

DIESEL SYSTEM SUPER CLEAN

Motor Active

Chemwatch: 86-2120

Version No: 2.1

Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Chemwatch Hazard Alert Code: 3

Initial Date: 20/03/2026

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Print Date: 24/03/2026

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

Product Identifier

Product name	DIESEL SYSTEM SUPER CLEAN
Chemical Name	Not Applicable
Synonyms	33398 (375ml), 33390, 33391, 33392, 33393, 33394, 33395, 33396, 33399
Proper shipping name	FLAMMABLE LIQUID, N.O.S. (contains xylene)
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Cleaning agent for Diesel Systems. Use according to manufacturer's directions.
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Details of the manufacturer or importer of the safety data sheet

Registered company name	Motor Active
Address	35 Slough Business Park, Holker Street Silverwater NSW 2128 Australia
Telephone	+61 2 9737 9422/1800 350 622
Fax	Not Available
Website	www.motoractive.com.au
Email	info@motoractive.com.au

Emergency telephone number

Association / Organisation	MotorActive
Emergency telephone number(s)	+61 2 9737 9422 (For General Information Monday to Friday 8:30am to 5:pm)
Other emergency telephone number(s)	13 11 26 (In Case of Emergency contact: Poison Information Hotline)

SECTION 2 Hazards identification

Classification of the substance or mixture

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

Chemwatch Hazard Ratings

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

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

Poisons Schedule	S5
Classification ^[1]	Flammable Liquids Category 3, Aspiration Hazard Category 1, Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 1, Acute Toxicity (Inhalation) Category 4, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Reproductive Toxicity Category 2, Specific Target Organ Toxicity - Repeated Exposure Category 2, Hazardous to the Aquatic Environment Acute Hazard Category 3, Hazardous to the Aquatic Environment Long-Term Hazard Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	
Signal word	Danger

Hazard statement(s)

H226	Flammable liquid and vapour.
H304	May be fatal if swallowed and enters airways.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H318	Causes serious eye damage.
H332	Harmful if inhaled.
H335	May cause respiratory irritation.
H336	May cause drowsiness or dizziness.
H361d	Suspected of damaging the unborn child.
H373	May cause damage to organs through prolonged or repeated exposure.
H402	Harmful to aquatic life.
H411	Toxic to aquatic life with long lasting effects.

Supplementary statement(s)

Not Applicable

CLP classification (additional)

Not Applicable

Precautionary statement(s) Prevention

P202	Do not handle until all safety precautions have been read and understood.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P260	Do not breathe mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P240	Ground and bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use non-sparking tools.
P243	Take action to prevent static discharges.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.
P331	Do NOT induce vomiting.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
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.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Collect spillage.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

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.
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No further product hazard information.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
64742-48-9.	60-<65	alkanes, C11-C13-iso-
104-76-7	10-<15	2-ethylhexanol
1330-20-7	10-<15	xylene

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CAS No	%[weight]	Name
27247-96-7	5-<10	<u>2-ethylhexyl nitrate</u>
70024-69-0	1-<5	<u>(C16-24)alkylbenzenesulfonic acid, calcium salt</u>
128-39-2	<1	<u>2,6-di-tert-butylphenol</u>
108-88-3	<1	<u>toluene</u>
69011-36-5	<1	<u>tridecanol, branched, ethoxylated</u>
121158-58-5	<0.1	<u>dodecylphenol, branched</u>
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▶ Immediately hold eyelids apart and flush the eye continuously with running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin or hair contact occurs: ▶ Immediately flush body and clothes with large amounts of water, using safety shower if available. ▶ Quickly remove all contaminated clothing, including footwear. ▶ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▶ Transport to hospital, or doctor.
Inhalation	▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor, without delay.
Ingestion	▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▶ Seek medical advice. ▶ Avoid giving milk or oils. ▶ Avoid giving alcohol. ▶ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

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.

To treat poisoning by the higher aliphatic alcohols (up to C7):

- ▶ Gastric lavage with copious amounts of water.
- ▶ It may be beneficial to instill 60 ml of mineral oil into the stomach.
- ▶ Oxygen and artificial respiration as needed.
- ▶ Electrolyte balance: it may be useful to start 500 ml. M/6 sodium bicarbonate intravenously but maintain a cautious and conservative attitude toward electrolyte replacement unless shock or severe acidosis threatens.
- ▶ To protect the liver, maintain carbohydrate intake by intravenous infusions of glucose.
- ▶ Haemodialysis if coma is deep and persistent. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products, Ed 5]

BASIC TREATMENT

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

ADVANCED TREATMENT

- ▶ Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- ▶ Positive-pressure ventilation using a bag-valve mask might be of use.
- ▶ Monitor and treat, where necessary, for arrhythmias.
- ▶ Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- ▶ If the patient is hypoglycaemic (decreased or loss of consciousness, tachycardia, pallor, dilated pupils, diaphoresis and/or dextrose strip or glucometer readings below 50 mg), give 50% dextrose.
- ▶ Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- ▶ Drug therapy should be considered for pulmonary oedema.
- ▶ Treat seizures with diazepam.
- ▶ Proparacaine hydrochloride should be used to assist eye irrigation.

EMERGENCY DEPARTMENT

- ▶ Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest

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radiographs and electrocardiograph.

- ▶ Positive end-expiratory pressure (PEEP)-assisted ventilation may be required for acute parenchymal injury or adult respiratory distress syndrome.
- ▶ Acidosis may respond to hyperventilation and bicarbonate therapy.
- ▶ Haemodialysis might be considered in patients with severe intoxication.
- ▶ Consult a toxicologist as necessary. BRONSTEIN, A.C. and CURRANCE, P.L. EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

For C8 alcohols and above.

Symptomatic and supportive therapy is advised in managing patients.

The material may induce methaemoglobinaemia following exposure.

- ▶ Initial attention should be directed at oxygen delivery and assisted ventilation if necessary. Hyperbaric oxygen has not demonstrated substantial benefits.
- ▶ Hypotension should respond to Trendelenburg's position and intravenous fluids; otherwise dopamine may be needed.
- ▶ Symptomatic patients with methaemoglobin levels over 30% should receive methylene blue. (Cyanosis, alone, is not an indication for treatment). The usual dose is 1-2 mg/kg of a 1% solution (10 mg/ml) IV over 50 minutes; repeat, using the same dose, if symptoms of hypoxia fail to subside within 1 hour.
- ▶ Thorough cleansing of the entire contaminated area of the body, including the scalp and nails, is of utmost importance.

BIOLOGICAL EXPOSURE INDEX - BEI

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

Determinant	Index	Sampling Time	Comment
1. Methaemoglobin in blood	1.5% of haemoglobin	During or end of shift	B, NS, SQ

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

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

SQ: Semi-quantitative determinant - Interpretation may be ambiguous; should be used as a screening test or confirmatory test.

For acute or short term repeated exposures to xylene:

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

BIOLOGICAL EXPOSURE INDEX - BEI

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

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

SECTION 5 Firefighting measures

Extinguishing media

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

Do not use water jets.

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
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Advice for firefighters

Fire Fighting	▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ May be violently or explosively reactive. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ If safe, switch off electrical equipment until vapour fire hazard removed. ▶ 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	▶ Liquid and vapour are flammable. ▶ Moderate fire hazard when exposed to heat or flame. ▶ Vapour forms an explosive mixture with air. ▶ Moderate explosion hazard when exposed to heat or flame. ▶ Vapour may travel a considerable distance to source of ignition. ▶ Heating may cause expansion or decomposition leading to violent rupture of containers. ▶ On combustion, may emit toxic fumes of carbon monoxide (CO). Combustion products include: ▶ carbon dioxide (CO₂) ▶ carbon monoxide (CO) - sulfur oxides (SO_x) ▶ other pyrolysis products typical of burning organic material.
HAZCHEM	●3Y

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

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Methods and material for containment and cleaning up

Minor Spills	▶ Remove all ignition sources. ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Control personal contact with the substance, by using protective equipment. ▶ Contain and absorb small quantities with vermiculite or other absorbent material. ▶ Wipe up. ▶ Collect residues in a flammable waste container.
Major Spills	▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ May be violently or explosively reactive. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Consider evacuation (or protect in place). ▶ No smoking, naked lights or ignition sources. ▶ Increase ventilation. ▶ Stop leak if safe to do so. ▶ Water spray or fog may be used to disperse /absorb vapour. ▶ Contain spill with sand, earth or vermiculite. ▶ Use only spark-free shovels and explosion proof equipment. ▶ Collect recoverable product into labelled containers for recycling. ▶ Absorb remaining product with sand, earth or vermiculite. ▶ Collect solid residues and seal in labelled drums for disposal. ▶ Wash area and prevent runoff into drains. ▶ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	The conductivity of this material may make it a static accumulator., A liquid is typically considered nonconductive if its conductivity is below 100 pS/m and is considered semi-conductive if its conductivity is below 10 000 pS/m., Whether a liquid is nonconductive or semi-conductive, the precautions are the same., A number of factors, for example liquid temperature, presence of contaminants, and anti-static additives can greatly influence the conductivity of a liquid. Even with proper grounding and bonding, this material can still accumulate an electrostatic charge. If sufficient charge is allowed to accumulate, electrostatic discharge and ignition of flammable air-vapour mixtures can occur. ▶ Containers, even those that have been emptied, may contain explosive vapours. ▶ Do NOT cut, drill, grind, weld or perform similar operations on or near containers. ▶ DO NOT allow clothing wet with material to stay in contact with skin ▶ Electrostatic discharge may be generated during pumping - this may result in fire. ▶ Ensure electrical continuity by bonding and grounding (earthing) all equipment. ▶ Restrict line velocity during pumping in order to avoid generation of electrostatic discharge (<=1 m/sec until fill pipe submerged to twice its diameter, then <= 7 m/sec). ▶ Avoid splash filling. ▶ Do NOT use compressed air for filling discharging or handling operations. ▶ Wait 2 minutes after tank filling (for tanks such as those on ▶ road tanker vehicles) before opening hatches or manholes. ▶ Wait 30 minutes after tank filling (for large storage tanks) ▶ before opening hatches or manholes. Even with proper ▶ grounding and bonding, this material can still accumulate an ▶ electrostatic charge. If sufficient charge is allowed to ▶ accumulate, electrostatic discharge and ignition of flammable ▶ air-vapour mixtures can occur. Be aware of handling ▶ operations that may give rise to additional hazards that result ▶ from the accumulation of static charges. These include but are ▶ not limited to pumping (especially turbulent flow), mixing, ▶ filtering, splash filling, cleaning and filling of tanks and ▶ containers, sampling, switch loading, gauging, vacuum truck ▶ operations, and mechanical movements. These activities may ▶ lead to static discharge e.g. spark formation. Restrict line ▶ velocity during pumping in order to avoid generation of ▶ electrostatic discharge (= 1 m/s until fill pipe submerged to ▶ twice its diameter, then = 7 m/s). Avoid splash filling. ▶ Do NOT use compressed air for filling, discharging, or handling operations ▶ Avoid skin contact, including inhalation. ▶ Wear protective clothing when risk of overexposure 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 generation of static electricity. ▶ DO NOT use plastic buckets. ▶ Earth all lines and equipment. ▶ Use spark-free tools when handling. ▶ 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.
Other information	▶ Store in original containers in approved flammable liquid storage area. ▶ Store away from incompatible materials in a cool, dry, well-ventilated area. ▶ DO NOT store in pits, depressions, basements or areas where vapours may be trapped. ▶ No smoking, naked lights, heat or ignition sources.

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- ▶ Storage areas should be clearly identified, well illuminated, clear of obstruction and accessible only to trained and authorised personnel - adequate security must be provided so that unauthorised personnel do not have access.
 - ▶ Store according to applicable regulations for flammable materials for storage tanks, containers, piping, buildings, rooms, cabinets, allowable quantities and minimum storage distances.
 - ▶ Use non-sparking ventilation systems, approved explosion proof equipment and intrinsically safe electrical systems.
 - ▶ Have appropriate extinguishing capability in storage area (e.g. portable fire extinguishers - dry chemical, foam or carbon dioxide) and flammable gas detectors.
 - ▶ Keep adsorbents for leaks and spills readily available.
 - ▶ Protect containers against physical damage and check regularly for leaks.
 - ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.
- In addition, for tank storages (where appropriate):
- ▶ Store in grounded, properly designed and approved vessels and away from incompatible materials.
 - ▶ For bulk storages, consider use of floating roof or nitrogen blanketed vessels; where venting to atmosphere is possible, equip storage tank vents with flame arrestors; inspect tank vents during winter conditions for vapour/ ice build-up.
 - ▶ Storage tanks should be above ground and diked to hold entire contents.

Conditions for safe storage, including any incompatibilities

Suitable container	▶ Packing as supplied by manufacturer. ▶ Plastic containers may only be used if approved for flammable liquid. ▶ Check that containers are clearly labelled and free from leaks. ▶ For low viscosity materials (i) : Drums and jerry cans must be of the non-removable head type. (ii) : Where a can is to be used as an inner package, the can must have a screwed enclosure. ▶ For materials with a viscosity of at least 2680 cSt. (23 deg. C) ▶ For manufactured product having a viscosity of at least 250 cSt. (23 deg. C) ▶ Manufactured product that requires stirring before use and having a viscosity of at least 20 cSt (25 deg. C): (i) Removable head packaging; (ii) Cans with friction closures and (iii) low pressure tubes and cartridges may be used. ▶ Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages ▶ In addition, where inner packagings are glass and contain liquids of packing group I there must be sufficient inert absorbent to absorb any spillage, unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
Storage incompatibility	▶ Avoid reaction with oxidising agents ▶ Avoid strong bases.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	alkanes, C11-C13-iso-	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available
Australia Workplace exposure limits for airborne contaminants (WEL list) (From 1 December 2026) - Appendix A - Workplace Exposure Limits	alkanes, C11-C13-iso-	Mineral spirits (mineral turpentine)	50 ppm / 296 mg/m3	593 mg/m3 / 100 ppm	Not Available	Not Available
Australia Workplace exposure limits for airborne contaminants (WEL list) (From 1 December 2026) - Appendix A - Workplace Exposure Limits	2-ethylhexanol	2-Ethylhexanol	1 ppm / 5.33 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	xylene	Xylene (o-, m-, p-isomers)	80 ppm / 350 mg/m3	655 mg/m3 / 150 ppm	Not Available	Not Available
Australia Workplace exposure limits for airborne contaminants (WEL list) (From 1 December 2026) - Appendix A - Workplace Exposure Limits	xylene	Xylene (o-, m-, p-isomers)	80 ppm / 350 mg/m3	655 mg/m3 / 150 ppm	Not Available	e Workers exposed to this chemical may require specific health monitoring (see regulations 368-378, Schedule 14 to the model WHS Regulations).
Australia Exposure Standards	toluene	Toluene	50 ppm / 191 mg/m3	574 mg/m3 / 150 ppm	Not Available	Not Available
Australia Workplace exposure limits for airborne contaminants (WEL list) (From 1 December 2026) - Appendix A - Workplace Exposure Limits	toluene	Toluene	20 ppm / 75 mg/m3	Not Available	Not Available	e Workers exposed to this chemical may require specific health monitoring (see regulations 368-378, Schedule 14 to the model WHS Regulations).

MATERIAL DATA

Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure. For flammable liquids and flammable gases, local exhaust ventilation or a process enclosure ventilation system may be required. Ventilation equipment should be explosion-resistant. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.
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	Type of Contaminant:	Air Speed:
	solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
	aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
Within each range the appropriate value depends on:		
Lower end of the range		Upper end of the range
1: Room air currents minimal or favourable to capture		1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only.		2: Contaminants of high toxicity
3: Intermittent, low production.		3: High production, heavy use
4: Large hood or large air mass in motion		4: Small hood-local control only
Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used. - Adequate ventilation is typically taken to be that which limits the average concentration to no more than 25% of the LEL within the building, room or enclosure containing the dangerous substance. - Ventilation for plant and machinery is normally considered adequate if it limits the average concentration of any dangerous substance that might potentially be present to no more than 25% of the LEL. However, an increase up to a maximum 50% LEL can be acceptable where additional safeguards are provided to prevent the formation of a hazardous explosive atmosphere. For example, gas detectors linked to emergency shutdown of the process might be used together with maintaining or increasing the exhaust ventilation on solvent evaporating ovens and gas turbine enclosures. - Temporary exhaust ventilation systems may be provided for non-routine higher-risk activities, such as cleaning, repair or maintenance in tanks or other confined spaces or in an emergency after a release. The work procedures for such activities should be carefully considered.. The atmosphere should be continuously monitored to ensure that ventilation is adequate and the area remains safe. Where workers will enter the space, the ventilation should ensure that the concentration of the dangerous substance does not exceed 10% of the LEL (irrespective of the provision of suitable breathing apparatus)		
Individual protection measures, such as personal protective equipment		
Eye and face protection	- Safety glasses with side shields. - Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent] - 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].	
Skin protection	See Hand protection below	
Hands/feet protection	- Wear chemical protective gloves, e.g. PVC. - Wear safety footwear or safety gumboots, e.g. Rubber NOTE: - The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. - Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. 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.	

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	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	▶ Overalls. ▶ PVC Apron. ▶ PVC protective suit may be required if exposure severe. ▶ Eyewash unit. ▶ Ensure there is ready access to a safety shower. ▶ Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity. ▶ For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets). ▶ Non sparking safety or conductive footwear should be considered. Conductive footwear describes a boot or shoe with a sole made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground the foot an shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
"Forsberg Clothing Performance Index".
The effect(s) of the following substance(s) are taken into account in the *computer-generated* selection:
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Material	CPI
PVA	A
VITON	A
BUTYL	C
BUTYL/NEOPRENE	C
CPE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVC	C
PVDC/PE/PVDC	C
SARANEX-23	C
SARANEX-23 2-PLY	C
TEFLON	C
VITON/CHLOROBUTYL	C
VITON/NEOPRENE	C

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

Ansell Glove Selection

Glove — In order of recommendation
AlphaTec® 38-612
AlphaTec® Solvex® 37-185
AlphaTec® 58-008
AlphaTec® Solvex® 37-675
AlphaTec® 15-554
AlphaTec® 58-530B
AlphaTec® 58-530W
AlphaTec® 58-735

Respiratory protection

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	A-AUS P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	-	A-AUS / Class 1 P2	-
up to 100 x ES	-	A-2 P2	A-PAPR-2 P2 ^

^ - 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

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AlphaTec® 79-700

AlphaTec® 58-005

The suggested gloves for use should be confirmed with the glove supplier.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Yellow transparent flammable liquid with a aromatic like odour; does not mix with water.		
Physical state	Liquid	Relative density (Water = 1)	0.821 @20C
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	>200
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)	200-210	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	55	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Flammable.	Oxidising properties	Not Available
Upper Explosive Limit (%)	>7	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	>0.6	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	>0.800	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (1%)	Not Applicable
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available
Heat of Combustion (kJ/g)	Not Available	Ignition Distance (cm)	Not Available
Flame Height (cm)	Not Available	Flame Duration (s)	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	Enclosed Space Ignition Deflagration Density (g/m3)	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

a) Acute Toxicity	There is sufficient evidence to classify this material as acutely toxic.
b) Skin Irritation/Corrosion	There is sufficient evidence to classify this material as skin corrosive or irritating.
c) Serious Eye Damage/Irritation	There is sufficient evidence to classify this material as eye damaging or irritating
d) Respiratory or Skin sensitisation	There is sufficient evidence to classify this material as sensitising to skin or the respiratory system
e) Mutagenicity	Based on available data, the classification criteria are not met.
f) Carcinogenicity	Based on available data, the classification criteria are not met.
g) Reproductivity	There is sufficient evidence to classify this material as toxic to reproductivity
h) STOT - Single Exposure	There is sufficient evidence to classify this material as toxic to specific organs through single exposure
i) STOT - Repeated Exposure	There is sufficient evidence to classify this material as toxic to specific organs through repeated exposure
j) Aspiration Hazard	There is sufficient evidence to classify this material as an aspiration hazard

Inhaled	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.
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Excessive exposure to 2-ethyl hexanol may result in headache, muscle weakness, nausea, giddiness, ataxia and central nervous system depression.

Exposure to aliphatic alcohols with more than 3 carbons may produce central nervous system effects such as headache, dizziness, drowsiness, muscle weakness, delirium, CNS depression, coma, seizure, and neurobehavioural changes. Symptoms are more acute with higher alcohols. Respiratory tract involvement may produce irritation of the mucosa, respiratory insufficiency, respiratory depression secondary to CNS depression, pulmonary oedema, chemical pneumonitis and bronchitis. Cardiovascular involvement may result in arrhythmias and hypotension. Gastrointestinal effects may include nausea and vomiting. Kidney and liver damage may result following massive exposures. The alcohols are potential irritants being, generally, stronger irritants than similar organic structures that lack functional groups (e.g. alkanes) but are much less irritating than the corresponding amines, aldehydes or ketones. Alcohols and glycols (diols) rarely represent serious hazards in the workplace, because their vapour concentrations are usually less than the levels which produce significant irritation which, in turn, produce significant central nervous system effects as well.

Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination.

Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.

Some aliphatic hydrocarbons produce axonal neuropathies. Isoparaflinic hydrocarbons produce injury to the kidneys of male rats. When albino rats were exposed to isoparaflins at 21.4 mg/l for 4 hours, all animals experienced weakness, tremors, salivation, mild to moderate convulsions, chromodacryorrhoea and ataxia within the first 24 hours. Symptoms disappeared after 24 hours.

Several studies have evaluated sensory irritation in laboratory animals or odor or sensory response in humans. When evaluated by a standard procedure to assess upper airway irritation, isoparaflins did not produce sensory irritation in mice exposed to up to 400 ppm isoparaflin in air. Human volunteers were exposed for six hours to 100 ppm isoparaflin. The subjects were given a self-administered questionnaire to evaluate symptoms, which included dryness of the mucous membranes, loss of appetite, nausea, vomiting, diarrhea, fatigue, headache, dizziness, feeling of inebriation, visual disturbances, tremor, muscular weakness, impairment of coordination or paresthesia. No symptoms associated with solvent exposure were observed. With a human expert panel, odour from liquid imaging copier emissions became weakly discernible at approximately 50 ppm.

Numerous long-term exposures have been conducted in animals with only one major finding observed. Renal tubular damage has been found in kidneys of male rats upon repeated exposures to isoparaflins. It does not occur in mice or in female rats. This male rat nephropathy has been observed with a number of hydrocarbons, including wholly vaporized unleaded gasoline. The phenomenon has been attributed to reversible binding of hydrocarbon to alpha2-globulin. Since humans do not synthesize alpha2-globulin or a similar protein, the finding is not considered to be of biological significance to man. No clinically significant renal abnormalities have been found in refinery workers exposed to hydrocarbons.

When evaluated for developmental toxicity in rats, isoparaflins were neither embryotoxic nor teratogenic. Isoparaflins were consistently negative on standard bacterial genotoxicity assays. They were also non-genotoxic in *in vivo* mammalian testing for somatic or germ cell mutations (mouse micronucleus test and rat dominant lethal assay, respectively).

Mullin et al: Jnl Applied Toxicology 10, pp 136-142, 2006

The acute toxicity of inhaled alkylbenzene is best described by central nervous system depression. These compounds may also act as general anaesthetics. Whole body symptoms of poisoning include light-headedness, nervousness, apprehension, a feeling of well-being, confusion, dizziness, drowsiness, ringing in the ears, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness, depression of breathing, and arrest. Heart stoppage may result from cardiovascular collapse. A slow heart rate and low blood pressure may also occur.

Alkylbenzenes are not generally toxic except at high levels of exposure. Their breakdown products have low toxicity and are easily eliminated from the body.

Headache, fatigue, lassitude, irritability and gastrointestinal disturbances (e.g., nausea, anorexia and flatulence) are the most common symptoms of xylene overexposure. Injury to the heart, liver, kidneys and nervous system has also been noted amongst workers. Transient memory loss, renal impairment, temporary confusion and some evidence of disturbance of liver function was reported in three workers overcome by gross exposure to xylene (10000 ppm). One worker died and autopsy revealed pulmonary congestion, oedema and focal alveolar haemorrhage. Volunteers inhaling xylene at 100 ppm for 5 to 6 hours showed changes in manual coordination reaction time and slight ataxia. Tolerance developed during the workweek but was lost over the weekend. Physical exercise may antagonise this effect. Xylene body burden in humans exposed to 100 or 200 ppm xylene in air depends on the amount of body fat with 4% to 8% of total absorbed xylene accumulating in adipose tissue.

Xylene is a central nervous system depressant. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.

Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful.

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 principal concern with exposure to inorganic nitrate is its biological reduction to reactive and toxic nitrite. Nitrate itself is relatively harmless. Where bacteria are present and the environment is anaerobic, nitrate can be reduced to nitrite. The main site for this reaction is mouth and stomach, but nitrite formation in the lower intestine and in the bladder (urinary infection) may also be of some toxicological importance.

Adults have tolerated large doses of nitrate as sodium and ammonium salt (> 100 mg NO₃⁻/kg) in some cases repeated for several days for medical or experimental purposes with only minor effects in some subjects (light methaemoglobinemia, diarrhoea, vomiting). Death and severe effects of nitrate ingestion are generally associated with doses above 10 g NO₃⁻. Doses between 2 and 9 g NO₃⁻ have been reported to cause methaemoglobinemia. These values correspond to 33 to 150 mg NO₃⁻/kg.

The half-life in the body for an oral dose of nitrate to be approximately 5 hours. As blood absorption depends on food matrix and route of exposure, and as larger doses may increase the urinary excretion rate, the biological half-life for both nitrate and nitrite should be expected to be 3 to 8 hours. Nitrate does not accumulate in the body.

The major acute toxic effect of nitrate and nitrite poisoning is methaemoglobinemia.

The lethal oral dose of nitrite for adults has been variously reported to be between 0.7 and 6 g NO₂⁻ (approximately 10 to 100 mg NO₂⁻/kg). Lower doses may apply for children (especially neonates), the elderly and people with certain enzyme deficiencies. The first symptoms of oral nitrite poisoning develop within 15 to 45 minutes.

In humans, inorganic nitrites produce smooth muscle relaxation, methaemoglobinemia and cyanosis. The primary effect of nitrite intoxication in animals is methaemoglobinemia whilst secondary effects include vasodilation, relaxation of smooth muscle and lowering of blood pressure. Other nitrite-induced toxic effects include abdominal pain, diarrhoea, atrophied intestinal villi and apoptotic cell death in the intestinal crypts. Nitrite may also cause sudden fall in blood pressure due to its vasodilating properties. Nitrite has vasodilating properties, probably through transformation into nitric oxide (NO) or a NO-containing molecule acting as a signal factor for smooth muscle relaxation. Fatal poisonings in infants, resulting from ingestion of nitrates in water or spinach, have been reported.

When sodium nitrite was administered in drinking water for 6 weeks (0.06-1%), mice showed a slight degeneration and spotty necrosis of hepatocytes and haemosiderin deposition in the liver, spleen and lymph nodes, indicating haemolysis. At 2%, mice died within 3 weeks. In rats, subject to the same treatment regime, abnormal blood and spleen colours, due to MHG, were seen in 0.5% and 1.0% treatment groups. Hepatic microsomal lipoperoxidation (as measured by malondialdehyde formation) was increased in male rats given 0.2% sodium nitrite in drinking water. Liver lysosomal enzymes (acid phosphatase and cathepsin) and superoxide dismutase activities were also increased. This data suggests that the nitrite stimulates generation of superoxide radicals in the liver causing damage to cellular and subcellular membranes. Decreased plasma vitamin E and greater reduced glutathione-per erythrocyte were also reported in male rats receiving sodium nitrite in drinking water.

The substance and/or its metabolites may bind to haemoglobin inhibiting normal uptake of oxygen. This condition, known as "methaemoglobinemia", is a form of oxygen starvation (anoxia).

Symptoms include cyanosis (a bluish discolouration skin and mucous membranes) and breathing difficulties. Symptoms may not be evident until several hours after exposure.

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	At about 15% concentration of blood methaemoglobin there is observable cyanosis of the lips, nose and earlobes. Symptoms may be absent although euphoria, flushed face and headache are commonly experienced. At 25-40%, cyanosis is marked but little disability occurs other than that produced on physical exertion. At 40-60%, symptoms include weakness, dizziness, lightheadedness, increasingly severe headache, ataxia, rapid shallow respiration, drowsiness, nausea, vomiting, confusion, lethargy and stupor. Above 60% symptoms include dyspnea, respiratory depression, tachycardia or bradycardia, and convulsions. Levels exceeding 70% may be fatal. Many aliphatic hydrocarbons create a burning sensation because they are irritating to the GI mucosa. Vomiting has been reported in up to one third of all hydrocarbon exposures. While most aliphatic hydrocarbons have little GI absorption, aspiration frequently occurs, either initially or in a semi-delayed fashion as the patient coughs or vomits, thereby resulting in pulmonary effects. Once aspirated, the hydrocarbons can create a severe pneumonitis. Rats given isoparaffinic hydrocarbons - isoalkanes- (after 18-24 hours fasting) showed lethargy and/or general weakness, ataxia and diarrhoea. Symptoms disappeared within 24-28 hours. Overexposure to non-ring alcohols causes nervous system symptoms. These include headache, muscle weakness and inco-ordination, giddiness, confusion, delirium and coma. Rats receiving large oral doses of 2-ethyl hexanol showed haematological effects, stomach irritation and effects on the liver, thymus and spleen. Ingestion of anionic surfactants/ hydrotropes may produce diarrhoea, intestinal distension and occasional vomiting. Lethal doses in animals range from 1 to 5 gm/kg.
Skin Contact	The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either ▶ produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or ▶ produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. Dermally, isoparaffins have produced slight to moderate irritation in animals and humans under occluded patch conditions where evaporation cannot freely occur. However, they are not irritating in non-occluded tests, which are a more realistic simulation of human exposure. They have not been found to be sensitisers in guinea pig or human patch testing. However, occasional rare idiosyncratic sensitisation reactions in humans have been reported. Rabbits exposed dermally to 2 ml/kg 2-ethyl hexanol, for 12 days, showed evidence of liver, lung, kidney and heart effects. Most liquid alcohols appear to act as primary skin irritants in humans. Significant percutaneous absorption occurs in rabbits but not apparently in man. Anionic surfactants/ hydrotropes generally produce skin reactions following the removal of natural oils. The skin may appear red and may become sore. Papular dermatitis may also develop. Sensitive individuals may exhibit cracking, scaling and blistering. Open cuts, abraded or irritated skin should not be exposed to this material The liquid may be miscible with fats or oils and may degrease the skin, producing a skin reaction described as non-allergic contact dermatitis. The material is unlikely to produce an irritant dermatitis as described in EC Directives . The material may accentuate any pre-existing dermatitis condition 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	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation. Instillation of isoparaffins into rabbit eyes produces only slight irritation. Direct eye contact with some concentrated anionic surfactants/ hydrotropes produces corneal damage, in some cases severe. Low concentrations may produce immediate discomfort, conjunctival hyperaemia, and oedema of the corneal epithelium. Healing may take several days. Temporary clouding of the cornea may occur.
Chronic	Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances that can cause occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. Harmful: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests. There is sufficient evidence to establish a causal relationship between human exposure to the material and impaired fertility There is sufficient evidence to establish a causal relationship between human exposure to the material and subsequent developmental toxic effects in the off-spring. Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in impaired fertility on the basis of: - clear evidence in animal studies of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects but which is not a secondary non-specific consequence of other toxic effects. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of: - clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects. On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. There is some evidence to provide a presumption that human exposure to the material may result in the development of heritable genetic damage, generally on the basis of: - appropriate animal studies, - other relevant information

Prolonged or repeated skin contact to 2-ethyl hexanol may result in irritation, reddening and sensitisation. Repeated oral and dermal exposures to animals has resulted in adverse liver and kidney effects.

Male and female rats receiving 1.25% 2-ethyl hexanol in the diet for 90 days showed evidence of kidney and liver effects.

The material produces teratogenic effects in laboratory rats when administered orally to pregnant Wistar rats on day 12 of gestation (1 and 2 ml/kg). These rats were sacrificed on day 20; the percent surviving foetuses with malformations in had increased to 2% and 22.2% in the low and high doses groups respectively. Maternal toxicity may have been a factor. Two subsequent studies indicate that birth defects in rats do not occur following inhalation of the vapour at maximum saturation concentration (850 mg/m3), or dermally at 3 ml/kg doses. Preliminary evidence suggests that absorption through human skin occurs more slowly than in rat.

Prolonged or repeated skin contact may cause degreasing with drying, cracking and dermatitis following.

The major concern of possible long-term effects of exposure to nitrate and nitrite is associated with formation of nitroso compounds, many of which are carcinogenic. This formation may take place wherever nitrite and nitrosable compounds are present, but it is favoured by acidic conditions or the presence of some bacteria. The gastrointestinal tract and especially the stomach is regarded as the main formation site, but nitrosation reactions can also take place in an infected urinary bladder.

Nitrite is mutagenic in a number of in vitro assays against microorganisms or cultured mammalian cells. Nitrates show no mutagenic activity in microbial tests under aerobic conditions. Activity has been reported under anaerobic conditions, probably due to reduction of nitrate into nitrite

The mutagenic effects of nitrites were observed in an in vivo and in vitro experiment using Syrian hamsters. In vivo assays have been equivocal, both positive and negative results having been reported

Exposure to sodium nitrite in drinking water resulted in an increased incidence of epithelial hyperplasia in the forestomach of male and female rats and in the glandular stomach of male mice.

There was equivocal evidence of carcinogenic activity of sodium nitrite in female B6C3F1 mice based on the positive trend in the incidences of squamous cell papillomas or carcinomas (combined) of the forestomach. There was no evidence of carcinogenic activity in male and female F344/N rats or B6C3F1 male mice exposed to 750, 1500 or 3000 ppm.

NTP Technical Report Series No. 495, May 2001

Under certain conditions, nitrites can react with secondary amines, either alone or in biological systems, to form carcinogenic nitrosamines.

Sodium nitrite (60 mg/kg) administered in drinking water to pregnant guinea pigs produced maternal anaemia and increased the incidences of abortion and foetal mortality. Administration of 2000-3000 mg/l sodium nitrite in drinking water, to pregnant rats, produced 30-53% foetal mortality. In rat dams given 0.025-0.5% in feed, sodium nitrite caused an increase in foetal and pup mortality and decreases in pre-weanling body weights.

Prolonged or repeated contact with xylenes may cause defatting dermatitis with drying and cracking. Chronic inhalation of xylenes has been associated with central nervous system effects, loss of appetite, nausea, ringing in the ears, irritability, thirst anaemia, mucosal bleeding, enlarged liver and hyperplasia. Exposure may produce kidney and liver damage. In chronic occupational exposure, xylene (usually mixed with other solvents) has produced irreversible damage to the central nervous system and ototoxicity (damages hearing and increases sensitivity to noise), probably due to neurotoxic mechanisms.

Industrial workers exposed to xylene with a maximum level of ethyl benzene of 0.06 mg/l (14 ppm) reported headaches and irritability and tired quickly. Functional nervous system disturbances were found in some workers employed for over 7 years whilst other workers had enlarged livers.

Xylene has been classed as a developmental toxin in some jurisdictions.

Small excess risks of spontaneous abortion and congenital malformation were reported amongst women exposed to xylene in the first trimester of pregnancy. In all cases, however, the women were also been exposed to other substances. Evaluation of workers chronically exposed to xylene has demonstrated lack of genotoxicity. Exposure to xylene has been associated with increased risks of haemopoietic malignancies but, again, simultaneous exposure to other substances (including benzene) complicates the picture. A long-term gavage study to mixed xylenes (containing 17% ethyl benzene) found no evidence of carcinogenic activity in rats and mice of either sex.

Chronic solvent inhalation exposures may result in nervous system impairment and liver and blood changes. [PATTYS]

DIESEL SYSTEM SUPER CLEAN	TOXICITY	IRRITATION
	Dermal (None) LD50: 5569.8 mg/kg ^[2]	Not Available
	Inhalation (None) LC50: 33.12 mg/L(vapours) ^{*[2]}	
	Inhalation (None) LC50: 4.516 mg/L(dusts&mists) ^{*[2]}	
alkanes, C11-C13-iso-	Oral (None) LD50: 7874.6 mg/kg ^[2]	
	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation (Rat) LC50: >5.266 mg/L4h ^[1]	Skin: adverse effect observed (irritating) ^[1]
2-ethylhexanol	Oral (Rat) LD50: >5000 mg/kg ^[2]	
	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 1970 mg/kg ^[2]	Eye (Rodent - rabbit): 20mg - Severe
	Oral (Rat) LD50: 2049 mg/kg ^[2]	Eye (Rodent - rabbit): 20mg/24H - Moderate
		Eye (Rodent - rabbit): 20ug - Moderate
		Eye: adverse effect observed (irritating) ^[1]
		Skin (Rodent - rabbit): 0.5mL - Severe
		Skin (Rodent - rabbit): 415mg - Mild
		Skin (Rodent - rabbit): 500mg/24H - Moderate
xylene		Skin: adverse effect observed (irritating) ^[1]
	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1700 mg/kg ^[2]	Eye (Human): 200ppm
	Inhalation (Rat) LC50: 5000 ppm4h ^[2]	Eye (Rodent - rabbit): 5mg/24H - Severe
	Oral (Mouse) LD50: 2119 mg/kg ^[2]	Eye (Rodent - rabbit): 87mg - Mild
		Eye: adverse effect observed (irritating) ^[1]
		Skin (Rodent - rabbit): 100% - Moderate
		Skin (Rodent - rabbit): 500mg/24H - Moderate
		Skin (Rodent - rat): 60uL/8H - Mild
		Skin: adverse effect observed (irritating) ^[1]

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2-ethylhexyl nitrate	TOXICITY	IRRITATION
	dermal (rat) LD50: >4820 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation (Rat) LC50: >1.15 mg/l4h ^[2]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >2000 mg/kg ^[2]	
(C16-24)alkylbenzenesulfonic acid, calcium salt	TOXICITY	IRRITATION
	Oral (Rat) LD50: >5000 mg/kg ^[1]	Not Available
2,6-di-tert-butylphenol	TOXICITY	IRRITATION
	dermal (rat) LD50: >1000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >5000 mg/kg ^[1]	Skin (Rodent - rat): 0.5mL - Moderate
		Skin: adverse effect observed (irritating) ^[1]
toluene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 12124 mg/kg ^[2]	Eye (Human): 300ppm
	Inhalation (Rat) LC50: >13350 ppm4h ^[2]	Eye (Rodent - rabbit): 0.1mL
	Oral (Rat) LD50: 636 mg/kg ^[2]	Eye (Rodent - rabbit): 0.1mL - Severe
		Eye (Rodent - rabbit): 100mg/30S - Mild
		Eye (Rodent - rabbit): 2mg/24H - Severe
		Eye (Rodent - rabbit): 870ug - Mild
		Eye: adverse effect observed (irritating) ^[1]
		Skin (Mammal - pig): 250uL/24H - Mild
		Skin (Rodent - rabbit): 20mg/24H - Moderate
		Skin (Rodent - rabbit): 435mg - Mild
		Skin (Rodent - rabbit): 500mg - Moderate
		Skin: adverse effect observed (irritating) ^[1]
		Skin: no adverse effect observed (not irritating) ^[1]
tridecanol, branched, ethoxylated	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: 1080 mg/kg ^[2]	Skin: no adverse effect observed (not irritating) ^[1]
dodecylphenol, branched	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[1]	Eye (Rodent - rabbit): 100uL - Moderate
	Oral (Rat) LD50: <5000 mg/kg ^[1]	Eye: adverse effect observed (irritating) ^[1]
		Skin (Rodent - rabbit): 500uL - Severe
		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

ALKANES, C11-C13-ISO-

C9 -C11 cyclic aliphatics were administered via oral gavage to 5 male and 5 female rats at a dose of 5000 mg/kg to assess acute oral toxicity. Animals were observed daily for 15 days post dosing. At a dose of 5000 mg/kg, signs of toxicity were sedation, dyspnea, hunched posture and ruffled fur. All animals had recovered until day 5 of observation and survived to study termination. All animals were free of abnormalities at postmortem examination. All surviving animals displayed increases in body weight over their day 0 values. The acute oral LD50 for C9 -C11 cyclic aliphatics is >5000 mg/kg. Classification as an oral toxicant is not warranted C9 -C11 cyclic aliphatics were administered via individual inhalation chambers for eight hours to eight Sprague-Dawley rats at vapor concentration of 0 (air), 1 g/m3 (170ppm), 2.5 g/m3 (430ppm), 5 g/m3 (860ppm) for three consecutive days. There was no mortality noted in any of the animals. Based on the conditions of this study, the LC50 for acute inhalation exposure to C9 -C11 cyclic aliphatics vapor is greater than the highest obtainable vapor concentration (5 g/m3). Classification as an acute inhalation toxicant is not warranted. Five male and five female rabbits were exposed to P-D 20/26 for 24h via an occluded patch. Dermal evaluations occurred at 24 hours post patch removal and twice daily until the study termination at day 14. Exposure had no effect on viability; all animals survived the exposure. The LD50 of P-D 20/26 was > 2000 mg/kg. Classification as an acute dermal toxicant is not warranted. Skin irritation: Three rabbits were subjected to a 4h dermal (shaved) exposure of 0.5 ml of ECOLANE 90 via a semi-occluded patch. Dermal responses were evaluated at 1, 24, 48, and 72h post-dosing and once a day for a total of 14 days according to the Draize method of scoring. A very slight or well-defined erythema was observed in all animals from day 1 up to day 9 or 10. A slight oedema was noted in two animals on day 1 only. Dryness of the skin was recorded in all animals from day 4 or 5 up to day 1 or 11. Mean scores over 24, 48 and 72 hours for each animal were 1.3, 1.3 and 2.0 for erythema and 0.0, 0.0 and 0.0 for oedema. Classification as a dermal irritant is not warranted. Eye Irritation: C9-C11, cyclic aliphatics was administered to the left eye of three male and three female rabbits to assess for ocular irritation. Ocular examinations occurred at 1h, 24h, 48h, 72h. Ocular damage was assessed and scored according to the Draize eye test. All animals survived the exposure. The mean corneal opacity, iris lesion, conjunctivae redness, and chemosis scores for C9-C11, cyclic aliphatics were 0, 0, 0, and 0 respectively. Classification as an ocular irritant is not warranted. Sensitisation: A Magnusson and Kligman Guinea-Pig Maximization test was conducted on 20 guinea pigs with Shellsol TD. Twenty guinea pigs were treated by intradermal injection (1.0% (w/v) Shellsol TD in vehicle) to induce sensitization and then further sensitized by dermal application of 50.0% (w/v) Shellsol TD. Guinea Pigs were challenged by topical application (25.0% (w/v) Shellsol TD in corn oil). All animals survived to termination of study. There was a very low incidence of clinical in-life observations noted throughout the test period. Following topical challenge with 25.0% (w/v) Shellsol TD, all animals were free of dermal irritation. Classification as a skin sensitizer is not warranted. In humans, MRD-88-296 showed no evidence of being a photocontact allergen and no evidence of being either a primary irritant or a contact allergen. Based on these data and results, MRD-88-296 would not be classified as a dermal irritant or as a dermal sensitizer. Repeat dose toxicity: oral Results of subchronic exposure of tetramethylcyclohexane (TMCH) to rats and dogs failed to show any treatment-related morphological or qualitative changes in the cellular elements of the peripheral blood picture. This result is consistent with a similar lack of effects noted after acute TMCH exposure. The NOAEL for rats was 30000ppm. The NOAEL for

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dogs was 1000ppm. In both instances, these were the highest levels tested. Genetic toxicity: in vitro No Shellsol TD treatments of any of the test strains, either in the absence or in the presence of S-9, resulted in a statistically significant increase in revertant numbers, when the data were analysed at the 1% level using Dunnetts test. This study was therefore considered to have provided no indication of any SHELLSOL TD mutagenic activity. The test to assess the genotoxicity of the test material was negative. This finding does not warrant the classification of this test material as a genotoxin. Genetic toxicity: in vivo MRD-77-43 when administered by vapor inhalation to male rats is not considered mutagenic by the dominant lethal test. This finding does not warrant the classification of MRD-77-43 as a genotoxin. Toxicity to Reproduction: The NOAEL ≥ 3000 mg/kg/day for male rat fertility. Male rats were given 0, 750, 1500 or 3000 mg/kg neat JP-8 daily by gavage for 70 days prior to mating with naive females to assess fertility and sperm parameters. Males were allowed to mate while continuing to receive treatment. Aside from a decrement in male body weight, no clinical signs were observed. There were no statistical differences noted in any reproductive parameter measured. Developmental toxicity: No adverse effects due to exposure to the test substance were seen in either dams or fetuses. No treatment related malformation effects were noted in the fetuses. The developmental NOAEC for rats by inhalation is ≥ 300 ppm. The test substance is also not teratogenic * REACH Dossier

The safety of isoparaffins as used in cosmetic products was reviewed by the Cosmetic Ingredient Review (CIR) Expert Panel.

These ingredients function mostly as solvents and also function as emollients in the 0001% to 90% concentration range. The CIR Expert Panel has reviewed relevant animal and clinical data and concluded that these ingredients are safe in the present practices of use and concentration

The CIR Expert Panel noted that most of the available data related to oral or inhalation exposure to isoparaffins, but the dermal and ocular exposure data that were available, suggested mild ocular irritation, mild-to-severe irritation, no sensitization or photosensitization, and no phototoxicity. No significant toxicity was identified in oral or inhalation exposure studies of the following end points: genotoxicity, reproductive and developmental toxicity, or carcinogenicity. Nephrotoxicity, however, was a concern. The Expert Panel noted the involvement of a2u-globulin in the mechanism for isoparaffin-induced nephrotoxicity/renal tubule cell proliferation in male rats of various strains in oral and inhalation exposure studies. Humans lack this protein and, thus, the Panel agreed that findings associated with the a2u-globulin protein in male rats were not relevant to humans. This view was consistent with the US EPA position that it was not possible for the agency to derive an oral RfD for chronic oral exposure or a reference concentration for chronic inhalation exposure to isooctane because the available studies were limited, in that they were designed to only investigate the endpoints specific to a2u-globulin-associated nephropathy. The EPA also concluded that there was inadequate evidence to assess the carcinogenic potential of isooctane, based on the absence of human epidemiological studies and chronic bioassays on this compound. However, the CIR Expert Panel noted that no significant tumor incidence was found following life-time dermal application of petrolatum (15% in isooctane) to mice and also found no evidence of any concern regarding carcinogenic potential from exposure to isoparaffins as used in cosmetics.

The potential adverse effects of inhaled aerosols depend on the specific chemical species, the concentration and the duration of the exposure and their site of deposition within the respiratory system. In practice, aerosols should have at least 99% of their particle diameters in the 10 to 110 nm range and the mean particle diameter in a typical aerosol spray has been reported as ~ 38 nm. Particles with an aerodynamic diameter of <10 nm are respirable. After reviewing the positive acute and subchronic inhalation toxicity data the Expert Panel determined that isoparaffins can be used safely in hair sprays, because the product particle size is not respirable.

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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 than 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.

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.

Subchronic toxicity: Repeat dose studies indicate these alcohols have a low order of subchronic toxicity by both the oral and dermal route. 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. Alkyl acetates are 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 alkyl 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 aliphatic hydrocarbons in a similar manner via oxidative conversion to alcohols, ketones, and eventual elimination as carbon dioxide and carboxylic acids. The ungraded alcohols can be conjugated either directly or as a metabolite with glucuronic acid, sulfuric acid or glycine and are readily 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

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	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
XYLENE	Reproductive effector in rats The substance is classified by IARC as Group 3: NOT classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.
2-ETHYLHEXYL NITRATE	Chemical with the aliphatic nitro group (-C-NO ₂) 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.
(C16-24)ALKYLBENZENESULFONIC ACID, CALCIUM SALT	for alkaryl sulfonate petroleum additives: Mammalian Toxicology - Acute. Existing data on acute mammalian toxicity indicates a low concern for acute toxicity. Acute oral toxicity: In all but one studies, there were no deaths that could be attributed to treatment with the test material when administered at the limit dose of 2000 or 5000 mg/kg. In some studies, the primary clinical observations were diarrhea and reduced food consumption (without a change in body weight). These effects are consistent with the gastrointestinal irritant properties of detergents in an oil-based vehicle. In other studies, decreased body weight gain or ruffled fur was observed. In one study where deaths occurred, animals were administered dose levels well above the 2000 mg/kg limit dose. Overall, the acute oral LD50 for these substances was greater than the 2000 mg/kg limit dose indicating a relatively low order of toxicity. Acute dermal toxicity: No mortality was observed for any tested substance when administered at the limit dose of 2000 or 5000 mg/kg. The principal clinical observation was erythema and/or edema at the site of dermal application. In some cases, the cutaneous findings included dry, flaky skin, desquamation and hyperkeratosis. Overall, the acute dermal LD50 for these substances was greater than the 2000 mg/kg limit dose indicating a relatively low order of toxicity. Acute inhalation toxicity: One member of the petroleum additive alkaryl sulfonate category (CAS RN: 6878396-0) was tested for acute inhalation toxicity (OECD Guideline 403, Acute Inhalation Toxicity). Rats were exposed whole-body to an aerosol of the substance at a nominal atmospheric concentration of 1.9 mg/L for four hours. This was the maximum attainable concentration due to the low volatility and high viscosity of the test material. No mortality was noted, and all animals fully recovered following depuration. Clinical signs of toxicity during exposure included reduced activity, matted coat, and closed eyes. Clinical signs of toxicity observed post exposure included lacrimation, nasal discharge, salivation rates, matted coat, hunched appearance, soft stools and closed eyes. No treatment-related macroscopic findings were noted. The lack of mortality at a concentration just below the limit dose of 2.0 mg/L indicates a relatively low order of toxicity for this substance. Mammalian Toxicology - Subchronic Toxicity. Existing data from repeated-dose toxicity studies indicates minimal signs of toxicity following repeated oral exposure. Adverse effects at the site of contact were observed following repeated dermal exposure (injury to the skin) and repeated inhalation (injury to the lungs). NOAELs range from 49.5 mg/m³ to 1000 mg/kg/day Mammalian Toxicology - Reproductive and Developmental Toxicity. A one-generation reproductive toxicity test was conducted on one member of the category (CAS # 115733-09-0). Exposure to the alkaryl sulfonate did not significantly impact reproduction or development and these results were bridged to the remainder of the category. Mammalian Toxicology - Mutagenicity. Existing data from bacterial reverse mutation assays and in vitro and in vivo chromosome aberration studies indicate a low concern for mutagenicity. Animal Irritation An acute eye irritation study indicates that calcium dodecylbenzenesulfonate caused irritation. Result: irritating at 0.1 ml An acute skin study indicate that calcium dodecylbenzenesulfonate is irritant to skin 0.5 ml according to OECD GHS guidelines. Respiratory irritation was not observed. There were no treatment-related changes in the haematological or urinalysis values in any of the animals. No signs of irritation of respiratory tract and nasal effects were observed. Linear alkylbenzene sulfonates (LAS) are classified as Irritant (Xi) with the risk phrases R38 (Irritating to skin) and R41 (Risk of serious damage to eyes) according to CESIO (CESIO 2000). LAS are not included in Annex 1 of list of dangerous substances of Council Directive 67/548/EEC. Linear alkylbenzene sulfonic acids (LABS) are strong acids (pKa<2) are classified as corrosive (R34) Branched materials exhibit comparable toxicity to linear species. Acute toxicity: The available data indicate minimal to moderate toxicity, with LD50 values ranging from 500 to 2000 mg/kg body weight (bw). Acute inhalation data also indicate a lack of significant toxicity. Available dermal exposure data also shows a lack of significant toxicity. LAS are readily absorbed by the gastrointestinal tract after oral administration in animals. LAS are not readily absorbed through the skin. The bulk is metabolised in the liver to sulfophenyl carboxyl acids. The metabolites are excreted primarily via the urine and faeces. The main urinary metabolites in rats are sulfophenyl butanoic acid and sulfophenyl pentanoic acid. Accumulation of LAS or its main metabolites has not been established in any organ after repeated oral ingestion. No serious injuries or fatalities in man have been reported following accidental ingestion of LAS-containing detergent. The main clinical signs observed after oral administration to rats of doses near or greater than the LD50 values consisted of reduced voluntary activity, diarrhoea, weakness etc. Death usually occurred within 24 hours of administration. Rats appear to be more sensitive to LAS than mice. LAS and branched alkylbenzene sulfonates may cause irritation of the eyes, skin and mucous membranes. LAS are relatively more irritating to the skin than the corresponding branched alkylbenzene sulfonates. The potential of LAS to irritate the skin depends on the concentration applied. LAS have been classified as irritating to skin at concentrations above 20% according to EU-criteria. Human skin can tolerate contact with solution of up to 1% LAS for 24 hours resulting in only mild irritation. Application of > 5% LAS to the eyes of rabbits produced irritation. Concentration of < 0.1% LAS produced mild to no irritation. Skin sensitization was not seen in 2,294 volunteers exposed to LAS or in 17,887 exposed to formulations of LAS. Repeat dose toxicity: A feeding study indicated that LAS, when administered for 2 years at extremely high levels (0.5%) in the diets to rats, produced no adverse effects on growth, health or feed efficiency. Genotoxicity: The mutagenic potential of LAS was tested using <i>Salmonella typhimurium</i> strains, using Ames test. In these studies, LAS was not mutagenic. The available long-term studies are inadequate for evaluating the carcinogenic potential of LAS in laboratory animals. The studies available (oral administration to rats and mice) do not show any evidence of carcinogenicity. Reproductive toxicity: In general no specific effect of LAS on reproductive processes has been seen, although dosages causing maternal toxicity may also induce some effects on reproduction. No teratogenic effects attributed to LAS exposure have been observed. Environmental and Health Assessment of Substances in Household Detergents and Cosmetic Detergent Products, Environment Project, 615, 2001. Torben Madsen et al: Miljøministeriet (Danish Environmental Protection Agency) For aromatic sulfonic acids Aromatic sulfonic acids are very corrosive as was demonstrated in skin and eye irritation studies, in the acute oral studies, and in the single repeated dose oral study. Health records from industrial manufacturing exposure, including manufacturing plant book of injuries and a physician report, show toluene-4-sulphonic acid (as handled in manufacturing plants; i.e., a 65% aqueous solution with < 5% free sulphuric acid) is an irritant to the eye and skin. Sensitisation: There is a single, key study for sensitization of the aromatic sulphonic acids. None of the tested animals showed positive responses in a, well documented, GLP guinea pig sensitization study with toluene-4-sulphonic acid (CAS No. 104-15-4). The test substance can be considered a non-sensitizer in guinea pigs as none of the test animals showed a positive response to combined intradermal and topical induction followed by topical challenge. Repeat dose toxicity: A GLP guideline study with p-toluenesulphonic acid (CAS No. 104-15-4) reported no adverse effects to male and female rats exposed orally for 28 days. The highest dose was 500 mg/kg bw/day (>490 mg/kg bw/day based on >98% active ingredient). Therefore the NOAEL was set at 500 mg/kg bw/day. Toxicity to reproduction: No fertility studies are reported for the aromatic sulphonic acids. There are however studies for the chemically related hydrotrope substances that looked at reproductive organs and development of offspring. Hydrotropes are the salt form of the sulphonic acids and

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therefore are used as read-across for this endpoint. The 90-day oral rat and oral mouse studies and the 2-year chronic dermal rat and mouse studies with the closely related compound sodium xylene sulfonate (CAS No. 1300-72-7) included examination of sex organs of both sexes. No treatment related effects on reproductive organs were reported at doses roughly equivalent to those in the developmental toxicity study. The NOAEL for both maternal and foetal toxicity was the highest dose tested - 3000 mg/kg bw /day which is equivalent to 936 mg active ingredient per kilogram body weight per day. The conclusion of the study was no indications of developmental toxicity including teratogenesis.

Genetic toxicity:

There is a fully documented, GLP Guideline (OECD 471) Ames Test and a fully documented, GLP Guideline (OECD 473) Chromosome Aberration Test for one of the aromatic sulphonic acids, p-toluenesulphonic acid (CAS No. 104-15-4). Both tests were conducted with and without metabolic activation. The Ames test exposed up to 5000 micrograms/plate and the chromosome aberration test exposed up to 1902 micrograms per liter of the test substance. These studies conclude the substance is neither mutagenic nor cytotoxic.

There is an additional, published report of an Ames Test for another of the aromatic sulphonic acids, benzenesulfonic acid (CAS No. 98-11-3). Exposures up to 10,000 micrograms/plate were done with and without metabolic activation. The conclusion is the same as for the p-toluenesulphonic acid; that is, not mutagenic and not cytotoxic.

There are no in vivo mutagenicity studies for the aromatic sulphonic acids, but there are two in vivo mouse micronucleus studies for the related hydrotropes – sodium cumene sulfonate (CAS 28348-53-0) and calcium xylene sulfonate (CAS 28088-63-3). Both are GLP-compliant Guideline mouse micronucleus studies with full documentation. Both studies conclude the test substances were not mutagenic in these assays.

Disulfonic acids have not been the subject of concern.

Carcinogenicity:

There are no carcinogenicity studies for the aromatic sulphonic acids. Two hydrotrope studies involve 2-year rat and mouse dermal exposures conducted under GLP. Up to 240 mg (rats) and 727 mg (mice) sodium xylenesulfonate/kg body weight in 50% ethanol were dosed 5 days per week for 104 weeks. There were no treatment related incidences of mononuclear cell leukemia, neoplasms, or nonneoplastic lesions of the skin and other organs. The increased incidence of epidermal hyperplasia may have been related to exposure to the test substance. The NOAEL was reported as 240 mg/kg bw/day for rats and 727 mg/kg bw/day for mice.

Elimination:

The US EPA has evaluated the metabolism of analogs in the sodium alkyl naphthalenesulfonate cluster (SANS), a group of sodium salts of naphthalenesulfonic acids. In a US EPA final rule for SANS, it was stated that "the 1- or 2-sulfonic acid sodium salt moieties on the naphthalene ring may provide a handle by which these compounds can be readily conjugated and eliminated."

Animal studies show that calcium sulfonates with a TBN greater than 300 are not skin sensitizers while the results in animals at a TBN (Total Base Number) of 300 exhibit a mixed skin sensitization response. However, human repeat insult patch tests clearly show that high TBN overbased calcium sulfonates (TBN = 300) are not sensitizers and that low TBN calcium sulfonates do not cause sensitization in a substantial number of persons at concentrations of 10% or lower within the definition of sensitization under EU Regulation (EC) No. 1272/2008.

The weight-of-evidence indicates that low TBN sodium and calcium sulfonates (TBN < 300) are skin sensitizers with a specific concentration limit (SCL) of 10% and that high TBN sodium and calcium sulfonates (TBN = 300) are not skin sensitizers. Studies in guinea pigs show that low TBN benzenesulfonic acid, mono-C20-24 (even)-sec-alkyl derivs., para-, sodium salts (EC No. None; CAS No. None; TBN = 3) is a skin sensitizer while benzenesulfonic acid, mono-C20-24 (even)-sec-alkyl derivs., para-, sodium salts (TBN = 448) is not a skin sensitizer. Studies in guinea pigs and human volunteers show that low TBN benzenesulfonic acid, 4-(mono-C15-36 branched alkyl derivs., C24 rich) and benzenesulfonic acid, 4-octadecyl, calcium salts (EC 939-141-9; TBN = 13) are skin sensitizers. Numerous well-conducted, reliable, controlled human (HRIPT) studies with benzene, polypropylene derivs., sulfonated, calcium salts (EC 616-278-7; TBN values ranging from 13 to 85), sulfonic acids, petroleum, calcium salts (EC 263-093-9; TBN = 30 to 100), and benzenesulfonic acid, 4-(mono-C15-36 branched alkyl derivs., C24 rich) and benzenesulfonic acid, 4-octadecyl, calcium salts (EC 939-141-6; TBN = 13) show that low TBN calcium sulfonates do not cause sensitization in a substantial number of subjects at 10% and lower. High TBN calcium sulfonates, sulfonic acids, petroleum, calcium salts (EC 263-093-9; TBN = 375 and 400) do not cause skin sensitization in guinea pigs. Results of guinea pigs studies at TBN = 300 are mixed; two studies of sulfonic acids, petroleum, calcium salts, (EC 263-093-9) report no skin sensitization while one study of sulfonic acids, petroleum, calcium salts (EC 263-093-9) and one study of benzene, polypropylene derivs., sulfonated, calcium salts (EC 616-278-7) report skin sensitization. However, numerous well-conducted, reliable, controlled human (HRIPT) studies with benzene, polypropylene derivs., sulfonated, calcium salts (EC 616-278-7; TBN = 300) and sulfonic acids, petroleum, calcium salts (EC 263-093-9; TBN = 300) also show that high TBN (TBN = 300) do not cause skin sensitization. In accordance with EU CLP Regulation (EC) No. 1272/2008, classification is required for low TBN sodium and calcium sulfonates (TBN < 300) with a specific concentration limit of 10% and classification is not required for high TBN calcium sulfonates (TBN = 300).

2,6-DI-TERT-BUTYLPHENOL

For hindered phenols:

Available data shows that acute toxicity of these substances is low.

Mutagenicity. Data from bacterial reverse mutation assays and *in vitro* and *in vivo* chromosome aberration studies were reviewed. All assays, with and without metabolic activation, were negative. The weight of evidence for mutagenic potential for this category indicates these substances are not mutagenic.

In Vitro Chromosome Aberration Studies. In vitro chromosome aberration studies are available for several members. All except 2,6-di-tert-butyl-p-cresol were negative.

In Vivo Chromosome Aberration Studies. In vivo studies evaluating chromosome damage are available for six of the hindered phenols. All in vivo evaluations were negative.

Repeated Dose Toxicity. Repeated dose toxicity data of approximately three months (90-day, 12- and 13-week) are available for some of the substances in this group. The liver was the target organ in rats for almost all of the substances with subchronic toxicity data in that species. Other target organs included thyroid and kidney and mesenteric lymph nodes. NOAELs in rats ranged from 100 ppm (approximately 5 mg/kg/day) to 10,000 ppm (500 mg/kg/day).

Carcinogenicity: Data is available for 2,6-di-tert-butyl-p-cresol (128-37-0); and 4,4'-thiobis-6-(t-butyl-m-cresol) (96-69-5). Liver adenomas were reported for 2,6-di-tert-butyl-p-cresol (128-37-0) and a NOAEL was established for the study at 25 mg/kg/day. 4,4'-Thiobis-6-(t-butyl-m-cresol) (96-69-5) was not carcinogenic in rats or mice, but the kidney was identified as a target organ in female rats.

for alkylphenolics category:

The alkylphenolics may be divided into three groups.

Group I: ortho-substituted mono-alkylphenols:

Group II: para-substituted mono-alkylphenols

Group III: di- and tri-substituted mixed alkyl phenols

The subdivision of the category alkylphenols into *ortho*, *para* and the di/tri-substituted mixed members is supported by several published investigations. In assessing antimicrobial and antifouling activity of twenty-three alkylphenols, a significant difference was noted between *para* and *ortho*-substituted materials. In particular, biological activity was found to vary parabolically with increasing hydrophobicity of the *para*-substituent while introduction of a bulky substituent at the *ortho*-position resulted in a very significant decrease in antimicrobial, antifouling, and membrane-perturbation potency. Several alkylphenolic analogs of butylated hydroxytoluene (BHT) were examined for hepatotoxicity in mice depleted of hepatic glutathione. The structural requirement of both hepatic and pulmonary toxicity was a phenol ring having benzylic hydrogen atoms at the para position and an ortho-alkyl group(s) that moderately hinders the phenolic hydroxyl group. It is noteworthy that in this model, neither of the Group III members TTBP (2,4,6-tri-tert-butylphenol) nor 2,6-DTBP (2,6-di-tert-butylphenol) showed either hepatic or pulmonary toxicity. Lastly, important differences were observed in gene activation (recombinant yeast cell assay – Lac-Z reporter gene) between *ortho*-substituted and *para*-substituted alkylphenol.

Acute toxicity: The acute (single-dose) toxicity of alkylphenols examined to date shows consistency, with LD50 values ranging from approximately 1000 mg/kg to over 2000 mg/kg. These data demonstrate a very low level of acute systemic toxicity and do not suggest any unique structural specificity, despite the general tendency for the chemicals to be, at least, irritants to skin.

Repeat dose toxicity: The available studies for members drawn from the three groups range from 28-day and 90-day general toxicity studies, through developmental toxicity and reproductive/developmental screening, to multigeneration reproductive studies are available for some category members.

For the overall category of alkylphenols, the dosage at which the relatively mild general toxicity appears tends only to fall below 100 mg/kg/day with extended treatment, with an overall NOAEL for the category of approximately 20 mg/kg/day. No unusual and no apparent structurally unique toxicity is evident.

DIESEL SYSTEM SUPER CLEAN

Repeat dose studies on OTBP (o-tert-butylphenol; Group I) and PTBP (p-tert-butylphenol; Group II) suggest the forestomach to be the main organ affected. OTBP also appears to have a mild (though statistically significant) protective effect against benzo[a]pyrene induced forestomach tumors. Long-term treatment with high dietary dose levels of PTBP caused hyperplastic changes in the forestomach epithelium of rats and hamsters, a likely consequence of the irritancy of the material. The relevance of this for human hazard is doubtful, particularly since there is no analogous structure in humans to the forestomach of rodents.

There was no evidence of an effect on reproductive function at dosages up to 150 mg/kg. One reproductive screening study reported increased 'breeding loss and also reduced pup weight gain and survival in early lactation at 750 mg/kg/day. It is reasonable to assume that these effects were secondary to "severe toxic symptoms" reported in the dams at this dosage. Other than an indication of a very mildly oestrogenic effect of PNP (p-nonylphenol; Group II) at a high dose levels (200-300 mg/kg/day) no effect on development was seen in a multigeneration study.

By means of the classification method of Verhaar * all the alkylphenols would be classified as Type 2 compounds (polar narcotics). Narcosis, a non-specific mode of toxicity is caused by disruption (perturbation) of the cell membrane. The ability to induce narcosis is dependent on the hydrophobicity of the substance with biochemical activation or reaction involved. Such narcotic effects are also referred to as minimum or base-line toxicity. Polar narcotics such as the category phenols are usually characterised by having hydrogen bond donor activity and are thought to act by a similar mechanism to the inert, narcotic compounds but exhibit above base-line toxicity. In fact, a large number of alkylphenols have been evaluated as intravenous anesthetic agents. While the structure-activity relationships were found to be complex, the anesthetic potency and kinetics appeared to be a function of both the lipophilic character and the degree of steric hindrance exerted by ortho substituents. Less steric hindrance resulted in lower potency, while greater crowding led to complete loss of anesthetic activity and greater lipophilicity resulted in slower kinetics. These data support the notion that the alkylphenols behave as polar narcotics. In addition, the anaesthetic activity/potency differences seen with varying structure and placement of substituents strongly supports the division of alkylphenols category into the ortho, para, and di/tri-substituted groups (i.e. Group I, II and III, respectively).

Genotoxicity: It reasonable to consider the mutagenic potential of all the alkylphenols together because only functional group is the phenolic, which is not a structural alert for mutagenicity. The data support this, since the results of genotoxicity testing are uniformly negative for all category substances examined

* Verhaar, H.J.M. van Leeuwen, C.J. and Hermens, J.L.M., Classifying Environmental Pollutants. 1: Structure-Activity Relationships for Prediction of Aquatic Toxicity, Chemosphere (25), pp 471 – 491 (1992).

TOLUENE

For toluene:

Acute Toxicity

Humans exposed to intermediate to high levels of toluene for short periods of time experience adverse central nervous system effects ranging from headaches to intoxication, convulsions, narcosis, and death. Similar effects are observed in short-term animal studies.

Humans - Toluene ingestion or inhalation can result in severe central nervous system depression, and in large doses, can act as a narcotic. The ingestion of about 60 mL resulted in fatal nervous system depression within 30 minutes in one reported case.

Constriction and necrosis of myocardial fibers, markedly swollen liver, congestion and haemorrhage of the lungs and acute tubular necrosis were found on autopsy.

Central nervous system effects (headaches, dizziness, intoxication) and eye irritation occurred following inhalation exposure to 100 ppm toluene 6 hours/day for 4 days.

Exposure to 600 ppm for 8 hours resulted in the same and more serious symptoms including euphoria, dilated pupils, convulsions, and nausea . Exposure to 10,000-30,000 ppm has been reported to cause narcosis and death

Toluene can also strip the skin of lipids causing dermatitis

Animals - The initial effects are instability and incoordination, lachrymation and sniffles (respiratory exposure), followed by narcosis.

Animals die of respiratory failure from severe nervous system depression. Cloudy swelling of the kidneys was reported in rats following inhalation exposure to 1600 ppm, 18-20 hours/day for 3 days

Subchronic/Chronic Effects:

Repeat doses of toluene cause adverse central nervous system effects and can damage the upper respiratory system, the liver, and the kidney. Adverse effects occur as a result from both oral and the inhalation exposures. A reported lowest-observed-effect level in humans for adverse neurobehavioral effects is 88 ppm.

Humans - Chronic occupational exposure and incidences of toluene abuse have resulted in hepatomegaly and liver function changes. It has also resulted in nephrotoxicity and, in one case, was a cardiac sensitiser and fatal cardiotoxin.

Neural and cerebellar dystrophy were reported in several cases of habitual "glue sniffing." An epidemiological study in France on workers chronically exposed to toluene fumes reported leukopenia and neutropenia. Exposure levels were not given in the secondary reference; however, the average urinary excretion of hippuric acid, a metabolite of toluene, was given as 4 g/L compared to a normal level of 0.6 g/L

Animals - The major target organs for the subchronic/chronic toxicity of toluene are the nervous system, liver, and kidney. Depressed immune response has been reported in male mice given doses of 105 mg/kg/day for 28 days. Toluene in corn oil administered to F344 male and female rats by gavage 5 days/week for 13 weeks, induced prostration, hypoactivity, ataxia, piloerection, lachrymation, excess salivation, and body tremors at doses 2500 mg/kg. Liver, kidney, and heart weights were also increased at this dose and histopathologic lesions were seen in the liver, kidneys, brain and urinary bladder. The no-observed-adverse effect level (NOAEL) for the study was 312 mg/kg (223 mg/kg/day) and the lowest-observed-adverse effect level (LOAEL) for the study was 625 mg/kg (446 mg/kg/day) .

Developmental/Reproductive Toxicity

Exposures to high levels of toluene can result in adverse effects in the developing human fetus. Several studies have indicated that high levels of toluene can also adversely effect the developing offspring in laboratory animals.

Humans - Variable growth, microcephaly, CNS dysfunction, attentional deficits, minor craniofacial and limb abnormalities, and developmental delay were seen in three children exposed to toluene in utero as a result of maternal solvent abuse before and during pregnancy

Animals - Sternebral alterations, extra ribs, and missing tails were reported following treatment of rats with 1500 mg/m³ toluene 24 hours/day during days 9-14 of gestation. Two of the dams died during the exposure. Another group of rats received 1000 mg/m³ 8 hours/day during days 1-21 of gestation. No maternal deaths or toxicity occurred, however, minor skeletal retardation was present in the exposed fetuses. CFLP Mice were exposed to 500 or 1500 mg/m³ toluene continuously during days 6-13 of pregnancy. All dams died at the high dose during the first 24 hours of exposure, however none died at 500 mg/m³. Decreased foetal weight was reported, but there were no differences in the incidences of skeletal malformations or anomalies between the treated and control offspring.

Absorption - Studies in humans and animals have demonstrated that toluene is readily absorbed via the lungs and the gastrointestinal tract. Absorption through the skin is estimated at about 1% of that absorbed by the lungs when exposed to toluene vapor.

Dermal absorption is expected to be higher upon exposure to the liquid; however, exposure is limited by the rapid evaporation of toluene . **Distribution** - In studies with mice exposed to radiolabeled toluene by inhalation, high levels of radioactivity were present in body fat, bone marrow, spinal nerves, spinal cord, and brain white matter. Lower levels of radioactivity were present in blood, kidney, and liver.

Accumulation of toluene has generally been found in adipose tissue, other tissues with high fat content, and in highly vascularised tissues . **Metabolism** - The metabolites of inhaled or ingested toluene include benzyl alcohol resulting from the hydroxylation of the methyl group. Further oxidation results in the formation of benzaldehyde and benzoic acid. The latter is conjugated with glycine to yield hippuric acid or reacted with glucuronic acid to form benzoyl glucuronide. o-cresol and p-cresol formed by ring hydroxylation are considered minor metabolites

Excretion - Toluene is primarily (60-70%) excreted through the urine as hippuric acid. The excretion of benzoyl glucuronide accounts for 10-20%, and excretion of unchanged toluene through the lungs also accounts for 10-20%. Excretion of hippuric acid is usually complete within 24 hours after exposure.

TRIDECANOL, BRANCHED, ETHOXYLATED

* [BASF Canada]

Polyethers (such as ethoxylated surfactants and polyethylene glycols) are highly susceptible to being oxidized in the air. They then form complex mixtures of oxidation products.

Animal testing reveals that whole the pure, non-oxidised surfactant is non-sensitizing, many of the oxidation products are sensitizers. The oxidation products also cause irritation.

Human beings have regular contact with alcohol ethoxylates through a variety of industrial and consumer products such as soaps, detergents, and other cleaning products . Exposure to these chemicals can occur through ingestion, inhalation, or contact with the skin or eyes. Studies of acute toxicity show that volumes well above a reasonable intake level would have to occur to produce any toxic response. Moreover, no fatal case of poisoning with alcohol ethoxylates has ever been reported. Multiple studies investigating the acute toxicity of alcohol ethoxylates have shown that the use of these compounds is of low concern in terms of oral and dermal toxicity .

DIESEL SYSTEM SUPER CLEAN

Clinical animal studies indicate these chemicals may produce gastrointestinal irritation such as ulcerations of the stomach, pilo-erection, diarrhea, and lethargy. Similarly, slight to severe irritation of the skin or eye was generated when undiluted alcohol ethoxylates were applied to the skin and eyes of rabbits and rats. The chemical shows no indication of being a genotoxin, carcinogen, or mutagen (HERA 2007). No information was available on levels at which these effects might occur, though toxicity is thought to be substantially lower than that of nonylphenol ethoxylates.

Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.

Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture.

On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autooxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose allergic contact dermatitis (ACD) to these compounds by patch testing

Overall, alcohol alkoxylates (AAs) are not expected to be systemically toxic, although some short chain ethylene glycol ethers, e.g. methyl and ethyl homologues are of concern for a range of adverse health effects. They include skin and eye irritation, liver and kidney damage, bone marrow and central nervous system (CNS) depression, testicular atrophy, developmental toxicity, and immunotoxicity. For higher propyl and butyl homologues, the toxicity involves haemolysis (anaemia) with secondary effects relating to haemosiderin accumulation in the spleen, liver and kidney, and compensatory haematopoiesis in the bone marrow. Systemic toxicity was shown to decrease with increasing alkyl chain lengths and/or alkoxylation degrees (ECETOC, 2005; US EPA, 2010). The chemicals ethylene glycol hexyl ether (with a longer alkyl chain length, CAS No. 112-25-4) and diethylene glycol butyl ether (with a higher ethoxylation degree, CAS No. 112-34-5) have no evidence of systemic effects including haemolysis.

Commercially available AAs are mixtures of homologues of varying carbon chain lengths and it is possible that some of the chemicals with an average alkyl chain length $C \geq 6$ may also contain shorter alkyl chains $C < 6$. It is not practical to quantify the proportion of shorter $C < 6$ chain lengths present in such chemicals, or these shorter chain lengths may not be present at all. The available data suggest a lack of systemic toxicity for the AE chemicals with potential short alkyl chain presence (NICNASa); therefore, the toxicity of the chemicals in this assessment is unlikely to be significantly affected by the presence of shorter chain alkyl groups.

Alcohol ethoxylates are according to CESIO (2000) classified as Irritant or Harmful depending on the number of EO-units:

EO < 5 gives Irritant (Xi) with R38 (Irritating to skin) and R41 (Risk of serious damage to eyes)

EO > 5-15 gives Harmful (Xn) with R22 (Harmful if swallowed) - R38/41

EO > 15-20 gives Harmful (Xn) with R22-41

>20 EO is not classified (CESIO 2000)

Oxo-AE, C13 EO10 and C13 EO15, are Irritating (Xi) with R36/38 (Irritating to eyes and skin).

AE are not included in Annex 1 of the list of dangerous substances of the Council Directive 67/548/EEC

In general, alcohol ethoxylates (AE) are readily absorbed through the skin of guinea pigs and rats and through the gastrointestinal mucosa of rats. AE are quickly eliminated from the body through the urine, faeces, and expired air (CO₂). Orally dosed AE was absorbed rapidly and extensively in rats, and more than 75% of the dose was absorbed. When applied to the skin of humans, the doses were absorbed slowly and incompletely (50% absorbed in 72 hours). Half of the absorbed surfactant was excreted promptly in the urine and smaller amounts of AE appeared in the faeces and expired air (CO₂). The metabolism of C12 AE yields PEG, carboxylic acids, and CO₂ as metabolites. The LD50 values after oral administration to rats range from about 1-15 g/kg body weight indicating a low to moderate acute toxicity.

The ability of nonionic surfactants to cause a swelling of the stratum corneum of guinea pig skin has been studied. The swelling mechanism of the skin involves a combination of ionic binding of the hydrophilic group as well as hydrophobic interactions of the alkyl chain with the substrate. One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been established that there is a connection between the potential of surfactants to denature protein in vitro and their effect on the skin.

Nonionic surfactants do not carry any net charge and, therefore, they can only form hydrophobic bonds with proteins. For this reason, proteins are not deactivated by nonionic surfactants, and proteins with poor solubility are not solubilized by nonionic surfactants. A substantial amount of toxicological data and information in vivo and in vitro demonstrates that there is no evidence for alcohol ethoxylates (AEs) being genotoxic, mutagenic or carcinogenic. No adverse reproductive or developmental effects were observed. The majority of available toxicity studies revealed NOAELs in excess of 100 mg/kg bw/d but the lowest NOAEL for an individual AE was established to be 50 mg/kg bw/day. This value was subsequently considered as a conservative, representative value in the risk assessment of AE. The effects were restricted to changes in organ weights with no histopathological organ changes with the exception of liver hypertrophy (indicative of an adaptive response to metabolism rather than a toxic effect). It is noteworthy that there was practically no difference in the NOAEL in oral studies of 90-day or 2 years of duration in rats. A comparison of the aggregate consumer exposure and the systemic NOAEL (taking into account an oral absorption value of 75%) results in a Margin of Exposure of 5,800. Taking into account the conservatism in the exposure assessment and the assigned systemic NOAEL, this margin of exposure is considered more than adequate to account for the inherent uncertainty and variability of the hazard database and inter and intra-species extrapolations.

AEs are not contact sensitizers. Neat AE are irritating to eyes and skin. The irritation potential of aqueous solutions of AEs depends on concentrations. Local dermal effects due to direct or indirect skin contact in certain use scenarios where the products are diluted are not of concern as AEs are not expected to be irritating to the skin at in-use concentrations. Potential irritation of the respiratory tract is not a concern given the very low levels of airborne AE generated as a consequence of spray cleaner aerosols or laundry powder detergent dust.

In summary, the human health risk assessment has demonstrated that the use of AE in household laundry and cleaning detergents is safe and does not cause concern with regard to consumer use.

For high boiling ethylene glycol ethers (typically triethylene- and tetraethylene glycol ethers):

Skin absorption: Available skin absorption data for triethylene glycol ether (TGBE), triethylene glycol methyl ether (TGME), and triethylene glycol ethylene ether (TGEE) suggest that the rate of absorption in skin of these three glycol ethers is 22 to 34 micrograms/cm²/hr, with the methyl ether having the highest permeation constant and the butyl ether having the lowest. The rates of absorption of TGBE, TGEE and TGME are at least 100-fold less than EGME, EGEE, and EGBE, their ethylene glycol monoalkyl ether counterparts, which have absorption rates that range from 214 to 2890 micrograms/cm²/hr. Therefore, an increase in either the chain length of the alkyl substituent or the number of ethylene glycol moieties appears to lead to a decreased rate of percutaneous absorption. However, since the ratio of the change in values of the ethylene glycol to the diethylene glycol series is larger than that

of the diethylene glycol to triethylene glycol series, the effect of the length of the chain and number of ethylene glycol moieties on absorption diminishes with an increased number of ethylene glycol moieties. Therefore, although tetraethylene glycol methyl ether (TetraME) and tetraethylene glycol butyl ether (TetraBE) are expected to be less permeable to skin than TGME and TGBE, the differences in permeation between these molecules may only be slight.

Metabolism: The main metabolic pathway for metabolism of ethylene glycol monoalkyl ethers (EGME, EGEE, and EGBE) is oxidation via alcohol and aldehyde dehydrogenases (ALD/ADH) that leads to the formation of an alkoxy acids. Alkoxy acids are the only toxicologically significant metabolites of glycol ethers that have been detected *in vivo*. The principal metabolite of TGME is believed to be 2-[2-(2-methoxyethoxy)ethoxy] acetic acid. Although ethylene glycol, a known kidney toxicant, has been identified as an impurity or a minor metabolite of glycol ethers in animal studies it does not appear to contribute to the toxicity of glycol ethers.

The metabolites of category members are not likely to be metabolized to any large extent to toxic molecules such as ethylene glycol or the mono alkoxy acids because metabolic breakdown of the ether linkages also has to occur

Acute toxicity: Category members generally display low acute toxicity by the oral, inhalation and dermal routes of exposure. Signs of toxicity in animals receiving lethal oral doses of TGBE included loss of righting reflex and flaccid muscle tone, coma, and heavy breathing. Animals administered lethal oral doses of TGEE exhibited lethargy, ataxia, blood in the urogenital area and piloerection before death.

Irritation: The data indicate that the glycol ethers may cause mild to moderate skin irritation. TGEE and TGBE are highly irritating to the eyes. Other category members show low eye irritation.

Repeat dose toxicity: Results of these studies suggest that repeated exposure to moderate to high doses of the glycol ethers in this category is required to produce systemic toxicity

Continued...

DIESEL SYSTEM SUPER CLEAN

In a 21-day dermal study, TGME, TGEE, and TGBE were administered to rabbits at 1,000 mg/kg/day. Erythema and oedema were observed. In addition, testicular degeneration (scored as trace in severity) was observed in one rabbit given TGEE and one rabbit given TGME. Testicular effects included spermatid giant cells, focal tubular hypospermatogenesis, and increased cytoplasmic vacuolisation. Due to a high incidence of similar spontaneous changes in normal New Zealand White rabbits, the testicular effects were considered not to be related to treatment. Thus, the NOAELs for TGME, TGEE and TGBE were established at 1000 mg/kg/day. Findings from this report were considered unremarkable.

A 2-week dermal study was conducted in rats administered TGME at doses of 1,000, 2,500, and 4,000 mg/kg/day. In this study, significantly-increased red blood cells at 4,000 mg/kg/day and significantly-increased urea concentrations in the urine at 2,500 mg/kg/day were observed. A few of the rats given 2,500 or 4,000 mg/kg/day had watery caecal contents and/or haemolysed blood in the stomach. These gross pathologic observations were not associated with any histologic abnormalities in these tissues or alterations in haematologic and clinical chemistry parameters. A few males and females treated with either 1,000 or 2,500 mg/kg/day had a few small scabs or crusts at the test site. These alterations were slight in degree and did not adversely affect the rats. In a 13-week drinking water study, TGME was administered to rats at doses of 400, 1,200, and 4,000 mg/kg/day. Statistically-significant changes in relative liver weight were observed at 1,200 mg/kg/day and higher. Histopathological effects included hepatocellular cytoplasmic vacuolisation (minimal to mild in most animals) and hypertrophy (minimal to mild) in males at all doses and hepatocellular hypertrophy (minimal to mild) in high dose females. These effects were statistically significant at 4,000 mg/kg/day. Cholangiofibrosis was observed in 7/15 high-dose males; this effect was observed in a small number of bile ducts and was of mild severity. Significant, small decreases in total test session motor activity were observed in the high-dose animals, but no other neurological effects were observed. The changes in motor activity were secondary to systemic toxicity.

Mutagenicity: Mutagenicity studies have been conducted for several category members. All in vitro and in vivo studies were negative at concentrations up to 5,000 micrograms/plate and 5,000 mg/kg, respectively, indicating that the category members are not genotoxic at the concentrations used in these studies. The uniformly negative outcomes of various mutagenicity studies performed on category members lessen the concern for carcinogenicity.

Reproductive toxicity: Although mating studies with either the category members or surrogates have not been performed, several of the repeated dose toxicity tests with the surrogates have included examination of reproductive organs. A lower molecular weight glycol ether, ethylene glycol methyl ether (EGME), has been shown to be a testicular toxicant. In addition, results of repeated dose toxicity tests with TGME clearly show testicular toxicity at an oral dose of 4,000 mg/kg/day four times greater than the limit dose of 1,000 mg/kg/day recommended for repeat dose studies. It should be noted that TGME is 350 times less potent for testicular effects than EGME. TGBE is not associated with testicular toxicity. TetraME is not likely to be metabolised by any large extent to 2-MAA (the toxic metabolite of EGME), and a mixture containing predominantly methylated glycol ethers in the C5-C11 range does not produce testicular toxicity (even when administered intravenously at 1,000 mg/kg/day).

Developmental toxicity: The bulk of the evidence shows that effects on the foetus are not noted in treatments with 1,000 mg/kg/day during gestation. At 1,250 to 1,650 mg/kg/day TGME (in the rat) and 1,500 mg/kg/day (in the rabbit), the developmental effects observed included skeletal variants and decreased body weight gain.

DODECYLPHENOL, BRANCHED

for tetrapropenyl phenol and its derivatives.

The chemical possesses properties indicating a potential hazard for human health (effects on fertility and developmental toxicity at doses that also cause maternal toxicity). Adequate screening-level data are available to characterize the human health hazard for the purposes of the OECD Cooperative Chemicals Assessment Programme.

SID Initial Assessment Profile (SIAM 22, 18-21 April 2006)

for para-C12-alkylphenols (typically tetrapropenylphenol)

Based on the toxicological findings presented in this review, para-C12-alkylphenols do not appear to meet the EU criteria for classification for acute toxicity by the oral and dermal routes of exposure, skin sensitisation, repeated dose toxicity or mutagenicity. No information is available relating to acute toxicity via inhalation exposure, and carcinogenicity. The following characteristics do suggest that the substance warrants consideration for classification:

Irritation: para-C12-alkylphenols apparently meet the EU criteria for classification as a skin irritant and a severe eye irritant. Classification for corrosivity could be considered.

Reproductive toxicity: • Fertility: The treatment-related effects on fertility, with supporting pathological changes indicating site of action, appear to meet the EU criteria for classification. The observation that the fertility effects only occurred in the presence of general toxicity might need to be taken into account in deciding the most appropriate category. Overall, these findings suggest that category 2 classification for acute toxicity may be most appropriate, although arguments for category 3 might be considered.

Developmental toxicity: para-C12-alkylphenols caused craniofacial (cleft palate, 3 pups from 1 litter) and long bone malformations (bent long bones) in rats, but only at doses that caused some non-specific maternal toxicity (reduced body weight gain). These findings are not considered to be a secondary non-specific consequence of general toxicity and hence classification for developmental toxicity should be considered. A decision on whether category 2 or 3 is most appropriate may need expert consideration.

Classification for the environment

The substance is classified by the producers as 'dangerous to the environment (with the symbol N) with the following risk phrases:

R50/53: Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment

This is based on the following data:

- Aquatic toxicity: 48-hour Daphnia EC50 <1 mg/L;
- log Kow >3 and measured fish bioconcentration factor of 823;
- Not readily biodegradable.

Acute toxicity data for the oral route of exposure are available for the rat only. LD50 values of 2,100 and 2,200 mg/kg were obtained in two separate studies. In one study, no deaths were observed with a single oral dose of 500 mg/kg but one rat at this dose level showed bloody urine that persisted for 48 hours post-dosing. A NOAEL for the effects of a single oral dose cannot be determined. Signs of toxicity observed in the acute oral toxicity studies included ruffled fur, diarrhoea, diuresis, retarded motion and ataxia.

Dermal: Two studies are available, both in rabbits. The findings indicate that deaths occur with doses above 3,160 mg/kg, and an LD50 of 15,000 mg/kg has been reported. Studies in rabbits indicate that tetrapropenylphenol is a severe skin irritant.

Eye: Studies in rabbits indicate that tetrapropenylphenol is an eye irritant capable of causing corneal opacity and iritis.

Respiratory tract: No data are available concerning respiratory tract irritancy. Given the evidence for skin and eye irritancy, it might be expected that inhaled tetrapropenylphenol would irritate the respiratory tract.

Corrosivity: Necrosis and eschar formation have been reported following dermal application of tetrapropenylphenol to rabbits. From the data available it is uncertain whether or not tetrapropenylphenol should be regarded as corrosive or as a severe skin irritant.

Sensitisation: Tetrapropenylphenol was not identified as a cause of skin sensitisation in two studies in guinea pigs

Repeated dose toxicity: Repeated dosing of tetrapropenylphenol to rats in oral studies, both dietary and gavage, produces effects in a number of organs including the reproductive organs in both sexes.

In the 28-day gavage study in rats, no toxicologically significant treatment-related effects were observed at 5 mg/kg/day. At the next higher dose of 20 mg/kg/day, adrenal cortical gland hypertrophy was observed in male rats. At 180 mg/kg/day and above, pathological changes and organ weight changes were observed in a number of organs, including prominent changes in the reproductive organs in both sexes. In the dietary studies, effects on the testes were noted at 250 mg/kg/day (28-day study) and 106 mg/kg/day (90-day study). No treatment-related toxicological effects were seen in the dietary studies at 25 and 28 mg/kg/day respectively.

No treatment-related changes were reported in dogs at doses up to 4,000 ppm in the diet (estimated by the author to be equivalent to 180 mg/kg/day assuming a body weight of 11 kg and a daily food consumption of 0.5 kg). The absence of treatment-related changes in dogs suggests the existence of a species difference.

Genotoxicity: From the in vitro and in vivo studies available it can be concluded that tetrapropenylphenol is not mutagenic.

Carcinogenicity: There are no carcinogenicity studies available for tetrapropenylphenol. Given the lack of evidence for mutagenicity it is likely that if tetrapropenylphenol had the potential to cause cancer it would involve a threshold mechanism. It is uncertain whether or not the effects on the uterus and the endometrial gland cysts in the one-generation fertility study at the top dose of 125 mg/kg/day indicate the possibility of uterine cancer at these sites following prolonged exposure.

Reproductive toxicity: Tetrapropenylphenol has an adverse effect on fertility in rats, causing a marked reduction in fertility at 125 mg/kg/day. At doses of 25 mg/kg/day and above tetrapropenylphenol causes a reduction in mean pup weight and pathological changes in the reproductive organs of both sexes. The reduction in fertility and effects on reproductive organs occurred at doses that also caused other toxic effects, including reduced bodyweight gain and food consumption and changes in the adrenals, kidneys and liver. However, this

	toxicity was not considered to be particularly severe such that the adverse effects on fertility could have been a secondary non-specific consequence of general toxicity. The NOAEL for reproductive effects is 5 mg/kg/day. Developmental toxicity: Tetrapropenylphenol is also a developmental toxicant in rats, causing teratogenic effects as shown by cleft palate and ectodactyly (reduced number of digits) as well as a general increase in the total number of skeletal malformations at 300 mg/kg/day. These manifestations of developmental toxicity occurred in the presence of overt maternal toxicity, indicated by a significant reduction in body weight gain. Environmental risk evaluation report: para-C12-alkylphenols (dodecylphenol and tetrapropenylphenol): Environment Agency UK
2-ETHYLHEXANOL & 2,6-DI-TERT-BUTYLPHENOL & DODECYLPHENOL, BRANCHED	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 & XYLENE	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
2-ETHYLHEXANOL & XYLENE & TOLUENE	The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.
(C16-24)ALKYLBENZENESULFONIC ACID, CALCIUM SALT & 2,6-DI-TERT-BUTYLPHENOL	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.
(C16-24)ALKYLBENZENESULFONIC ACID, CALCIUM SALT & DODECYLPHENOL, BRANCHED	No significant acute toxicological data identified in literature search.

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					
DIESEL SYSTEM SUPER CLEAN	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
alkanes, C11-C13-iso-	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	72h	Algae or other aquatic plants	>1000mg/l	Not Available
	EC50	72h	Algae or other aquatic plants	>1000mg/l	Not Available
	LC50	96h	Fish	>1000mg/l	Not Available
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
xylene	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	4.6mg/l	2
	EC50	48h	Crustacea	1.8mg/l	2
	NOEC(ECx)	73h	Algae or other aquatic plants	0.44mg/l	2
	LC50	96h	Fish	2.6mg/l	2

DIESEL SYSTEM SUPER CLEAN

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	1.18mg/l	2
EC50	48h	Crustacea	0.83mg/l	2

2-ethylhexyl nitrate

	NOEC(ECx)	72h	Algae or other aquatic plants	0.46mg/l	2
	LC50	96h	Fish	2mg/l	2
(C16-24)alkylbenzenesulfonic acid, calcium salt	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
2,6-di-tert-butylphenol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	0.51mg/l	2
	EC50	48h	Crustacea	0.45mg/l	1
	NOEC(ECx)	504h	Crustacea	0.035mg/l	2
	EC50	96h	Algae or other aquatic plants	0.56mg/l	2
	LC50	96h	Fish	>0.1mg/l	2
toluene	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	12.5mg/L	4
	EC50	48h	Crustacea	3.78mg/L	5
	NOEC(ECx)	168h	Crustacea	0.74mg/l	2
	EC50	96h	Algae or other aquatic plants	>376.71mg/L	4
	LC50	96h	Fish	5-35mg/l	4
tridecanol, branched, ethoxylated	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	1-10mg/l	Not Available
	EC50	48h	Crustacea	1-10mg/l	Not Available
	EC50(ECx)	48h	Crustacea	1-10mg/l	Not Available
	LC50	96h	Fish	2.3mg/l	Not Available
dodecylphenol, branched	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	0.15mg/l	2
	EC50	48h	Crustacea	0.037mg/l	2
	NOEC(ECx)	504h	Crustacea	0.004mg/l	2
	LC50	96h	Fish	3.2mg/l	2
Legend:		Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. US EPA, Ecotox database - Aquatic Toxicity Data 4. ECETOC Aquatic Hazard Assessment Data 5. NITE (Japan) - Bioconcentration Data 6. METI (Japan) - Bioconcentration Data 7. Vendor Data			

Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
2-ethylhexanol	LOW	LOW
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
2,6-di-tert-butylphenol	HIGH	HIGH
toluene	LOW (Half-life = 28 days)	LOW (Half-life = 4.33 days)
dodecylphenol, branched	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
2-ethylhexanol	LOW (BCF = 27)
xylene	MEDIUM (BCF = 740)
2,6-di-tert-butylphenol	HIGH (LogKOW = 4.92)
toluene	LOW (BCF = 90)
tridecanol, branched, ethoxylated	LOW (LogKOW = 3.59)
dodecylphenol, branched	MEDIUM (BCF = 850)

Mobility in soil

Ingredient	Mobility
2-ethylhexanol	LOW (Log KOC = 26.01)
2,6-di-tert-butylphenol	LOW (Log KOC = 14220)
toluene	LOW (Log KOC = 268)
dodecylphenol, branched	LOW (Log KOC = 382000)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	- Containers may still present a chemical hazard/ danger when empty. - Return to supplier for reuse/ recycling if possible. Otherwise: - If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. - Where possible retain label warnings and SDS and observe all notices pertaining to the product. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: - Reduction - Reuse - Recycling - Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. DO NOT allow wash water from cleaning or process equipment to enter drains. - It may be necessary to collect all wash water for treatment before disposal. - In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. - Where in doubt contact the responsible authority. - Recycle wherever possible. - Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. - Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material). - Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	
HAZCHEM	•3Y

Land transport (ADG)

14.1. UN number or ID number	1993	
14.2. UN proper shipping name	FLAMMABLE LIQUID, N.O.S. (contains xylene)	
14.3. Transport hazard class(es)	Class	3
	Subsidiary Hazard	Not Applicable
14.4. Packing group	III	
14.5. Environmental hazard	Environmentally hazardous	
14.6. Special precautions for user	Special provisions	223 274
	Limited quantity	5 L

Air transport (ICAO-IATA / DGR)

14.1. UN number	1993	
14.2. UN proper shipping name	Flammable liquid, n.o.s. * (contains xylene)	
14.3. Transport hazard class(es)	ICAO/IATA Class	3
	ICAO / IATA Subsidiary Hazard	Not Applicable
	ERG Code	3L
14.4. Packing group	III	
14.5. Environmental hazard	Environmentally hazardous	
14.6. Special precautions for user	Special provisions	A3
	Cargo Only Packing Instructions	366
	Cargo Only Maximum Qty / Pack	220 L
	Passenger and Cargo Packing Instructions	355
	Passenger and Cargo Maximum Qty / Pack	60 L

	Passenger and Cargo Limited Quantity Packing Instructions	Y344
	Passenger and Cargo Limited Maximum Qty / Pack	10 L

Sea transport (IMDG-Code / GGVSee)

14.1. UN number	1993	
14.2. UN proper shipping name	FLAMMABLE LIQUID, N.O.S. (contains xylene)	
14.3. Transport hazard class(es)	IMDG Class	3
	IMDG Subsidiary Hazard	Not Applicable
14.4. Packing group	III	
14.5 Environmental hazard	Marine Pollutant	
14.6. Special precautions for user	EMS Number	F-E, S-E
	Special provisions	223 274 955
	Limited Quantities	5 L

14.7. Maritime transport in bulk according to IMO instruments

14.7.1. Transport in bulk according to Annex II of MARPOL and the IBC code
Not Applicable

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

Product name	Group
alkanes, C11-C13-iso-	Not Applicable
2-ethylhexanol	Not Applicable
xylene	Not Applicable
2-ethylhexyl nitrate	Not Applicable
(C16-24)alkylbenzenesulfonic acid, calcium salt	Not Applicable
2,6-di-tert-butylphenol	Not Applicable
toluene	Not Applicable
tridecanol, branched, ethoxylated	Not Applicable
dodecylphenol, branched	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
alkanes, C11-C13-iso-	Not Applicable
2-ethylhexanol	Not Applicable
xylene	Not Applicable
2-ethylhexyl nitrate	Not Applicable
(C16-24)alkylbenzenesulfonic acid, calcium salt	Not Applicable
2,6-di-tert-butylphenol	Not Applicable
toluene	Not Applicable
tridecanol, branched, ethoxylated	Not Applicable
dodecylphenol, branched	Not Applicable

SECTION 15 Regulatory information

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

alkanes, C11-C13-iso- is found on the following regulatory lists
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
2-ethylhexanol is found on the following regulatory lists
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
xylene is found on the following regulatory lists
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

2-ethylhexyl nitrate is found on the following regulatory lists

- Australian Inventory of Industrial Chemicals (AIIC)
- 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

(C16-24)alkylbenzenesulfonic acid, calcium salt is found on the following regulatory lists

- Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
- Australia Industrial Chemicals Environmental Management (IChEMS Register) Instrument 2022 - Schedule 3 - Relevant industrial chemicals that have the potential to cause harm to the environment
- Australian Inventory of Industrial Chemicals (AIIC)

2,6-di-tert-butylphenol is found on the following regulatory lists

- Australian Inventory of Industrial Chemicals (AIIC)
- International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

toluene is found on the following regulatory lists

- Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
- Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
- Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
- Australian Inventory of Industrial Chemicals (AIIC)
- Chemical Footprint Project - Chemicals of High Concern List
- International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

tridecanol, branched, ethoxylated is found on the following regulatory lists

- Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
- Australian Inventory of Industrial Chemicals (AIIC)

dodecylphenol, branched is found on the following regulatory lists

- Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
- Australian Inventory of Industrial Chemicals (AIIC)
- Chemical Footprint Project - Chemicals of High Concern List

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (alkanes, C11-C13-iso-; 2-ethylhexanol; xylene; 2-ethylhexyl nitrate; (C16-24)alkylbenzenesulfonic acid, calcium salt; 2,6-di-tert-butylphenol; toluene; tridecanol, branched, ethoxylated)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory 'Active'
Taiwan - TCSI	Yes
Mexico - INSQ	No (2-ethylhexyl nitrate; (C16-24)alkylbenzenesulfonic acid, calcium salt; tridecanol, branched, ethoxylated; dodecylphenol, branched)
Vietnam - NCI	Yes
Russia - FBEPH	No ((C16-24)alkylbenzenesulfonic acid, calcium salt)
UAE - Control List (Banned/Restricted Substances)	No (alkanes, C11-C13-iso-; 2-ethylhexanol; xylene; 2-ethylhexyl nitrate; (C16-24)alkylbenzenesulfonic acid, calcium salt; 2,6-di-tert-butylphenol; toluene; tridecanol, branched, ethoxylated; dodecylphenol, branched)
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	20/03/2026
Initial Date	20/03/2026

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

DIESEL SYSTEM SUPER CLEAN

- 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
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration
- MARPOL: International Convention for the Prevention of Pollution from Ships
- IMSBC: International Maritime Solid Bulk Cargoes Code
- IGC: International Gas Carrier Code
- IBC: International Bulk Chemical Code

- 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
- 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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