
Exposure at or below the TLV-TWA is thought to prevent induction of narcosis.
NOTE P: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.01% w/w benzene (EINECS No 200-753-7). Note E shall also apply when the substance is classified as 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 "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.

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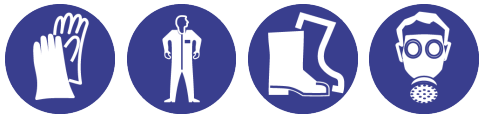
	▸ Open-vessel systems are prohibited. ▸ Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation. ▸ Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless 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 The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Body protection	See Other protection below
Other protection	▸ 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

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garments and hood.
- Overalls.
- P.V.C apron.
- Barrier cream.
- Skin cleansing cream.
- Eye wash unit.

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 Premium Diesel Fuel Treatment

Material	CPI
BUTYL	C
NEOPRENE	C
NITRILE	C
PVA	C
TEFLON	C
VITON	C

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

Respiratory protection

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

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

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

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

- Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Light amber to amber

Physical state	Liquid	**Relative density (Water = 1)**	0.853
Odour	Aromatic. Petroleum-like odor.	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	75	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Combustible.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Insoluble in water.	**pH as a solution (Not Available%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Continued...

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.
Ingestion	Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result. Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis). The material has **NOT** been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.
Skin Contact	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. Limited evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	Although the liquid is not thought to be an irritant (as classified by EC Directives), direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn).
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

MOPAR Premium Diesel Fuel Treatment	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
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]	
2-ethylhexyl nitrate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >4820 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50; >4.6 mg/kg/1h.[2]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; >9640 mg/kg[2]	
	Oral (Rat) LD50; 7500 mg/kg[2]	
solvent naphtha petroleum, 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]

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naphtha petroleum, light aromatic solvent	**TOXICITY**	**IRRITATION**
	Inhalation(Rat) LC50; >3670 ppm/8 h *[2]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; >5000 mg/kg *[2]	Skin: adverse effect observed (irritating)[1]
2-ethylhexanol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 1970 mg/kg[2]	Eye (rabbit): 20 mg/24h - moderate
	Oral (Rat) LD50; 2049 mg/kg[2]	Eye (rabbit): 4.17 mg - SEVERE
		Eye: adverse effect observed (irritating)[1]
		Skin (rabbit): 415 mg (open)-mild
		Skin (rabbit): 500 mg/24h-moderate
		Skin: adverse effect observed (irritating)[1]
Heavy Aromatic Naptha	**TOXICITY**	**IRRITATION**
	Inhalation(Rat) LC50; >3670 ppm/8 h *[2]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; >5000 mg/kg *[2]	Skin: adverse effect observed (irritating)[1]
trimethylbenzenes	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50; 8970 mg/kg[2]	Eye (rabbit): 500 mg/24h - mild
		Skin (rabbit): 500 mg/24h-moderate
Xylene (mixed isomers)	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >1700 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
	Inhalation(Rat) LC50; 5000 ppm4h[2]	Skin: adverse effect observed (irritating)[1]
	Oral (Mouse) LD50; 2119 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]	
1,2,4-trimethyl benzene	**TOXICITY**	**IRRITATION**
	Inhalation(Rat) LC50; 18000 mg/m3/4h[2]	Not Available
cumene	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 2000 mg/kg[2]	Eye (rabbit): 500 mg/24h mild
	Inhalation(Rat) LC50; 39 mg/L4h[2]	Eye (rabbit): 86 mg mild
	Oral (Rat) LD50; 1400 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin (rabbit): 10 mg/24h mild
		Skin (rabbit):100 mg/24h moderate
		Skin: no adverse effect observed (not irritating)[1]
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]	
Mesitylene	**TOXICITY**	**IRRITATION**
	Inhalation (Human) TCLo: 10 ppm[2]	Eye (rabbit): 500 mg/24h mild
	Inhalation(Rat) LC50; 24000 mg/m3/4h[2]	Eye: adverse effect observed (irritating)[1]
		Skin (rabbit): 20 mg/24h moderate
		Skin: adverse effect observed (irritating)[1]
Legend:	*1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*	

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distillates, petroleum, light, hydrotreated	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 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.
2-ethylhexyl nitrate	Chemical with the aliphatic nitro group (-C-NO2) have been added to a list of DNA-reactive subgroups recognised by the National Toxicological Program (NTP, U.S. Dept Health and Human Services) for possible carcinogenic activity.
2-ethylhexanol	For alkyl alcohols C6-13: This group of products are very similar in terms of physicochemical and toxicological properties. Interpolation of data can be used to assess the alkyl alcohols for which data is not available. **Acute toxicity:** All of these alcohols have a low order of toxicity in rats via the oral route. The LD50 for C6-branched and linear alcohols were >3700 mg/kg; LD50s for the C6-8, C7-9, C8-10, C9-11 and C11-14 branched alkyl alcohols were all >2000 mg/kg. These alcohols have a low order of toxicity via the dermal route. Dermal LD50s were greater tha 2600 mg/kg. **Subchronic toxicity**: Repeat dose studies indicate these alcohols have a low order of subchronic toxicity by both the oral and dermal route.

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	Further they demonstrate that these alcohols display a consistent degree of subchronic toxicity by these routes **Developmental toxicity:** Studies demonstrate that the alcohols are not selective developmental toxicants by either the oral or inhalation route of exposure. Inhalation of alkyl alcohols C6-13 is a primary concern during industrial use, particularly for lower molecular weight alcohols. Collectively the weight of evidence demonstrates that these alcohols have a low order of maternal toxicity and do not induce signs of developmental toxicity until maternal toxicity is observed The NOAELs for inhalation reflect the maximum achievable vapour concentration. **Reproductive toxicity:** Developmental toxicity studies for several of these alcohols, conducted by the oral route, produce consistent results and demonstrate that these substances do not affect reproductive parameters.Although a slight increase in resorptions was observed in several studies, this occurred only in the highest dose group and in the presence of overt maternal toxicity. **Genotoxicity:** The weight of evidence from existing data supports the conclusion that these materials are not genotoxic. Further data to support this assessment comes from a series of alkyl acetates C6-13. Alkly acetates arre produced from alkyl alcohols and undergo metabolism by esterases to produce acetic acid and the corresponding alkyl alcohol. There is no evidence for genotoxicity with these compounds in a variety of strains of S. typhimurium in the presence or absence of metabolic activation. C6, C6-8, C7-9 and C11-14 alkyl acetates produced negative results in the Ames test. Based on data for structurally similar substances these alcohols are not expected to be clastogenic. Alkyl acetates can also be used to predict clastogenic potential of a lkyl alcohols. Although there is evidence of cytotoxicity at extremely high doses, no clastogenic activity was seen in a homologous family of alkyl acetates. **Metabolism:**:Alkyl alcohols are broken down, in the body, by mitochondrial beta-oxidation or by cytochrome P450 omega and and omega-minus oxidation. The alcohol undergoes various oxidative steps to yield other alcohols, ketones, aldehydes, carboxylic acids and carbon dioxide, Data for monohydric, aliphatic alcohols show a systematic variation according to molecular weight in a manner similar to other homologous series. The body handles aliphatioc hydrocarbons in a similar manner via oxidative conversion to alcohols, ketones, and eventual elimination as carbon dioxide and carboxylic acids. The undegraded alcohols can be conjugated either directly or as a metabolite with glucuronic acid, sulfuric acid or glycine and are reapidly excreted. Intermediate aldehydes may be reactive and bind with DNA and/ or proteins. The Branched Chain Saturated Alcohol (BCSA) group of fragrance ingredients was evaluated for safety. The 15 materials tested have a low order of acute toxicity. Following repeated application, seven BCSA tested were of low systemic toxicity In humans, no evidence of skin irritation was found at concentrations of 2-10%. Undiluted, 11 materials evaluated caused moderate to severe eye irritation. As current end product use levels are between 0.001% and 1.7%, eye irritation is not a concern. The materials have no or low sensitizing potential. For individuals who are already sensitized, an elicitation reaction is possible. Due to lack of UVA/UVB light-absorbing structures, and review of phototoxic/photoallergy data, the BCSA are not expected to elicit phototoxicity or photoallergy. Studies performed on eight BCSA and three metabolites show no in vivo or in vitro genotoxicity. A valid carcinogenicity study showed that 2-ethyl-1-hexanol is a weak inducer of liver tumours in female mice, however, the relevance of this effect and mode of action to humans is still a matter of debate. Current opinion holds that there are no safety concerns regarding BCSA under the present levels of use and exposure. The common characteristic structural elements of the alcohols with saturated branched chain are one hydroxyl group per molecule, a C4-C12 carbon chain with one or several methyl side chains. Two members of the group, 2-ethyl-1-butanol and 2-ethyl-1-hexanol, contain an ethyl side chain. One member contains a methoxy group. Metabolism studies are lacking for this compound, however, a methoxy group is enzymatically not readily cleaved and if it were so, another primary alcohol group would be formed. The Research Institute for Fragrance Materials (RIFM) Expert Panel
trimethylbenzenes	NOTE: This data is for mixed isomers of unstated proportions.
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.
1,2,4-trimethyl benzene	CHEMWATCH 2325 1,3,5-trimethylbenzene
CUMENE	Cumene is reasonably anticipated to be a human carcinogen based on sufficient evidence of carcinogenicity from studies in experimental animals. Cumene caused tumours at several tissue sites, including lung and liver in mice and kidney in male rats. Several proposed mechanisms of carcinogenesis support the relevance to humans of lung and liver tumours in experimental animals. Specifically, there is evidence that humans and experimental animals metabolise cumene through similar metabolic pathways. There is also evidence that cumene is genotoxic in some tissues, based on findings of DNA damage in rodent lung and liver. Furthermore, mutations of the K-ras oncogene and p53 tumor-suppressor gene observed in cumene-induced lung tumours in mice, along with altered expression of many other genes, resemble molecular alterations found in human lung and other cancers. The relevance of the kidney tumors to cancer in humans is uncertain; there is evidence that a species-specific mechanism not relevant to humans contributes to their induction, but it is possible that other mechanisms relevant to humans, such as genotoxicity, may also contribute to kidney-tumour formation in male rats. For aromatic terpenes: **Acute toxicity:** Mammalian LD50 for p-cymene have shown it to have low toxic potential. Similar studies with cumene have concurred with these results In general, the studies indicate that p-cymene (p-methylisopropylbenzene) or cumene (isopropylbenzene) is rapidly absorbed by oral or inhalation routes. They undergo oxidation (hydroxylation) of the side chain isopropyl substituent and, in the case of p-cymene, the methyl substituent to yield polar oxygenated metabolites. These metabolites are either excreted unchanged in the urine or undergo Phase II conjugation with glucuronic acid and/or glycine followed by excretion in the urine. Unchanged p-cymene or cumene were not detected in the urine or faeces. Humans (5 males and 5 females/group) exposed to an atmosphere containing 49, 98, or 147 ppm cumene for 7 hours showed 64% absorption at 0.5 hours and 45% at 7 hours. Maximum excretion is observed at 6 to 8 hours and is essentially complete at 48 hours. Approximately 35% of the

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	dose inhaled was excreted as 2-phenyl-2-propanol **Repeat Dose Toxicity:** Subacute Studies: Groups of 7 to 12 male rats were exposed to 0, 50, or 250 ppm of p-cymene for 6 hours/day, 5 days/week for 4 weeks with an 8-week recovery period. there was no overt toxicity in the treated rats and no effect on body weight or terminal weight of the brain, cerebellum or whole brain. There was also no effect on regional enzyme activities, regional protein synthesis or regional neurotransmitter concentrations. Cumene has been tested by the National Toxicology Program (NTP) in both rats and mice. Animals were exposed to up to 4,000 ppm cumene by whole-body inhalation for 12-13 days over a period of 16-17 days. In rats, all animals died at 4,000 ppm, and about half the animals died at the next exposure concentration (2,000 ppm). Varying degrees of ataxia were reported in surviving rats exposed to 500 to 2,000 ppm cumene. Increased relative liver and kidney weights were reported in rats exposed to cumene. In exposed male rats, hyaline droplets in the renal cortical tubules were reported. At 2,000 ppm, superlative inflammation of the lung was reported in 40% of the rats. In mice, all animals died at the 2 highest exposures (2,000 and 4,000 ppm). At 1,000 ppm, 80% of the female mice died and male mice showed varying degrees of ataxia. Increased relative liver and kidney weights were reported in mice exposed to cumene. Decreased thymus weight was reported in male mice exposed to 1,000 ppm of cumene. No histopathological findings accompanied the organ weight changes. A NOAEL of 1,000 ppm was determined for female rats and male mice and a NOAEL of 500 ppm was determined for female mice based on mortality and histopathological findings. **Chronic toxicity:** The US EPA concluded that there is some evidence that suggests that cumene is not likely to produce a carcinogenic response (i.e., numerous genotoxic tests, including gene mutation, chromosomal aberration, and primary DNA damage tests, all but one of which were negative or not reproducible) In addition, EPA noted that cumene does not appear to metabolise to highly reactive chemical species and in terms of metabolism, cumene is analogous to methyl benzene for which a 2-year inhalation study was conducted by NTP and no evidence of carcinogenic activity was reported in either rats or mice. Given that the only structural difference between p-cymene and cumene is the presence of a second alkyl substituent (isopropylbenzene versus p-methylisopropylbenzene), similar conclusions can be drawn for p-cymene, particularly since the pharmacokinetic, metabolic and toxicologic data that are available support this conclusion. **Reproductive toxicity:** Taking into consideration the rapid metabolism and excretion of cumene, the US EPA concluded, “cumene has low potential for reproductive toxicity.” **Developmental toxicity:** Even at maternally toxic concentrations exposure to cumene vapor did not produce developmental toxicity in rats. However the US EPA determined that the changes in gestational parameters of the rabbits, though not significant, were consistent in indicating possible developmental effects and therefore set the NOAEL in rabbits for both developmental and maternal effects at 1,206 ppm and the LOAEL at 2,297 ppm, respectively (as reported in EPA, 1997). Since both cumene and p-cymene exhibit such similar pharmacokinetic and metabolic profiles, and show no evidence of toxicity at levels of exposure similar to those experienced by humans, further teratogenic or developmental testing is not recommended **Genotoxicity:** The genotoxicity database on p-cymene and cumene shows no mutagenic potential in the Ames assay. In cytogenetic assays, there is no evidence of a genotoxic potential in vitro. In whole animals, the genotoxicity results for cumene are mixed showing weakly positive results in micronuclei induction in rats, but no evidence of genotoxicity in mice. Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [*National Toxicology Program: U.S. Dep. of Health & Human Services 2002*]
Mesitylene	CHEMWATCH 12171 1,2,4-trimethylbenzene
distillates, petroleum, light, hydrotreated & solvent naphtha petroleum, heavy aromatic & naphtha petroleum, light aromatic solvent & Heavy Aromatic Naptha	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.
solvent naphtha petroleum, heavy aromatic & naphtha petroleum, light aromatic solvent & Heavy Aromatic Naptha	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.
naphtha petroleum, light aromatic solvent & Heavy Aromatic Naptha	Inhalation (rat) TCLo: 1320 ppm/6h/90D-I * [Devoe] For Low Boiling Point Naphthas (LBPNs): **Acute toxicity:** LBPNs generally have low acute toxicity by the oral (median lethal dose [LD50] in rats > 2000 mg/kg-bw), inhalation (LD50 in rats > 5000 mg/m3) and dermal (LD50 in rabbits > 2000 mg/kg-bw) routes of exposure Most LBPNs are mild to moderate eye and skin irritants in rabbits, with the exception of heavy catalytic cracked and heavy catalytic reformed naphthas, which have higher primary skin irritation indices. **Sensitisation:** LBPNs do not appear to be skin sensitizers, but a poor response in the positive control was also noted in these studies **Repeat dose toxicity:** The lowest-observed-adverse-effect concentration (LOAEC) and lowest-observed-adverse-effect level (LOAEL) values identified following short-term (2-89 days) and subchronic (greater than 90 days) exposure to the LBPN substances. These values were determined for a variety of endpoints after considering the toxicity data for all LBPNs in the group. Most of the studies were carried out by the inhalation route of exposure. Renal effects, including increased kidney weight, renal lesions (renal tubule dilation, necrosis) and hyaline droplet formation, observed in male rats exposed orally or by inhalation to most LBPNs, were considered species- and sex-specific These effects were determined to be due to a mechanism of action not relevant to humans -specifically, the interaction between hydrocarbon metabolites and alpha-2-microglobulin, an enzyme not produced in substantial amounts in female rats, mice and other species, including humans. The resulting nephrotoxicity and subsequent carcinogenesis in male rats were therefore not considered in deriving LOAEC/LOAEL values. Only a limited number of studies of short-term and subchronic duration were identified for site-restricted LBPNs. The lowest LOAEC identified in these studies, via the inhalation route, is 5475 mg/m3, based on a concentration-related increase in liver weight in both male and female rats following a 13-week exposure to light catalytic cracked naphtha. Shorter exposures of rats to this test substance resulted in nasal irritation at 9041 mg/m3 No systemic toxicity was reported following dermal exposure to light catalytic cracked naphtha, but skin irritation and accompanying histopathological changes were increased, in a dose-dependent manner, at doses as low as 30 mg/kg-bw per day when applied 5 days per week for 90 days in rats No non-cancer chronic toxicity studies (= 1 year) were identified for site-restricted LBPNs and very few non-cancer chronic toxicity studies were

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identified for other LBPNs. An LOAEC of 200 mg/m3 was noted in a chronic inhalation study that exposed mice and rats to unleaded gasoline (containing 2% benzene). This inhalation LOAEC was based on ocular discharge and ocular irritation in rats. At the higher concentration of 6170 mg/m3, increased kidney weight was observed in male and female rats (increased kidney weight was also observed in males only at 870 mg/m3). Furthermore, decreased body weight in male and female mice was also observed at 6170 mg/m3
A LOAEL of 714 mg/kg-bw was identified for dermal exposure based on local skin effects (inflammatory and degenerative skin changes) in mice following application of naphtha for 105 weeks. No systemic toxicity was reported.

Genotoxicity:

Although few genotoxicity studies were identified for the site-restricted LBPNs, the genotoxicity of several other LBPN substances has been evaluated using a variety of in vivo and in vitro assays. While in vivo genotoxicity assays were negative overall, the in vitro tests exhibited mixed results.
For in vivo genotoxicity tests, LBPNs exhibited negative results for chromosomal aberrations and micronuclei induction, but exhibited positive results in one sister chromatid exchange assay although this result was not considered definitive for clastogenic activity as no genetic material was unbalanced or lost. Mixtures that were tested, which included a number of light naphthas, displayed mixed results (i.e., both positive and negative for the same assay) for chromosomal aberrations and negative results for the dominant lethal mutation assay. Unleaded gasoline (containing 2% benzene) was tested for its ability to induce unscheduled deoxyribonucleic acid (DNA) synthesis (UDS) and replicative DNA synthesis (RDS) in rodent hepatocytes and kidney cells. UDS and RDS were induced in mouse hepatocytes via oral exposure and RDS was induced in rat kidney cells via oral and inhalation exposure. Unleaded gasoline (benzene content not stated) exhibited negative results for chromosomal aberrations and the dominant lethal mutation assay and mixed results for atypical cell foci in rodent renal and hepatic cells.
For in vitro genotoxicity studies, LBPNs were negative for six out of seven Ames tests, and were also negative for UDS and for forward mutations LBPNs exhibited mixed or equivocal results for the mouse lymphoma and sister chromatid exchange assays, as well as for cell transformation and positive results for one bacterial DNA repair assay. Mixtures that were tested, which included a number of light naphthas, displayed negative results for the Ames and mouse lymphoma assays Gasoline exhibited negative results for the Ames test battery, the sister chromatid exchange assay and for one mutagenicity assay . Mixed results were observed for UDS and the mouse lymphoma assay.
While the majority of in vivo genotoxicity results for LBPN substances are negative, the potential for genotoxicity of LBPNs as a group cannot be discounted based on the mixed in vitro genotoxicity results.

Carcinogenicity:

Although a number of epidemiological studies have reported increases in the incidence of a variety of cancers, the majority of these studies are considered to contain incomplete or inadequate information. Limited data, however, are available for skin cancer and leukemia incidence, as well as mortality among petroleum refinery workers. It was concluded that there is limited evidence supporting the view that working in petroleum refineries entails a carcinogenic risk (Group 2A carcinogen). IARC (1989a) also classified gasoline as a Group 2B carcinogen; it considered the evidence for carcinogenicity in humans from gasoline to be inadequate and noted that published epidemiological studies had several limitations, including a lack of exposure data and the fact that it was not possible to separate the effects of combustion products from those of gasoline itself. Similar conclusions were drawn from other reviews of epidemiological studies for gasoline (US EPA 1987a, 1987b). Thus, the evidence gathered from these epidemiological studies is considered to be inadequate to conclude on the effect
s of human exposure to LBPN substances.
No inhalation studies assessing the carcinogenicity of the site-restricted LBPNs were identified. Only unleaded gasoline has been examined for its carcinogenic potential, in several inhalation studies. In one study, rats and mice were exposed to 0, 200, 870 or 6170 mg/m3 of a 2% benzene formulation of the test substance, via inhalation, for approximately 2 years. A statistically significant increase in hepatocellular adenomas and carcinomas, as well as a non-statistical increase in renal tumours, were observed at the highest dose in female mice. A dose-dependent increase in the incidence of primary renal neoplasms was also detected in male rats, but this was not considered to be relevant to humans, as discussed previously.Carcinogenicity was also assessed for unleaded gasoline, via inhalation, as part of initiation/promotion studies. In these studies, unleaded gasoline did not appear to initiate tumour formation, but did show renal cell and hepatic tumour promotion ability, when rats and mice were exposed, via inhalation, for durations ranging from 13 weeks to approximately 1 year using an initiation/promotion protocol However, further examination of data relevant to the composition of unleaded gasoline demonstrated that this is a highly-regulated substance; it is expected to contain a lower percentage of benzene and has a discrete component profile when compared to other substances in the LBPN group.
Both the European Commission and the International Agency for Research on Cancer (IARC) have classified LBPN substances as carcinogenic. All of these substances were classified by the European Commission (2008) as Category 2 (R45: may cause cancer) (benzene content = 0.1% by weight). IARC has classified gasoline, an LBPN, as a Group 2B carcinogen (possibly carcinogenic to humans) and “occupational exposures in petroleum refining” as Group 2A carcinogens (probably carcinogenic to humans).
Several studies were conducted on experimental animals to investigate the dermal carcinogenicity of LBPNs. The majority of these studies were conducted through exposure of mice to doses ranging from 694-1351 mg/kg-bw, for durations ranging from 1 year to the animals lifetime or until a tumour persisted for 2 weeks. Given the route of exposure, the studies specifically examined the formation of skin tumours. Results for carcinogenicity via dermal exposure are mixed. Both malignant and benign skin tumours were induced with heavy catalytic cracked naphtha, light catalytic cracked naphtha, light
straight-run naphtha and naphtha Significant increases in squamous cell carcinomas were also observed when mice were dermally treated with Stoddard solvent, but the latter was administered as a mixture (90% test substance), and the details of the study were not available. In contrast, insignificant increases in tumour formation or no tumours were observed when light alkylate naphtha, heavy catalytic reformed naphtha, sweetened naphtha, light catalytically cracked naphtha
or unleaded gasoline was dermally applied to mice. Negative results for skin tumours were also observed in male mice dermally exposed to sweetened naphtha using an initiation/promotion protocol.
Reproductive/ Developmental toxicity:
No reproductive or developmental toxicity was observed for the majority of LBPN substances evaluated. Most of these studies were carried out by inhalation exposure in rodents.
NOAEC values for reproductive toxicity following inhalation exposure ranged from 1701 mg/m3 (CAS RN 8052-41-3) to 27 687 mg/m3 (CAS RN 64741-63-5) for the LBPNs group evaluated, and from 7690 mg/m3 to 27 059 mg/m3 for the site-restricted light catalytic cracked and full-range catalytic reformed naphthas. However, a decreased number of pups per litter and higher frequency of post-implantation loss were observed following inhalation exposure of female rats to hydrotreated heavy naphtha (CAS RN 64742-48-9) at a concentration of 4679 mg/m3, 6 hours per day, from gestational days 7-20. For dermal exposures, NOAEL values of 714 mg/kg-bw (CAS RN 8030-30-6) and 1000 mg/kg-bw per day (CAS RN 68513-02-0) were noted . For oral exposures, no adverse effects on reproductive parameters were reported when rats were given site-restricted light catalytic cracked naphtha at 2000 mg/kg on gestational day 13 .
For most LBPNs, no treatment-related developmental effects were observed by the different routes of exposure However, developmental toxicity was observed for a few naphthas. Decreased foetal body weight and an increased incidence of ossification variations were observed when rat dams were exposed to light aromatized solvent naphtha, by gavage, at 1250 mg/kg-bw per day. In addition, pregnant rats exposed by inhalation to hydrotreated heavy naphtha at 4679 mg/m3 delivered pups with higher birth weights. Cognitive and memory impairments were also observed in the offspring.
Low Boiling Point Naphthas [Site-Restricted]
For C9 aromatics (typically trimethylbenzenes - TMBs)
Acute Toxicity
Acute toxicity studies (oral, dermal and inhalation routes of exposure) have been conducted in rats using various solvent products containing predominantly mixed C9 aromatic hydrocarbons (CAS RN 64742-95-6). Inhalation LC50 s range from 6,000 to 10,000 mg/m 3 for C9 aromatic naphtha and 18,000 to 24,000 mg/m3 for 1,2,4 and 1,3,5-TMB, respectively. A rat oral LD50 reported for 1,2,4-TMB is 5 grams/kg bw and a rat dermal LD50 for the C9 aromatic naphtha is >4 ml/kg bw. These data indicate that C9 aromatic solvents show that LD50/LC50 values are greater than limit doses for acute toxicity studies established under OECD test guidelines.
Irritation and Sensitization
Several irritation studies, including skin, eye, and lung/respiratory system, have been conducted on members of the category. The results indicate that C9 aromatic hydrocarbon solvents are mildly to moderately irritating to the skin, minimally irritating to the eye, and have the potential to irritate the respiratory tract and cause depression of respiratory rates in mice. Respiratory irritation is a key endpoint in the current occupational exposure limits established for C9 aromatic hydrocarbon solvents and trimethylbenzenes. No evidence of skin sensitization was identified.

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	Repeated Dose Toxicity Inhalation: The results from a subchronic (3 month) neurotoxicity study and a one-year chronic study (6 hr/day, 5 days/week) indicate that effects from inhalation exposure to C9 Aromatic Hydrocarbon Solvents on systemic toxicity are slight. A battery of neurotoxicity and neurobehavioral endpoints were evaluated in the 3-month inhalation study on C9 aromatic naphtha tested at concentrations of 0, 101, 452, or 1320 ppm (0, 500, 2,220, or 6,500 mg/m3). In this study, other than a transient weight reduction in the high exposure group (not statistically significant at termination of exposures), no effects were reported on neuropathology or neuro/behavioral parameters. The NOAEL for systemic and/or neurotoxicity was 6,500 mg/m3, the highest concentration tested. In an inhalation study of a commercial blend, rats were exposed to C9 aromatic naphtha concentrations of 0, 96, 198, or 373 ppm (0, 470, 970, 1830 mg/m3) for 6 hr/day, 5 days/week, for 12 months. Liver and kidney weights were increased in the high exposure group but no accompanying histopathology was observed in these organs. The NOAEL was considered to be the high exposure level of 373 ppm, or 1830 mg/m3. In two subchronic rat inhalation studies, both of three months duration, rats were exposed to the individual TMB isomers (1,2,4-and 1,3,5-) to nominal concentrations of 0, 25, 100, or 250 ppm (0, 123, 492, or 1230 mg/m3). Respiratory irritation was observed at 492 (100 ppm) and 1230 mg/m3 (250 ppm) and no systemic toxicity was observed in either study. For both pure isomers, the NOELs are 25 ppm or 123 mg/m3 for respiratory irritation and 250 ppm or 1230 mg/m3 for systemic effects. Oral: The C9 aromatic naphtha has not been tested via the oral route of exposure. Individual TMB isomers have been evaluated in a series of repeated-dose oral studies ranging from 14 days to 3 months over a wide range of doses. The effects observed in these studies included increased liver and kidney weights, changes in blood chemistry, increased salivation, and decreased weight gain at higher doses. Organ weight changes appeared to be adaptive as they were not accompanied by histopathological effects. Blood changes appeared sporadic and without pattern. One study reported hyaline droplet nephropathy in male rats at the highest dose (1000 mg/kg bw-day), an effect that is often associated with alpha-2mu-globulin-induced nephropathy and not considered relevant to humans. The doses at which effects were detected were 100 mg/kg-bw day or above (an exception was the pilot 14 day oral study - LOAEL 150 mg/kg bw-day - but the follow up three month study had a LOAEL of 600 mg/kg/bw-day with a NOAEL of 200 mg/kg bw-day). Since effects generally were not severe and could be considered adaptive or spurious, oral exposure does not appear to pose a high toxicity hazard for pure trimethylbenzene isomers. Mutagenicity In vitro genotoxicity testing of a variety of C9 aromatics has been conducted in both bacterial and mammalian cells. In vitro point mutation tests were conducted with Salmonella typhimurium and Escherichia coli bacterial strains, as well as with cultured mammalian cells such as the Chinese hamster cell ovary cells (HGPRT assay) with and without metabolic activation. In addition, several types of in vitro chromosomal aberration tests have been performed (chromosome aberration frequency in Chinese hamster ovary and lung cells, sister chromatid exchange in CHO cells). Results were negative both with and without metabolic activation for all category members. For the supporting chemical 1,2,3-TMB, a single in vitro chromosome aberration test was weakly positive. In in vivo bone marrow cytogenetics test, rats were exposed to C9 aromatic naphtha at concentrations of 0, 153, 471, or 1540 ppm (0, 750, 2,310, or 7,560 mg/m3) 6 hr/day, for 5 days. No evidence of in vivo somatic cell genotoxicity was detected. Based on the cumulative results of these assays, genetic toxicity is unlikely for substances in the C9 Aromatic Hydrocarbon Solvents Category Reproductive and Developmental Toxicity Results from the three-generation reproduction inhalation study in rats indicate limited effects from C9 aromatic naphtha. In each of three generations (F0, F1 and F2), rats were exposed to High Flash Aromatic Naphtha (CAS RN 64742-95-6) via whole body inhalation at target concentrations of 0, 100, 500, or 1500 ppm (actual mean concentrations throughout the full study period were 0, 103, 495, or 1480 ppm, equivalent to 0, 505, 2430, or 7265 mg/m3 , respectively). In each generation, both sexes were exposed for 10 weeks prior to and two weeks during mating for 6 hrs/day, 5 days/wks. Female rats in the F0, F1, and F2 generation were then exposed during gestation days 0-20 and lactation days 5-21 for 6 hrs/day, 7 days/wk. The age at exposure initiation differed among generations; F0 rats were exposed starting at 9 weeks of age, F1 exposure began at 5-7 weeks, and F2 exposure began at postnatal day (PND) 22. In the F0 and F1 parental generations, 30 rats/sex /group were exposed and mated. However, in the F2 generation, 40/sex/group were initially exposed due to concerns for toxicity, and 30/sex /group were randomly selected for mating, except that all survivors were used at 1480 ppm. F3 litters were not exposed directly and were sacrificed on lactation day 21. Systemic Effects on Parental Generations: The F0 males showed statistically and biologically significantly decreased mean body weight by ~15% at 1480 ppm when compared with controls. Seven females died or were sacrificed in extremis at 1480 ppm. The F0 female rats in the 495 ppm exposed group had a 13% decrease in body weight gain when adjusted for initial body weight when compared to controls. The F1 parents at 1480 ppm had statistically significantly decreased mean body weights (by ~13% (females) and 22% (males)), and locomotor activity. F1 parents at 1480 ppm had increased ataxia and mortality (six females). Most F2 parents (70/80) exposed to 1480 ppm died within the first week. The remaining animals survived throughout the rest of the exposure period. At week 4 and continuing through the study, F2 parents at 1480 ppm had statistically significant mean body weights much lower than controls (~33% for males; ~28% for females); body weights at 495 ppm were also reduced significantly (by 13% in males and 15% in females). The male rats in the 495 ppm exposed group had a 12% decrease in body weight gain when adjusted for initial body weight when compared to controls. Based on reduced body weight observed, the overall systemic toxicity LOAEC is 495 ppm (2430 mg/m3). Reproductive Toxicity-Effects on Parental Generations: There were no pathological changes noted in the reproductive organs of any animal of the F0, F1, or F2 generation. No effects were reported on sperm morphology, gestational period, number of implantation sites, or post-implantation loss in any generation. Also, there were no statistically or biologically significant differences in any of the reproductive parameters, including: number of mated females, copulatory index, copulatory interval, number of females delivering a litter, number of females delivering a live litter, or male fertility in the F0 or in the F2 generation. Male fertility was statistically significantly reduced at 1480 ppm in the F1 rats. However, male fertility was not affected in the F0 or in the F2 generations; therefore, the biological significance of this change is unknown and may or may not be attributed to the test substance. No reproductive effects were observed in the F0 or F1 dams exposed to 1480 ppm (7265 mg/m3). Due to excessive mortality at the highest concentration (1480 ppm, only six dams available) in the F2 generation,, a complete evaluation is precluded. However, no clear signs of reproductive toxicity were observed in the F2 generation. Therefore, the reproductive NOAEC is considered 495 ppm (2430 mg/m3), which excludes analysis of the highest concentration due to excessive mortality. Developmental Toxicity - Effects on Pups: Because of significant maternal toxicity (including mortality) in dams in all generations at the highest concentration (1480 ppm), effects in offspring at 1480 ppm are not reported here. No significant effects were observed in the F1 and F2 generation offspring at 103 or 495 ppm. However, in F3 offspring, body weights and body weight gain were reduced by ~ 10-11% compared with controls at 495 ppm for approximately a week (PND 14 through 21). Maternal body weight was also depressed by ~ 12% throughout the gestational period compared with controls. The overall developmental LOAEC from this study is 495 ppm (2430 mg/m3) based on the body weights reductions observed in the F3 offspring. Conclusion: No effects on reproductive parameters were observed at any exposure concentration, although a confident assessment of the group exposed at the highest concentration was not possible. A potential developmental effect (reduction in mean pup weight and weight gain) was observed at a concentration that was also associated with maternal toxicity.
naphtha petroleum, light aromatic solvent & Heavy Aromatic Naptha & trimethylbenzenes & 1,2,4-trimethyl benzene & Mesitylene	For trimethylbenzenes: Absorption of 1,2,4-trimethylbenzene occurs after oral, inhalation, or dermal exposure. Occupationally, inhalation and dermal exposures are the most important routes of absorption although systemic intoxication from dermal absorption is not likely to occur due to the dermal irritation caused by the chemical prompting quick removal. Following oral administration of the chemical to rats, 62.6% of the dose was recovered as urinary metabolites indicating substantial absorption . 1,2,4-Trimethylbenzene is lipophilic and may accumulate in fat and fatty tissues. In the blood stream, approximately 85% of the chemical is bound to red blood cells Metabolism occurs by side-chain oxidation to form alcohols and carboxylic acids which are then conjugated with glucuronic acid, glycine, or sulfates for urinary excretion . After a single oral dose to rats of 1200 mg/kg, urinary metabolites consisted of approximately 43.2% glycine, 6.6% glucuronic, and 12.9% sulfuric acid conjugates . The two principle metabolites excreted by rabbits after oral administration of 438 mg/kg/day for 5 days were 2,4-dimethylbenzoic acid and 3,4-dimethylhippuric acid . The major routes of excretion of 1,2,4-trimethyl- benzene are exhalation of parent compound and elimination of urinary metabolites. Half-times for urinary metabolites were reported as 9.5 hours for glycine, 22.9 hours for glucuronide, and 37.6 hours for sulfuric acid conjugates. **Acute Toxicity** Direct contact with liquid 1,2,4-trimethylbenzene is irritating to the skin and breathing the vapor is irritating to the respiratory tract causing pneumonitis. Breathing high concentrations of the chemical vapor causes headache, fatigue, and drowsiness. In humans liquid 1,2,4-trimethylbenzene is irritating to the skin and inhalation of vapor causes chemical pneumonitis . High concentrations of vapor (5000-9000 ppm) cause headache, fatigue, and drowsiness . The concentration of 5000 ppm is roughly equivalent to a total of 221 mg/kg assuming a 30 minute

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	exposure period (see end note 1). 2. Animals - Mice exposed to 8130-9140 ppm 1,2,4-trimethylbenzene (no duration given) had loss of righting response and loss of reflexes Direct dermal contact with the chemical (no species given) causes vasodilation, erythema, and irritation (U.S. EPA). Seven of 10 rats died after an oral dose of 2.5 mL of a mixture of trimethylbenzenes in olive oil (average dose approximately 4.4 g/kg) . Rats and mice were exposed by inhalation to a coal tar distillate containing about 70% 1,3,5- and 1,2,4-trimethylbenzene; no pathological changes were noted in either species after exposure to 1800-2000 ppm for up to 48 continuous hours, or in rats after 14 exposures of 8 hours each at the same exposure levels . No effects were reported for rats exposed to a mixture of trimethyl- benzenes at 1700 ppm for 10 to 21 days **Neurotoxicity** 1,2,4-Trimethylbenzene depresses the central nervous system. Exposure to solvent mixtures containing the chemical causes headache, fatigue, nervousness, and drowsiness. Occupationally, workers exposed to a solvent containing 50% 1,2,4-trimethylbenzene had nervousness, headaches, drowsiness, and vertigo (U.S. EPA). Headache, fatigue, and drowsiness were reported for workers exposed (no dose given) to paint thinner containing 80% 1,2,4- and 1,3,5-trimethylbenzenes Results of the developmental toxicity study indicate that the C9 fraction caused adverse neurological effects at the highest dose (1500 ppm) tested. **Subchronic/Chronic Toxicity** Long-term exposure to solvents containing 1,2,4-trimethylbenzene may cause nervousness, tension, and bronchitis. Painters that worked for several years with a solvent containing 50% 1,2,4- and 30% 1,3,5-trimethylbenzene showed nervousness, tension and anxiety, asthmatic bronchitis, anemia, and alterations in blood clotting; haematological effects may have been due to trace amounts of benzene Rats given 1,2,4-trimethylbenzene orally at doses of 0.5 or 2.0 g/kg/day, 5 days/week for 4 weeks. All rats exposed to the high dose died and 1 rat in the low dose died (no times given); no other effects were reported. Rats exposed by inhalation to 1700 ppm of a trimethylbenzene isomeric mixture for 4 months had decreased weight gain, lymphopenia and neutrophilia . **Genotoxicity:** Results of mutagenicity testing, indicate that the C9 fraction does not induce gene mutations in prokaryotes (Salmonella tymphimurium/mammalian microsome assay); or in mammalian cells in culture (in Chinese hamster ovary cells with and without activation). The C9 fraction does not does not induce chromosome mutations in Chinese hamster ovary cells with and without activation; does not induce chromosome aberrations in the bone marrow of Sprague-Dawley rats exposed by inhalation (6 hours/day for 5 days); and does not induce sister chromatid exchange in Chinese hamster ovary cells with and without activation. **Developmental/Reproductive Toxicity:** A three-generation reproductive study on the C9 fraction was conducted CD rats (30/sex/group) were exposed by inhalation to the C9 fraction at concentrations of 0, 100, 500, or 1500 ppm (0, 100, 500, or 1500 mg/kg/day) for 6 hours/day, 5 days/week. There was evidence of parental and reproductive toxicity at all dose levels. Indicators of parental toxicity included reduced body weights, increased salivation, hunched posture, aggressive behavior, and death. Indicators of adverse reproductive system effects included reduced litter size and reduced pup body weight. The LOEL was 100 ppm; a no-observed-effect level was not established Developmental toxicity, including possible develop- mental neurotoxicity, was evident in rats in a 3-generation reproductive study No effects on fecundity or fertility occurred in rats treated dermally with up to 0.3 mL/rat/day of a mixture of trimethyl- benzenes, 4-6 hours/day, 5 days/week over one generation
2-ethylhexanol & trimethylbenzenes & 1,2,4-trimethyl benzene & CUMENE & Mesitylene	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-ethylhexanol & ethylbenzene	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
2-ethylhexanol & trimethylbenzenes & CUMENE & Mesitylene	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.
trimethylbenzenes & naphthalene & Mesitylene	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
ethylbenzene & naphthalene	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 & CUMENE & naphthalene	**WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
1,2,4-trimethyl benzene & Mesitylene	Other Toxicity data is available for CHEMWATCH 12172 1,2,3-trimethylbenzene

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 Premium Diesel Fuel Treatment

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

distillates, petroleum, light, hydrotreated

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

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2-ethylhexyl nitrate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	1.57mg/l	2
EC50	48h	Crustacea	>12.6mg/l	2
EC10(ECx)	72h	Algae or other aquatic plants	0.76mg/l	2
LC50	96h	Fish	2mg/l	2

solvent naphtha petroleum, 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

naphtha petroleum, light aromatic solvent

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	96h	Algae or other aquatic plants	64mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	1mg/l	1
EC50	72h	Algae or other aquatic plants	19mg/l	1
EC50	48h	Crustacea	6.14mg/l	1

2-ethylhexanol

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	11.5mg/l	1
EC50	48h	Crustacea	39mg/l	1
EC10(ECx)	72h	Algae or other aquatic plants	3.2mg/l	1
LC50	96h	Fish	>7.5mg/l	2

Heavy Aromatic Naptha

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	96h	Algae or other aquatic plants	64mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	1mg/l	1
EC50	72h	Algae or other aquatic plants	19mg/l	1
EC50	48h	Crustacea	6.14mg/l	1

trimethylbenzenes

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

Xylene (mixed isomers)

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

1,2,4-trimethyl benzene

Endpoint	Test Duration (hr)	Species	Value	Source
BCF	1344h	Fish	31-207	7
EC50(ECx)	96h	Algae or other aquatic plants	2.356mg/l	2
EC50	48h	Crustacea	ca.6.14mg/l	1
LC50	96h	Fish	3.41mg/l	2
EC50	96h	Algae or other aquatic plants	2.356mg/l	2

cumene

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	96h	Crustacea	0.4mg/l	1
EC50	72h	Algae or other aquatic plants	1.29mg/l	2
EC50	48h	Crustacea	4mg/l	1
LC50	96h	Fish	2.7mg/l	2

Continued...

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
Mesitylene	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	BCF	1680h	Fish	23-342	7
	EC50	48h	Crustacea	13mg/L	5
	NOEC(ECx)	384h	Crustacea	0.257mg/l	2
	LC50	96h	Fish	5.216mg/l	2
	EC50	96h	Algae or other aquatic plants	3.084mg/l	2
Legend:	*Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*				

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
2-ethylhexanol	LOW	LOW
Xylene (mixed isomers)	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
ethylbenzene	HIGH (Half-life = 228 days)	LOW (Half-life = 3.57 days)
1,2,4-trimethyl benzene	LOW (Half-life = 56 days)	LOW (Half-life = 0.67 days)
cumene	HIGH	HIGH
naphthalene	HIGH (Half-life = 258 days)	LOW (Half-life = 1.23 days)
Mesitylene	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
distillates, petroleum, light, hydrotreated	LOW (BCF = 159)
solvent naphtha petroleum, heavy aromatic	LOW (BCF = 159)
2-ethylhexanol	LOW (BCF = 27)
Xylene (mixed isomers)	MEDIUM (BCF = 740)
ethylbenzene	LOW (BCF = 79.43)
1,2,4-trimethyl benzene	LOW (BCF = 275)
cumene	LOW (BCF = 35.5)
naphthalene	HIGH (BCF = 18000)
Mesitylene	LOW (BCF = 342)

Mobility in soil

Ingredient	Mobility
2-ethylhexanol	LOW (KOC = 26.01)
ethylbenzene	LOW (KOC = 517.8)
1,2,4-trimethyl benzene	LOW (KOC = 717.6)
cumene	LOW (KOC = 817.2)
naphthalene	LOW (KOC = 1837)
Mesitylene	LOW (KOC = 703)

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.

Continued...

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

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO

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

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

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

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

Not Applicable

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

Product name	Group
distillates, petroleum, light, hydrotreated	Not Available
2-ethylhexyl nitrate	Not Available
solvent naphtha petroleum, heavy aromatic	Not Available
naphtha petroleum, light aromatic solvent	Not Available
2-ethylhexanol	Not Available
Heavy Aromatic Naptha	Not Available
trimethylbenzenes	Not Available
Xylene (mixed isomers)	Not Available
ethylbenzene	Not Available
1,2,4-trimethyl benzene	Not Available
cumene	Not Available
naphthalene	Not Available
Mesitylene	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
distillates, petroleum, light, hydrotreated	Not Available
2-ethylhexyl nitrate	Not Available
solvent naphtha petroleum, heavy aromatic	Not Available
naphtha petroleum, light aromatic solvent	Not Available
2-ethylhexanol	Not Available
Heavy Aromatic Naptha	Not Available
trimethylbenzenes	Not Available
Xylene (mixed isomers)	Not Available
ethylbenzene	Not Available
1,2,4-trimethyl benzene	Not Available
cumene	Not Available
naphthalene	Not Available
Mesitylene	Not Available

Continued...

SECTION 15 Regulatory information

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

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

2-ethylhexyl nitrate is found on the following regulatory lists

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 2A: Probably carcinogenic to humans
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

solvent naphtha petroleum, 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

naphtha petroleum, light aromatic solvent 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 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

2-ethylhexanol is found on the following regulatory lists

US - Massachusetts - Right To Know Listed Chemicals
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
US TSCA Section 4/12 (b) - Sunset Dates/Status

Heavy Aromatic Naptha 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 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

trimethylbenzenes is found on the following regulatory lists

US - Massachusetts - Right To Know Listed Chemicals
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

Xylene (mixed isomers) is found on the following regulatory lists

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

1,2,4-trimethyl benzene is found on the following regulatory lists

US - Massachusetts - Right To Know Listed Chemicals
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 Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

cumene is found on the following regulatory lists

Continued...

Chemical Footprint Project - Chemicals of High Concern List	US EPA Integrated Risk Information System (IRIS)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs	US EPCRA Section 313 Chemical List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans	US National Toxicology Program (NTP) 15th Report Part B. Reasonably Anticipated to be a Human Carcinogen
US - California Hazardous Air Pollutants Identified as Toxic Air Contaminants	US NIOSH Recommended Exposure Limits (RELs)
US - California Proposition 65 - Carcinogens	US OSHA Permissible Exposure Limits (PELs) Table Z-1
US - California Safe Drinking Water and Toxic Enforcement Act of 1986 - Proposition 65 List	US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US - Massachusetts - Right To Know Listed Chemicals	US TSCA Chemical Substance Inventory - Interim List of Active Substances
US Clean Air Act - Hazardous Air Pollutants	US TSCA Section 4/12 (b) - Sunset Dates/Status
US DOE Temporary Emergency Exposure Limits (TEELs)	

naphthalene is found on the following regulatory lists

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

Mesitylene is found on the following regulatory lists

US - Massachusetts - Right To Know Listed Chemicals	US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US DOE Temporary Emergency Exposure Limits (TEELs)	US TSCA Chemical Substance Inventory - Interim List of Active Substances
US EPA Integrated Risk Information System (IRIS)	US TSCA Section 4/12 (b) - Sunset Dates/Status
US NIOSH Recommended Exposure Limits (RELs)	

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	No
Skin Corrosion or Irritation	No
Respiratory or Skin Sensitization	No
Serious eye damage or eye irritation	No
Specific target organ toxicity (single or repeated exposure)	No
Aspiration Hazard	Yes
Germ cell mutagenicity	No
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 (mixed isomers)	100	45.4

Continued...

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

State Regulations

US. California Proposition 65

WARNING: This product can expose you to chemicals including **distillates, petroleum, light, hydrotreated, ethylbenzene, cumene, naphthalene**, which are known to the State of California to cause cancer. 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 (distillates, petroleum, light, hydrotreated; 2-ethylhexyl nitrate; solvent naphtha petroleum, heavy aromatic; naphtha petroleum, light aromatic solvent; 2-ethylhexanol; Heavy Aromatic Naptha; trimethylbenzenes; Xylene (mixed isomers); ethylbenzene; 1,2,4-trimethyl benzene; cumene; naphthalene; Mesitylene)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (2-ethylhexyl nitrate)
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.*

SECTION 16 Other information

Revision Date	09/21/2022
Initial Date	08/04/2022

SDS Version Summary

Version	Date of Update	Sections Updated
2.11	09/21/2022	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
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

Continued...

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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