



DURAM ROCKTUFF (BLACK, DARK GREY, LIGHT GREY, YELLOW)

Duram Pty Ltd

Chemwatch: 5234-49

Version No: 5.1.8.7

Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Chemwatch Hazard Alert Code: 3

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

Product Identifier

Product name	DURAM ROCKTUFF (BLACK, DARK GREY, LIGHT GREY, YELLOW)
Chemical Name	Not Applicable
Synonyms	Rocktuff. Liquid polyurethane, anti-slip protective coating.
Proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)
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	Anti-slip protective coating for: Domestic: decks, balconies, patios, garages, steps, pool surround. Schools: playgrounds, demountable classrooms, walkways, toilets. Hospitality: kitchen and bar areas. Building Construction: flooring, traffic areas, driveways, bridges.
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Details of the supplier of the safety data sheet

Registered company name	Duram Pty Ltd
Address	51 Prince William Drive Seven Hills NSW 2147 Australia
Telephone	+61 2 9624 4007
Fax	+61 2 9624 4079
Website	www.duram.com.au
Email	mail@duram.com.au

Emergency telephone number

Association / Organisation	CHEMTREC Australia (Sydney)
Emergency telephone numbers	+612 9037 2994 24 hours / 7 days
Other emergency telephone numbers	Not Available

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	2	
Toxicity	2	2	
Body Contact	2	2	
Reactivity	1	1	
Chronic	3	3	

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

Poisons Schedule	Not Applicable
Classification [1]	Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Skin Sensitizer Category 1, Respiratory Sensitizer Category 1, Germ cell mutagenicity Category 2, Carcinogenicity Category 2, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Specific target organ toxicity - single exposure Category 3 (narcotic effects), Aspiration Hazard Category 1, Acute Aquatic Hazard Category 3, Chronic Aquatic Hazard Category 3, Flammable Liquid Category 3
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

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

H315	Causes skin irritation.
H319	Causes serious eye irritation.
H317	May cause an allergic skin reaction.
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H341	Suspected of causing genetic defects.
H351	Suspected of causing cancer.
H335	May cause respiratory irritation.
H336	May cause drowsiness or dizziness.
H304	May be fatal if swallowed and enters airways.
H412	Harmful to aquatic life with long lasting effects.
H226	Flammable liquid and vapour.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P261	Avoid breathing mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P284	[In case of inadequate ventilation] wear respiratory 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.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P331	Do NOT induce vomiting.
P342+P311	If experiencing respiratory symptoms: Call a POISON CENTER/doctor/physician/first aider.
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.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].

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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SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

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Mixtures

CAS No	%[weight]	Name
Not Available	30-60	urethane polymer (non-hazardous)
64742-95-6	10-30	<u>naphtha petroleum, light aromatic solvent</u>
9002-88-4	10-30	<u>polyethylene</u>
95-63-6	1-10	<u>1,2,4-trimethyl benzene</u>
13463-67-7	<10	<u>titanium dioxide</u>
117-81-7	<10	<u>di-sec-octyl phthalate</u>
25686-28-6	<5	<u>MDI homopolymer</u>
101-68-8	<5	<u>4,4'-diphenylmethane diisocyanate (MDI)</u>
6358-31-2	<5	<u>C.I. Pigment Yellow 74</u>
108-67-8	<5	<u>1,3,5-trimethyl benzene</u>
1333-86-4	<5	<u>carbon black</u>
7631-86-9	<5	<u>silica amorphous</u>
98-82-8	<1	<u>cumene</u>
108-83-8	<1	<u>diisobutyl ketone</u>
68037-01-4	<1	<u>1-decene homopolymer, hydrogenated</u>
41556-26-7	<1	<u>bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate</u>
4083-64-1	<1	<u>p-toluenesulfonyl isocyanate</u>
125643-61-0	<1	<u>C7-9 branched alkyl-3,5-di-tert-butyl-4-hydroxyhydrocinnamate</u>
1185-81-5	<1	<u>dibutyltin dilauryl mercaptide</u>
82919-37-7	<1	<u>methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate</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	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ 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	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation. <p>For thermal burns:</p> <ul style="list-style-type: none"> ▶ Decontaminate area around burn. ▶ Consider the use of cold packs and topical antibiotics. <p>For first-degree burns (affecting top layer of skin)</p> <ul style="list-style-type: none"> ▶ Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides. ▶ Use compresses if running water is not available. ▶ Cover with sterile non-adhesive bandage or clean cloth. ▶ Do NOT apply butter or ointments; this may cause infection. ▶ Give over-the counter pain relievers if pain increases or swelling, redness, fever occur. <p>For second-degree burns (affecting top two layers of skin)</p> <ul style="list-style-type: none"> ▶ Cool the burn by immerse in cold running water for 10-15 minutes. ▶ Use compresses if running water is not available. ▶ Do NOT apply ice as this may lower body temperature and cause further damage. ▶ Do NOT break blisters or apply butter or ointments; this may cause infection. ▶ Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape. <p>To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort):</p> <ul style="list-style-type: none"> ▶ Lay the person flat. ▶ Elevate feet about 12 inches. ▶ Elevate burn area above heart level, if possible. ▶ Cover the person with coat or blanket. ▶ Seek medical assistance. <p>For third-degree burns</p> <p>Seek immediate medical or emergency assistance.</p> <p>In the mean time:</p> <ul style="list-style-type: none"> ▶ Protect burn area cover loosely with sterile, nonstick bandage or, for large areas, a sheet or other material that will not leave lint in wound. ▶ Separate burned toes and fingers with dry, sterile dressings. ▶ Do not soak burn in water or apply ointments or butter; this may cause infection. ▶ To prevent shock see above. ▶ For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway. ▶ Have a person with a facial burn sit up. ▶ Check pulse and breathing to monitor for shock until emergency help arrives.

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Inhalation	<ul style="list-style-type: none"> ▶ 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. <p>Following uptake by inhalation, move person to an area free from risk of further exposure. Oxygen or artificial respiration should be administered as needed. Asthmatic-type symptoms may develop and may be immediate or delayed up to several hours. Treatment is essentially symptomatic. A physician should be consulted.</p>
Ingestion	<ul style="list-style-type: none"> ▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▶ Seek medical advice. ▶ Avoid giving milk or oils. ▶ Avoid giving alcohol.

Indication of any immediate medical attention and special treatment needed

For acute or short term repeated exposures to petroleum distillates or related hydrocarbons:

- ▶ Primary threat to life, from pure petroleum distillate 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) 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.
- ▶ Lavage is indicated in patients who require decontamination; ensure use of cuffed endotracheal tube in adult patients. [Ellenhorn and Barceloux: Medical Toxicology]

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.

Treat symptomatically.

For sub-chronic and chronic exposures to isocyanates:

- ▶ This material may be a potent pulmonary sensitiser which causes bronchospasm even in patients without prior airway hyperreactivity.
- ▶ Clinical symptoms of exposure involve mucosal irritation of respiratory and gastrointestinal tracts.
- ▶ Conjunctival irritation, skin inflammation (erythema, pain vesiculation) and gastrointestinal disturbances occur soon after exposure.
- ▶ Pulmonary symptoms include cough, burning, substernal pain and dyspnoea.
- ▶ Some cross-sensitivity occurs between different isocyanates.
- ▶ Noncardiogenic pulmonary oedema and bronchospasm are the most serious consequences of exposure. Markedly symptomatic patients should receive oxygen, ventilatory support and an intravenous line.
- ▶ Treatment for asthma includes inhaled sympathomimetics (epinephrine [adrenalin], terbutaline) and steroids.
- ▶ Activated charcoal (1 g/kg) and a cathartic (sorbitol, magnesium citrate) may be useful for ingestion.
- ▶ Mydriatics, systemic analgesics and topical antibiotics (Sulamyd) may be used for corneal abrasions.
- ▶ There is no effective therapy for sensitised workers.

[Ellenhorn and Barceloux; Medical Toxicology]

NOTE: Isocyanates cause airway restriction in naive individuals with the degree of response dependant on the concentration and duration of exposure. They induce smooth muscle contraction which leads to bronchoconstrictive episodes. Acute changes in lung function, such as decreased FEV₁, may not represent sensitivity.

[Karol & Jin, Frontiers in Molecular Toxicology, pp 56-61, 1992]

Personnel who work with isocyanates, isocyanate prepolymers or polyisocyanates should have a pre-placement medical examination and periodic examinations thereafter, including a pulmonary function test. Anyone with a medical history of chronic respiratory disease, asthmatic or bronchial attacks, indications of allergic responses, recurrent eczema or sensitisation conditions of the skin should not handle or work with isocyanates. Anyone who develops chronic respiratory distress when working with isocyanates should be removed from exposure and examined by a physician. Further exposure must be avoided if a sensitivity to isocyanates or polyisocyanates has developed.

SECTION 5 Firefighting measures

Extinguishing media

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

Special hazards arising from the substrate or mixture

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

Fire Fighting	<ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ 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.

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	<ul style="list-style-type: none"> ▶ 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). <p>Combustion products include: carbon dioxide (CO₂) carbon monoxide (CO) isocyanates and minor amounts of hydrogen cyanide formaldehyde acrolein nitrogen oxides (NO_x) other pyrolysis products typical of burning organic material. May emit clouds of acrid smoke</p>
HAZCHEM	*3Y

SECTION 6 Accidental release measures**Personal precautions, protective equipment and emergency procedures**

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> ▶ 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	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> ▶ 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. <p>For isocyanate spills of less than 40 litres (2 m²):</p> <ul style="list-style-type: none"> ▶ Evacuate area from everybody not dealing with the emergency, keep them upwind and prevent further access, remove ignition sources and, if inside building, ventilate area as well as possible. ▶ Notify supervision and others as necessary. ▶ Put on personal protective equipment (suitable respiratory protection, face and eye protection, protective suit, gloves and impermeable boots). ▶ Control source of leakage (where applicable). ▶ Dike the spill to prevent spreading and to contain additions of decontaminating solution. ▶ Prevent the material from entering drains. ▶ Estimate spill pool volume or area. ▶ Absorb and decontaminate. - Completely cover the spill with wet sand, wet earth, vermiculite or other similar absorbent. - Add neutraliser (for suitable formulations: see below) to the adsorbent materials (equal to that of estimated spill pool volume). Intensify contact between spill, absorbent and neutraliser by carefully mixing with a rake and allow to react for 15 minutes ▶ Shovel absorbent/decontaminant solution mixture into a steel drum. ▶ Decontaminate surface. - Pour an equal amount of neutraliser solution over contaminated surface. - Scrub area with a stiff bristle brush, using moderate pressure. - Completely cover decontaminant with vermiculite or other similar absorbent. - After 5 minutes, shovel absorbent/decontamination solution mixture into the same steel drum used above. ▶ Monitor for residual isocyanate. If surface is decontaminated, proceed to next step. If contamination persists, repeat decontaminate procedure immediately above ▶ Place loosely covered drum (release of carbon dioxide) outside for at least 72 hours. Label waste-containing drum appropriately. Remove waste materials for incineration. ▶ Decontaminate and remove personal protective equipment. ▶ Return to normal operation. ▶ Conduct accident investigation and consider measures to prevent reoccurrence. <p>Decontamination: Treat isocyanate spills with sufficient amounts of isocyanate decontaminant preparation ("neutralising fluid"). Isocyanates and polyisocyanates are generally not miscible with water. Liquid surfactants are necessary to allow better dispersion of isocyanate and neutralising fluids/preparations. Alkaline neutralisers react faster than water/surfactant mixtures alone. Typically, such a preparation may consist of: Sawdust: 20 parts by weight Kieselguhr 40 parts by weight plus a mixture of {ammonia (s.g. 0.880) 8% v/v non-ionic surfactant 2% v/v water 90% v/v}. Let stand for 24 hours</p>

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Three commonly used neutralising fluids each exhibit advantages in different situations.

Formulation A :

liquid surfactant	0.2-2%
sodium carbonate	5-10%
water to	100%

Formulation B

liquid surfactant	0.2-2%
concentrated ammonia	3-8%
water to	100%

Formulation C

ethanol, isopropanol or butanol	50%
concentrated ammonia	5%
water to	100%

After application of any of these formulae, let stand for 24 hours.

Formulation B reacts faster than Formulation A. However, ammonia-based neutralisers should be used only under well-ventilated conditions to avoid overexposure to ammonia or if members of the emergency team wear suitable respiratory protection. Formulation C is especially suitable for cleaning of equipment from unreacted isocyanate and neutralizing under freezing conditions. Regard has to be taken to the flammability of the alcoholic solution.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ 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. ▶ Avoid all personal 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.
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Other information	<ul style="list-style-type: none"> ▶ 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. ▶ 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. <p>In addition, for tank storages (where appropriate):</p> <ul style="list-style-type: none"> ▶ 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. <p>for commercial quantities of isocyanates:</p> <ul style="list-style-type: none"> - Isocyanates should be stored in adequately banded areas. Nothing else should be kept within the same bunding. Pre-polymers need not be segregated. Drums of isocyanates should be stored under cover, out of direct sunlight, protected from rain, protected from physical damage and well away from moisture, acids and alkalis. - Where isocyanates are stored at elevated temperatures to prevent solidifying, adequate controls should be installed to prevent the high temperatures and precautions against fire should be taken. - Where stored in tanks, the more reactive isocyanates should be blanketed with a non-reactive gas such as nitrogen and equipped with absorptive type breather valve (to prevent vapour emissions).. - Transfer systems for isocyanates in bulk storage should be fully enclosed and use pump or vacuum systems. Warning signs, in appropriate languages, should be posted where necessary. - Areas in which polyurethane foam products are stored should be supplied with good general ventilation. Residual amounts of unreacted isocyanate may be present in the finished foam, resulting in hazardous atmospheric concentrations.
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Ideal storage temperature range is dependent on the specific polymer due to viscosity and melting point differences between the polymers. Use 25 deg C (77 deg F) to 30 deg C (86 deg F) as a guideline to most liquid isocyanates for optimum storage temperature. If some isocyanates are stored at or below a temperature of 25 deg C (77 deg F), crystallization and settling of the isocyanate may occur. Storage in a cold warehouse can cause crystals to form. These crystals can settle to the bottom of the container. If crystals do form, they can be melted easily with moderate heat. It is suggested that a container the size of a drum be warmed for 16-24 hours at sufficient temperature to melt the crystals. When the crystals are melted, the container should be agitated by rolling or stirring, until the contents are homogenous. Since heated isocyanate will generate vapors more rapidly than product stored at 25 deg C (77 deg F), be sure to follow the precautions under the Personal Protection.

Conditions for safe storage, including any incompatibilities

Suitable container	<p>Pails.</p> <ul style="list-style-type: none"> ▶ 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. ▶ Packaging as recommended by manufacturer. <p>Check that containers are clearly labelled and free from leaks</p>
Storage incompatibility	<ul style="list-style-type: none"> ▶ Vigorous reactions, sometimes amounting to explosions, can result from the contact between aromatic rings and strong oxidising agents. ▶ Aromatics can react exothermically with bases and with diazo compounds. ▶ Avoid contamination with water, alkalis and detergent solutions. ▶ Material reacts with water and generates gas, pressurises containers with even drum rupture resulting. ▶ DO NOT reseal container if contamination is suspected. ▶ Open all containers with care.

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	titanium dioxide	Titanium dioxide	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	di-sec-octyl phthalate	Di-sec-octyl phthalate	5 mg/m3	10 mg/m3	Not Available	Not Available
Australia Exposure Standards	4,4'-diphenylmethane diisocyanate (MDI)	Methylene bisphenyl isocyanate (MDI)	0.02 mg/m3	0.07 mg/m3	Not Available	Not Available
Australia Exposure Standards	carbon black	Carbon black	3 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Precipitated silica	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica, fused	0.05 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Silica gel	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Fumed silica (respirable dust)	2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Fume (thermally generated)(respirable dust)	2 mg/m3	Not Available	Not Available	(e) Containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Diatomaceous earth (uncalcined)	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	cumene	Cumene	25 ppm / 125 mg/m3	375 mg/m3 / 75 ppm	Not Available	Not Available
Australia Exposure Standards	diisobutyl ketone	Diisobutyl ketone	25 ppm / 145 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	p-toluenesulfonyl isocyanate	Isocyanates, all (as-NCO)	0.02 mg/m3	0.07 mg/m3	Not Available	Not Available
Australia Exposure Standards	dibutyltin dilauryl mercaptide	Tin, organic compounds (as Sn)	0.1 mg/m3	0.2 mg/m3	Not Available	(g) Some compounds in these groups are classified as carcinogenic or as sensitisers. Check individual classification details on the safety data sheet for information on classification.

DURAM ROCKTUFF (BLACK, DARK GREY, LIGHT GREY, YELLOW)

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
naphtha petroleum, light aromatic solvent	1,200 mg/m3	6,700 mg/m3	40,000 mg/m3
polyethylene	16 mg/m3	170 mg/m3	1,000 mg/m3
1,2,4-trimethyl benzene	140 mg/m3	360 mg/m3	2,200 mg/m3
1,2,4-trimethyl benzene	Not Available	Not Available	480 ppm
titanium dioxide	30 mg/m3	330 mg/m3	2,000 mg/m3
di-sec-octyl phthalate	10 mg/m3	1,000 mg/m3	6,100 mg/m3
4,4'-diphenylmethane diisocyanate (MDI)	0.45 mg/m3	Not Available	Not Available
4,4'-diphenylmethane diisocyanate (MDI)	29 mg/m3	40 mg/m3	240 mg/m3
1,3,5-trimethyl benzene	Not Available	Not Available	480 ppm
carbon black	9 mg/m3	99 mg/m3	590 mg/m3
silica amorphous	18 mg/m3	200 mg/m3	1,200 mg/m3
silica amorphous	18 mg/m3	100 mg/m3	630 mg/m3
silica amorphous	120 mg/m3	1,300 mg/m3	7,900 mg/m3
silica amorphous	45 mg/m3	500 mg/m3	3,000 mg/m3
silica amorphous	18 mg/m3	740 mg/m3	4,500 mg/m3
cumene	Not Available	Not Available	Not Available
diisobutyl ketone	75 ppm	330 ppm	2000* ppm
1-decene homopolymer, hydrogenated	30 mg/m3	330 mg/m3	2,000 mg/m3

Ingredient	Original IDLH	Revised IDLH
naphtha petroleum, light aromatic solvent	Not Available	Not Available
polyethylene	Not Available	Not Available
1,2,4-trimethyl benzene	Not Available	Not Available
titanium dioxide	5,000 mg/m3	Not Available
di-sec-octyl phthalate	5,000 mg/m3	Not Available
MDI homopolymer	Not Available	Not Available
4,4'-diphenylmethane diisocyanate (MDI)	75 mg/m3	Not Available
C.I. Pigment Yellow 74	Not Available	Not Available
1,3,5-trimethyl benzene	Not Available	Not Available
carbon black	1,750 mg/m3	Not Available
silica amorphous	3,000 mg/m3	Not Available
cumene	900 ppm	Not Available
diisobutyl ketone	500 ppm	Not Available
1-decene homopolymer, hydrogenated	Not Available	Not Available
bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate	Not Available	Not Available
p-toluenesulfonyl isocyanate	Not Available	Not Available
C7-9 branched alkyl-3,5-di-tert-butyl-4-hydroxyhydrocinnamate	Not Available	Not Available
dibutyltin dilauryl mercaptide	25 mg/m3	Not Available
methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
naphtha petroleum, light aromatic solvent	E	≤ 0.1 ppm
1,2,4-trimethyl benzene	E	≤ 0.1 ppm
MDI homopolymer	E	≤ 0.1 ppm
1,3,5-trimethyl benzene	E	≤ 0.1 ppm
bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate	D	> 0.1 to ≤ 1 ppm

Notes:

Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.

DURAM ROCKTUFF (BLACK, DARK GREY, LIGHT GREY, YELLOW)


Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate	D	> 0.1 to ≤ 1 ppm
Notes:	<i>Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.</i>	

MATERIAL DATA

NOTE P: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.01% w/w benzene (EINECS No 200-753-7). Note E shall also apply when the substance is classified as a carcinogen. This note applies only to certain complex oil-derived substances in Annex VI.

European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

Exposure controls

Appropriate engineering controls	<p>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:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>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.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <ul style="list-style-type: none"> ▶ Spraying of material or material in admixture with other components must be carried out in conditions conforming to local state regulations (AS/NZS 4114, UNI EN 12215:2010, ANSI/AIHA Z9.3-2007 or national equivalent). ▶ Local exhaust ventilation with full face positive-pressure air supplied breathing apparatus (hood or helmet type) is required. ▶ Spraying should be performed in a spray booth fitted with an effective exhaust system which complies with local environmental legislation. ▶ The spray booth area must be isolated from unprotected personnel whilst spraying is in progress and until all spraying mist has cleared. <p>NOTE: Isocyanate vapours will not be adequately absorbed by organic vapour respirators. 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.</p> <table border="1" style="width: 100%;"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> </tbody> </table> <p>Within each range the appropriate value depends on:</p> <table border="1" style="width: 100%;"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>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 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 4-10 m/s (800-2000 f/min.) for extraction of solvents generated by spraying at a point 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.</p>	Type of Contaminant:	Air Speed:	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.)	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
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Personal protection															
Eye and face protection	<ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Chemical goggles. ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent] 														
Skin protection	See Hand protection below														
Hands/feet protection	<p>NOTE:</p> <ul style="list-style-type: none"> ▶ 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. <p>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.</p> <p>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.</p> <p>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.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> · 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. 														

DURAM ROCKTUFF (BLACK, DARK GREY, LIGHT GREY, YELLOW)

	<ul style="list-style-type: none"> • 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. <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> • Excellent when breakthrough time > 480 min • Good when breakthrough time > 20 min • Fair when breakthrough time < 20 min • Poor when glove material degrades <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>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.</p> <p>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.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> • 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 <p>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.</p> <ul style="list-style-type: none"> ▶ Isocyanate resistant materials include Teflon, Viton, nitrile rubber and some PVA gloves. ▶ Protective gloves and overalls should be worn as specified in the appropriate national standard. ▶ Contaminated garments should be removed promptly and should not be re-used until they have been decontaminated. ▶ NOTE: Natural rubber, neoprene, PVC can be affected by isocyanates
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ 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:

DURAM ROCKTUFF (BLACK, DARK GREY, LIGHT GREY, YELLOW)

Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVA	C
PVC	C
VITON	C

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

C: Poor to Dangerous Choice for other than short term immersion

NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -

* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

Type AB-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	AB-AUS P3	-	AB-PAPR-AUS / Class 1 P3
up to 50 x ES	-	AB-AUS / Class 1 P3	-
up to 100 x ES	-	AB-2 P3	AB-PAPR-2 P3 ^

^ - 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)

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Coloured viscous flammable liquid; does not mix with water.
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DURAM ROCKTUFF (BLACK, DARK GREY, LIGHT GREY, YELLOW)

Physical state	Liquid	Relative density (Water = 1)	1.1
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Applicable	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	160	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	58	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Flammable.	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (%)	Not Applicable
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ▶ Unstable in the presence of incompatible materials. ▶ Product is considered stable. ▶ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	<p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may produce toxic effects. 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.</p> <p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Inhalation hazard is increased at higher temperatures.</p> <p>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</p>
Ingestion	<p>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.</p> <p>Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).</p>
Skin Contact	<p>Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>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.</p>
Eye	<p>Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p>
Chronic	<p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial number of individuals at a greater frequency than would be expected from the response of a normal population.</p> <p>Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking.</p> <p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of</p>

DURAM ROCKTUFF (BLACK, DARK GREY, LIGHT GREY, YELLOW)

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.

There is sufficient evidence to provide a strong presumption that human exposure to the material may result in impaired fertility on the basis of: - clear evidence in animal studies of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects but which is not a secondary non-specific consequence of other toxic effects.

There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of:

- clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.

Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

2-Ethylhexanoic acid (2-EHA) its esters and its salts are of concern to human health because of their potential to induce carcinogenicity, liver toxicity and developmental/reproductive toxicity. 2-EHA is of low acute oral and dermal toxicity, is a mild skin irritant and a severe eye irritant. It is not mutagenic in Ames test, but is capable of inducing chromosome aberration and sister chromatid exchanges *in vitro*, liver toxicity and liver tumours after repeated dose treatment. In addition, 2-EHA has been associated with reproductive and developmental toxicity in experimental animals.

2-EHA is quickly resorbed orally, dermally and following inhalation and almost fully excreted mainly in urine. As in the case of fatty acids, degradation mainly takes place by means of peroxisomal beta-oxidation.

2-EHA has been shown to be a liver and developmental toxicant in animal studies at high doses; in developmental toxicity studies, it was postulated that the maternal liver toxicity began a cascade of effects that included metallothionein (MT) induction, zinc accumulation in the liver due to MT binding, and a resulting zinc deficiency in the developing embryo; the zinc deficiency causes the developmental toxicity; a reproductive/developmental toxicity study was also performed with up to 1% dietary di-2-ethylhexyl terephthalate (DEHT; a 2-ethylhexanoic acid precursor); no reproductive or developmental effects were observed, suggesting that the process of metabolic conversion of DEHT to 2-ethylhexanol and subsequent hydrolysis to 2-ethylhexanoic acid results in a time course of 2-ethylhexanoic acid appearance such that allows clearance before sufficient levels can arise to produce acute liver toxicity.

The closely related substance, 2-ethylhexanol is metabolised to form 2-ethylhexanoic acid and so presents a similar toxicity profile..

2-Ethylhexanoic acid (CAS No. 149-57-5) is classified as hazardous, as a Category 3 reproductive toxin, with the risk phrase 'Possible risk of harm to the unborn child' (Xn; R63). These effects were noted in the absence of signs of maternal toxicity. The lowest observed adverse effect level (LOAEL) for developmental toxicity was reported to be 100 mg/kg bw/day. Effects on fertility were also reported, with evidence being sufficient to warrant classification as potentially toxic to fertility.

2-Ethylhexanol (CAS No. 104-76-7) was reported to cause developmental toxicity, but not teratogenicity, in rats following treatment via the oral route (NICNAS a). These effects were noted in the absence of signs of marked maternal toxicity. The no observed adverse effect level (NOAEL) for developmental toxicity was reported to be 130 mg/kg bw/day.

Various studies on reproduction toxicity have produced indications of an embryotoxic effect of 2-EHA. After oral administration, NOAEL values for maternal toxicity and foetotoxic effects of 2-EHA were determined in rabbits at 25 and >250 mg/kg body weight/day and in rats at 250 and 100 mg/kg body weight/day. The foetotoxic findings in rats were based on a reduced skeleton ossification at the next higher dose (250 mg/kg body weight/day). No teratogenic effects were observed in this study. In comparison with the structural isomer valproic acid, a known human teratogen, 2-EHA does have similar reprotoxic effects at maternal toxic doses in animal experiments but a far lower potency

Following sub-chronic oral administration of 2-EHA, critical effects like liver changes (higher relative liver weight, histological changes in hepatocytes) were observed in rats and mice and histological renal tubule results were observed in mice. Furthermore, statistically significant, higher cholesterol values were found in all treated male rats (61, 303 and 917 mg/kg body weight/day) and in male and female mice in the middle and high dose groups (885-3139 mg/kg body weight/day). In rats the maximum dose with no adverse effect (NOAEL) was 61 mg/kg body weight/day. In bacterial test systems, mutagenicity studies produced negative findings. In test systems with mammalian cells, by contrast, the findings were weakly positive. Cytogenetic and SCE studies involving CHO cells were positive, one SCE test in human lymphocytes was questionably positive and one experiment concerning tritium-thymidine incorporation into the DNA of mouse lymphocytes was negative.

Furthermore, An unpublished micronucleus study on the bone marrow of CD-1 mice was conducted in compliance with OECD Guideline 474. No significant increase in the micronuclei was observed at doses of 400, 800 or 1,600 mg/kg body weight (Inveresk Research International Ltd, 1994). Furthermore, *in vitro* and *in vivo* genotoxicity data (micronucleus test, dominant lethal test) are available for 2-ethylhexanol which is rapidly and quantitatively converted into 2-EHA in metabolism studies. This data do not indicate any genotoxic potential which means that such an effect of 2-EHA is not likely either. As 2-EHA can induce both DNA synthesis and inhibition of intercellular communication in hepatic cells, a tumour-promoting potential in rodents, comparable to that of other peroxisome proliferators, cannot be ruled out. The carcinogenic effect of peroxisome proliferators in rodents (e.g. of di(2-ethylhexyl)phthalate, DEHP) is not deemed to be relevant for humans.

Calcium/zinc and barium/zinc salts of 2-EHA are used as thermo-stabilisers for PVC, together with co-stabilisers like polyols or epoxy compounds, in order to capture the hydrochloride cleaved during the thermal loading of PVC; in addition various salts are used in other food and beverage containers as plasticisers. The migration of 2-EHA from the sealing compounds in the metal lids. has been demonstrated in food contamination. The potential for human exposure to 2-EHA therefore is significant.

For di-sec-octyl phthalate:

Oral studies of 90-days to 2-years in rat, 1-year in guinea pig and up to 1-year in dog have shown a no-effect level of about 60 mg/kg/day. Higher doses produced growth retardation and increased weights of livers and kidneys.

Rats and mice fed on diets containing 6000-12000 (rats) and 3000-6000 (mice) mg/kg body weight for 103 weeks showed an increased incidence of hepatocellular carcinomas in female rats and male and female mice, and an increased incidence of either hepatocellular carcinomas or neoplastic nodules in male rats. About 35% of the hepatocellular carcinomas in mice had metastasised to the lungs.

The substance can cause testicular damage in rats (dietary and gavage studies) with a no-effect level in 0.3% to 0.5% in the diet. Inhalation or dermal exposures did not produce testicular effects. When the substance was fed to pregnant rats (5 ml/kg) it produced slight effects on embryonic and foetal development with skeletal abnormalities more common.

Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/or memory loss, tremor in the fingers and tongue, vertigo, olfactory disorders, paraesthesias of the extremities, weight loss and anaemia and degenerative changes in the liver and kidney. Chronic exposure by petroleum workers, to the lighter hydrocarbons, has been associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness and paraesthesias), psychological and neurophysiological deficits, bone marrow toxicities (including hypoplasia possibly due to benzene) and hepatic and renal involvement. Chronic dermal exposure to petroleum hydrocarbons may result in defatting which produces localised dermatoses.

Surface cracking and erosion may also increase susceptibility to infection by microorganisms. One epidemiological study of petroleum refinery workers has reported elevations in standard mortality ratios for skin cancer along with a dose-response relationship indicating an association between routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have been unable to confirm this finding.

Hydrocarbon solvents are liquid hydrocarbon fractions derived from petroleum processing streams, containing only carbon and hydrogen atoms,

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with carbon numbers ranging from approximately C5-C20 and boiling between approximately 35-370 deg C. Many of the hydrocarbon solvents have complex and variable compositions with constituents of 4 types, alkanes (normal paraffins, isoparaffins, and cycloparaffins) and aromatics (primarily alkylated one- and two-ring species). Despite the compositional complexity, most hydrocarbon solvent constituents have similar toxicological properties, and the overall toxicological hazards can be characterized in generic terms. Hydrocarbon solvents can cause chemical pneumonitis if aspirated into the lung, and those that are volatile can cause acute CNS effects and/or ocular and respiratory irritation at exposure levels exceeding occupational recommendations. Otherwise, there are few toxicologically important effects. The exceptions, n-hexane and naphthalene, have unique toxicological properties

Animal studies:

No deaths or treatment related signs of toxicity were observed in rats exposed to light alkylate naphtha (paraffinic hydrocarbons) at concentrations of 668, 2220 and 6646 ppm for 6 hrs/day, 5 days/wk for 13 weeks. Increased liver weights and kidney toxicity (male rats) was observed in high dose animals. Exposure to pregnant rats at concentrations of 137, 3425 and 6850 ppm did not adversely affect reproduction or cause maternal or foetal toxicity. Lifetime skin painting studies in mice with similar naphthas have shown weak or no carcinogenic activity following prolonged and repeated exposure. Similar

naphthas/distillates, when tested at nonirritating dose levels, did not show any significant carcinogenic activity indicating that this tumorigenic response is likely related to chronic irritation and not to dose. The mutagenic potential of naphthas has been reported to be largely negative in a variety of mutagenicity tests. The exact relationship between these results and human health is not known. Some components of this product have been shown to produce a species specific, sex hormonal dependent kidney lesion in male rats from repeated oral or inhalation exposure. Subsequent research has shown that the kidney damage develops via the formation of an alpha-2u-globulin, a mechanism unique to the male rat. Humans do not form alpha-2u-globulin, therefore, the kidney effects resulting from this mechanism are not relevant in human.

Isocyanate vapours/mists are irritating to the upper respiratory tract and lungs; the response may be severe enough to produce bronchitis with wheezing, gasping and severe distress, even sudden loss of consciousness, and pulmonary oedema. Possible neurological symptoms arising from isocyanate exposure include headache, insomnia, euphoria, ataxia, anxiety neurosis, depression and paranoia. Gastrointestinal disturbances are characterised by nausea and vomiting. Pulmonary sensitisation may produce asthmatic reactions ranging from minor breathing difficulties to severe allergic attacks; this may occur following a single acute exposure or may develop without warning after a period of tolerance. A respiratory response may occur following minor skin contact. Skin sensitisation is possible and may result in allergic dermatitis responses including rash, itching, hives and swelling of extremities.

Isocyanate-containing vapours/ mists may cause inflammation of eyes and nasal passages.

Onset of symptoms may be immediate or delayed for several hours after exposure. Sensitised people can react to very low levels of airborne isocyanates. Unprotected or sensitised persons should not be allowed to work in situations allowing exposure to this material.

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	Not Available	Not Available
naphtha petroleum, light aromatic solvent	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1900 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation(Rat) LC50; >4.42 mg/L4h ^[1]	Skin: adverse effect observed (irritating) ^[1]
	Oral(Rat) LD50; >4500 mg/kg ^[1]	
polyethylene	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Not Available
	Oral(Rat) LD50; >2000 mg/kg ^[1]	
1,2,4-trimethyl benzene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >3160 mg/kg ^[2]	Not Available
	Inhalation(Rat) LC50; 10.2 mg/L4h ^[1]	
	Oral(Rat) LD50; 6000 mg/kg ^[1]	
titanium dioxide	TOXICITY	IRRITATION
	dermal (hamster) LD50: >=10000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation(Rat) LC50; >2.28 mg/L4h ^[1]	Skin (human): 0.3 mg /3D (int)-mild *
	Oral(Rat) LD50; >=2000 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
di-sec-octyl phthalate	TOXICITY	IRRITATION
	dermal (rat) LD50: 250 mg/kg ^[2]	Eye (rabbit): 500 mg/24h mild
	Inhalation(Rat) LC50; >10.62 mg/L4h ^[2]	Eye: adverse effect observed (irritating) ^[1]
	Oral(Mouse) LD50; >9860 mg/kg ^[2]	Skin (rabbit): 500 mg/24h mild
		Skin: adverse effect observed (irritating) ^[1]
MDI homopolymer	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >9400 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation(Rat) LC50; 0.368 mg/L4h ^[1]	Skin: adverse effect observed (irritating) ^[1]
	Oral(Rat) LD50; >2000 mg/kg ^[1]	
4,4'-diphenylmethane diisocyanate (MDI)	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >6200 mg/kg ^[2]	Dermal Sensitiser *
	Inhalation(Rat) LC50; 0.368 mg/L4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral(Rat) LD50; >2000 mg/kg ^[1]	Skin (rabbit): 500 mg /24 hours
		Skin: adverse effect observed (irritating) ^[1]

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C.I. Pigment Yellow 74	TOXICITY	IRRITATION
	Oral(Rat) LD50; >2000 mg/kg ^[1]	Eye (human): non irritant Skin (human): non irritant
1,3,5-trimethyl benzene	TOXICITY	IRRITATION
	dermal (rat) LD50: >3460 mg/kg ^[1]	Eye (rabbit): 500 mg/24h mild
	Inhalation(Rat) LC50; 10.2 mg/L4h ^[1]	Eye: adverse effect observed (irritating) ^[1]
	Oral(Rat) LD50; 6000 mg/kg ^[1]	Skin (rabbit): 20 mg/24h moderate Skin: adverse effect observed (irritating) ^[1]
carbon black	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1] Oral(Rat) LD50; >8000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1] Skin: no adverse effect observed (not irritating) ^[1]
silica amorphous	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): non-irritating *
	Inhalation(Rat) LC50; >0.139 mg/L4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral(Rat) LD50; >1000 mg/kg ^[1]	Skin (rabbit): non-irritating * Skin: no adverse effect observed (not irritating) ^[1]
cumene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 2000 mg/kg ^[2]	Eye (rabbit): 500 mg/24h mild
	Inhalation(Rat) LC50; 39 mg/L4h ^[2]	Eye (rabbit): 86 mg mild
	Oral(Rat) LD50; ~1400 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1] Skin (rabbit): 10 mg/24h mild Skin (rabbit):100 mg/24h moderate Skin: no adverse effect observed (not irritating) ^[1]
diisobutyl ketone	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (human): 25 ppm/15min - mild
	Inhalation(Guinea) LC50; >14.5 mg/l4h ^[1]	Eye (rabbit): 500 mg
	Oral(Rat) LD50; >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1] Skin (g.pig): repeated - SEVERE Skin (g.pig): Strong * Skin (rabbit): 10 mg/24h - mild Skin (rabbit): 500 mg - mild Skin: adverse effect observed (irritating) ^[1] Skin: no adverse effect observed (not irritating) ^[1]
1-decene homopolymer, hydrogenated	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye*(rabbit):0-4/110.0-nonirritant
	Inhalation(Rat) LC50; 0.9 mg/l4h ^[1] Oral(Rat) LD50; >2000 mg/kg ^[1]	Skin**(rabbit)-0.5/8.0-nonirritant
bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate	TOXICITY	IRRITATION
	Oral(Rat) LD50; 2369-3920 mg/kg ^[2]	Not Available
p-toluenesulfonyl isocyanate	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Not Available
	Inhalation(Rat) LC50; >320 ppm4h ^[2] Oral(Rat) LD50; 2234 mg/kg ^[2]	
C7-9 branched alkyl-3,5-di-tert-butyl-4-hydroxyhydrocinnamate	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1] Oral(Rat) LD50; >500 mg/kg ^[1]	Eye (rabbit): non-irritating * Skin (rat): non-irritating *
dibutyltin dilauryl mercaptide	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1000<2000 mg/kg ^[1] Oral(Rat) LD50; >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1] Skin: adverse effect observed (irritating) ^[1]

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methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate	TOXICITY	IRRITATION
	Not Available	Not Available
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		

<p>NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT</p>	<p>For C9 aromatics (typically trimethylbenzenes - TMBs)</p> <p>Acute Toxicity</p> <p>Acute toxicity studies (oral, dermal and inhalation routes of exposure) have been conducted in rats using various solvent products containing predominantly mixed C9 aromatic hydrocarbons (CAS RN 64742-95-6). Inhalation LC50's range from 6,000 to 10,000 mg/m³ for C9 aromatic naphtha and 18,000 to 24,000 mg/m³ for 1,2,4 and 1,3,5-TMB, respectively. A rat oral LD50 reported for 1,2,4-TMB is 5 grams/kg bw and a dermal LD50 for the C9 aromatic naphtha is >4 ml/kg bw. These data indicate that C9 aromatic solvents show that LD50/LC50 values are greater than limit doses for acute toxicity studies established under OECD test guidelines.</p> <p>Irritation and Sensitization</p> <p>Several irritation studies, including skin, eye, and lung/respiratory system, have been conducted on members of the category. The results indicate that C9 aromatic hydrocarbon solvents are mildly to moderately irritating to the skin, minimally irritating to the eye, and have the potential to irritate the respiratory tract and cause depression of respiratory rates in mice. Respiratory irritation is a key endpoint in the current occupational exposure limits established for C9 aromatic hydrocarbon solvents and trimethylbenzenes. No evidence of skin sensitization was identified.</p> <p>Repeated Dose Toxicity</p> <p>Inhalation: The results from a subchronic (3 month) neurotoxicity study and a one-year chronic study (6 hr/day, 5 days/week) indicate that effects from inhalation exposure to C9 Aromatic Hydrocarbon Solvents on systemic toxicity are slight. A battery of neurotoxicity and neurobehavioral endpoints were evaluated in the 3-month inhalation study on C9 aromatic naphtha tested at concentrations of 0, 101, 452, or 1320 ppm (0, 500, 2,220, or 6,500 mg/m³). In this study, other than a transient weight reduction in the high exposure group (not statistically significant at termination of exposures), no effects were reported on neuropathology or neuro/behavioral parameters. The NOAEL for systemic and/or neurotoxicity was 6,500 mg/m³, the highest concentration tested. In an inhalation study of a commercial blend, rats were exposed to C9 aromatic naphtha concentrations of 0, 96, 198, or 373 ppm (0, 470, 970, 1830 mg/m³) for 6 hr/day, 5 days/week, for 12 months. Liver and kidney weights were increased in the high exposure group but no accompanying histopathology was observed in these organs. The NOAEL was considered to be the high exposure level of 373 ppm, or 1830 mg/m³. In two subchronic rat inhalation studies, both of three months duration, rats were exposed to the individual TMB isomers (1,2,4-and 1,3,5-) to nominal concentrations of 0, 25, 100, or 250 ppm (0, 123, 492, or 1230 mg/m³). Respiratory irritation was observed at 492 (100 ppm) and 1230 mg/m³ (250 ppm) and no systemic toxicity was observed in either study. For both pure isomers, the NOELs are 25 ppm or 123 mg/m³ for respiratory irritation and 250 ppm or 1230 mg/m³ for systemic effects.</p> <p>Oral: The C9 aromatic naphtha has not been tested via the oral route of exposure. Individual TMB isomers have been evaluated in a series of repeated-dose oral studies ranging from 14 days to 3 months over a wide range of doses. The effects observed in these studies included increased liver and kidney weights, changes in blood chemistry, increased salivation, and decreased weight gain at higher doses. Organ weight changes appeared to be adaptive as they were not accompanied by histopathological effects. Blood changes appeared sporadic and without pattern. One study reported hyaline droplet nephropathy in male rats at the highest dose (1000 mg/kg bw-day), an effect that is often associated with alpha-2mu-globulin-induced nephropathy and not considered relevant to humans. The doses at which effects were detected were 100 mg/kg-bw day or above (an exception was the pilot 14 day oral study - LOAEL 150 mg/kg bw-day - but the follow up three month study had a LOAEL of 600 mg/kg-bw-day with a NOAEL of 200 mg/kg bw-day). Since effects generally were not severe and could be considered adaptive or spurious, oral exposure does not appear to pose a high toxicity hazard for pure trimethylbenzene isomers.</p> <p>Mutagenicity</p> <p>In vitro genotoxicity testing of a variety of C9 aromatics has been conducted in both bacterial and mammalian cells. In vitro point mutation tests were conducted with Salmonella typhimurium and Escherichia coli bacterial strains, as well as with cultured mammalian cells such as the Chinese hamster cell ovary cells (HGPRT assay) with and without metabolic activation. In addition, several types of in vitro chromosomal aberration tests have been performed (chromosome aberration frequency in Chinese hamster ovary and lung cells, sister chromatid exchange in CHO cells). Results were negative both with and without metabolic activation for all category members. For the supporting chemical 1,2,3-TMB, a single in vitro chromosome aberration test was weakly positive. In in vivo bone marrow cytogenetics test, rats were exposed to C9 aromatic naphtha at concentrations of 0, 153, 471, or 1540 ppm (0, 750, 2,310, or 7,560 mg/m³) 6 hr/day, for 5 days. No evidence of in vivo somatic cell genotoxicity was detected. Based on the cumulative results of these assays, genetic toxicity is unlikely for substances in the C9 Aromatic Hydrocarbon Solvents Category.</p> <p>Reproductive and Developmental Toxicity</p> <p>Results from the three-generation reproduction inhalation study in rats indicate limited effects from C9 aromatic naphtha. In each of three generations (F0, F1 and F2), rats were exposed to High Flash Aromatic Naphtha (CAS RN 64742-95-6) via whole body inhalation at target concentrations of 0, 100, 500, or 1500 ppm (actual mean concentrations throughout the full study period were 0, 103, 495, or 1480 ppm, equivalent to 0, 505, 2430, or 7265 mg/m³, respectively). In each generation, both sexes were exposed for 10 weeks prior to and two weeks during mating for 6 hrs/day, 5 days/wks. Female rats in the F0, F1, and F2 generation were then exposed during gestation days 0-20 and lactation days 5-21 for 6 hrs/day, 7 days/wk. The age at exposure initiation differed among generations; F0 rats were exposed starting at 9 weeks of age, F1 exposure began at 5-7 weeks, and F2 exposure began at postnatal day (PND) 22. In the F0 and F1 parental generations, 30 rats/sex/group were exposed and mated. However, in the F2 generation, 40/sex/group were initially exposed due to concerns for toxicity, and 30/sex/group were randomly selected for mating, except that all survivors were used at 1480 ppm. F3 litters were not exposed directly and were sacrificed on lactation day 21.</p> <p>Systemic Effects on Parental Generations:</p> <p>The F0 males showed statistically and biologically significantly decreased mean body weight by ~15% at 1480 ppm when compared with controls. Seven females died or were sacrificed in extremis at 1480 ppm. The F0 female rats in the 495 ppm exposed group had a 13% decrease in body weight gain when adjusted for initial body weight when compared to controls. The F1 parents at 1480 ppm had statistically significantly decreased mean body weights (by ~13% (females) and 22% (males)), and locomotor activity. F1 parents at 1480 ppm had increased ataxia and mortality (six females). Most F2 parents (70/80) exposed to 1480 ppm died within the first week. The remaining animals survived throughout the rest of the exposure period. At week 4 and continuing through the study, F2 parents at 1480 ppm had statistically significant mean body weights much lower than controls (~33% for males; ~28% for females); body weights at 495 ppm were also reduced significantly (by 13% in males and 15% in females). The male rats in the 495 ppm exposed group had a 12% decrease in body weight gain when adjusted for initial body weight when compared to controls. Based on reduced body weight observed, the overall systemic toxicity LOAEC is 495 ppm (2430 mg/m³).</p> <p>Reproductive Toxicity-Effects on Parental Generations: There were no pathological changes noted in the reproductive organs of any animal of the F0, F1, or F2 generation. No effects were reported on sperm morphology, gestational period, number of implantation sites, or post-implantation loss in any generation. Also, there were no statistically or biologically significant differences in any of the reproductive parameters, including: number of mated females, copulatory index, copulatory interval, number of females delivering a litter, number of females delivering a live litter, or male fertility in the F0 or in the F2 generation. Male fertility was statistically significantly reduced at 1480 ppm in the F1 rats. However, male fertility was not affected in the F0 or in the F2 generations; therefore, the biological significance of this change is unknown and may or may not be attributed to the test substance. No reproductive effects were observed in the F0 or F1 dams exposed to 1480 ppm (7265 mg/m³). Due to excessive mortality at the highest concentration (1480 ppm, only six dams available) in the F2 generation, a complete evaluation is precluded. However, no clear signs of reproductive toxicity were observed in the F2 generation. Therefore, the reproductive NOAEC is considered 495 ppm (2430 mg/m³), which excludes analysis of the highest concentration due to excessive mortality.</p> <p>Developmental Toxicity - Effects on Pups: Because of significant maternal toxicity (including mortality) in dams in all generations at the highest concentration (1480 ppm), effects in offspring at 1480 ppm are not reported here. No significant effects were observed in the F1 and F2</p>
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	<p>generation offspring at 103 or 495 ppm. However, in F3 offspring, body weights and body weight gain were reduced by ~ 10-11% compared with controls at 495 ppm for approximately a week (PND 14 through 21). Maternal body weight was also depressed by ~ 12% throughout the gestational period compared with controls. The overall developmental LOAEC from this study is 495 ppm (2430 mg/m3) based on the body weights reductions observed in the F3 offspring.</p> <p>Conclusion: No effects on reproductive parameters were observed at any exposure concentration, although a confident assessment of the group exposed at the highest concentration was not possible. A potential developmental effect (reduction in mean pup weight and weight gain) was observed at a concentration that was also associated with maternal toxicity.</p> <p>* [Devoe] .</p>
POLYETHYLENE	<p>polyethylene pyrolyzate</p> <p>Inclusion of polyethylene in the diet of rats at 8 g/kg/day did not result in treatment-related effects. Polyethylene implanted into rats and mice has reportedly caused local tumorigenic activity at doses of 33 to 2120 mg/kg, but the relevance to human exposure is not certain.</p>
1,2,4-TRIMETHYL BENZENE	CHEMWATCH 2325 1,3,5-trimethylbenzene
TITANIUM DIOXIDE	<p>* IUCLID</p> <p>Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.</p> <p>For titanium dioxide:</p> <p>Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals. (General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) With regard to inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral ingestion of fine titanium dioxide showed particle size-dependent absorption by the gastrointestinal tract and large interindividual variations in blood levels of titanium dioxide. Studies on the application of sunscreens containing ultrafine titanium dioxide to healthy skin of human volunteers revealed that titanium dioxide particles only penetrate into the outermost layers of the stratum corneum, suggesting that healthy skin is an effective barrier to titanium dioxide. There are no studies on penetration of titanium dioxide in compromised skin.</p> <p>Respiratory effects that have been observed among groups of titanium dioxide-exposed workers include decline in lung function, pleural disease with plaques and pleural thickening, and mild fibrotic changes. However, the workers in these studies were also exposed to asbestos and/or silica.</p> <p>No data were available on genotoxic effects in titanium dioxide-exposed humans.</p> <p>Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain. Clearance of titanium dioxide is also affected by pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols. Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide are more slowly cleared than their fine counterparts.</p> <p>Titanium dioxide causes varying degrees of inflammation and associated pulmonary effects including lung epithelial cell injury, cholesterol granulomas and fibrosis. Rodents experience stronger pulmonary effects after exposure to ultrafine titanium dioxide particles compared with fine particles on a mass basis. These differences are related to lung burden in terms of particle surface area, and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium.</p> <p>Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the generation of reactive oxygen species by both particle types. This effect is stronger for ultrafine than for fine titanium oxide, and is markedly enhanced by exposure to simulated sunlight/ultraviolet light.</p> <p>Animal carcinogenicity data</p> <p>Pigmentary and ultrafine titanium dioxide were tested for carcinogenicity by oral administration in mice and rats, by inhalation in rats and female mice, by intratracheal administration in hamsters and female rats and mice, by subcutaneous injection in rats and by intraperitoneal administration in male mice and female rats.</p> <p>In one inhalation study, the incidence of benign and malignant lung tumours was increased in female rats. In another inhalation study, the incidences of lung adenomas were increased in the high-dose groups of male and female rats. Cystic keratinizing lesions that were diagnosed as squamous-cell carcinomas but re-evaluated as non-neoplastic pulmonary keratinizing cysts were also observed in the high-dose groups of female rats. Two inhalation studies in rats and one in female mice were negative.</p> <p>Intratracheally instilled female rats showed an increased incidence of both benign and malignant lung tumours following treatment with two types of titanium dioxide. Tumour incidence was not increased in intratracheally instilled hamsters and female mice.</p> <p>In-vivo studies have shown enhanced micronucleus formation in bone marrow and peripheral blood lymphocytes of intraperitoneally instilled mice. Increased Hprt mutations were seen in lung epithelial cells isolated from titanium dioxide-instilled rats. In another study, no enhanced oxidative DNA damage was observed in lung tissues of rats that were intratracheally instilled with titanium dioxide. The results of most in-vitro genotoxicity studies with titanium dioxide were negative.</p>
DI-SEC-OCTYL PHTHALATE	<p>Di-sec-octyl phthalate (DEHP) is not acutely toxic in small laboratory animals via the oral route. The oral LD50 reported for mice is 26.3 g/kg; for rats, it is 33.8 g/kg . No skin irritation or sensitisation potential has been demonstrated in either animals or humans, and the lethal dermal dose in rabbits is about 25 ml/kg. Deaths in rats and chronic diffuse inflammation of the lung in mice exposed to DEHP at unspecified levels have been reported.</p> <p>Long-term dietary toxicity studies in rats, guinea pigs, and dogs have established a no-effect dose level of about 60 mg/kg/day, and no carcinogenic or histologic abnormalities were observed at this level . Higher doses were associated with growth retardation and increased liver and kidney weights but not histologic abnormalities. Metabolic studies have demonstrated that laboratory animals do not appreciably metabolise DEHP . Teratogenicity studies in pregnant rats indicated that fertility is unaffected at doses of 0.1, 0.2, or 0.33 percent of the acute intraperitoneal LD50 dose for rats, although slight effects on embryonic and foetal development were observed in these animals; skeletal deformities were the most common teratogenic effects observed . Mutagenic effects were observed at intravenous doses of one-third, one-half, and two-thirds of the acute LD50; these effects are consistent with DEHP's ability to produce dominant lethal mutations .</p> <p>A study of workers exposed to a mixture of the vapors of diethyl phthalate, dibutyl phthalate, and di-2-ethylhexyl phthalate reported that exposures to 1.7 to 66 mg/m3 of various combinations of airborne phthalates (including butyl phthalate, higher aryl phthalates, dioctyl phthalate and others) and noted complaints of pain, numbness, and spasms in the upper and lower extremities after six to seven years of exposure. Polyneuritis was observed in 32 percent of the workers studied, and 78 percent of these workers showed depression of vestibular receptors</p> <p>The material may produce peroxisome proliferation. Peroxisomes are single, membrane limited, cytoplasmic organelles that are found in the cells of animals, plants, fungi and protozoa. Peroxisome proliferators include certain hypolipidaemic drugs, phthalate ester plasticisers, industrial solvents, herbicides, food flavours, leukotriene D4 antagonists and hormones. Numerous studies in rats and mice have demonstrated the hepatocarcinogenic effects of peroxisome proliferators, and these compounds have been unequivocally established as carcinogens.</p> <p>However it is generally conceded that compounds inducing proliferation in rats and mice have little, if any, effect on human liver except at very high doses or extreme conditions of exposure.</p> <p>Transitional Phthalate Esters: produced from alcohols with straight-chain carbon backbones of C4 to C6. This subcategory also includes a phthalate produced from benzyl alcohol as one ester group with the second ester composed of an alkyl group with a C5 carbon backbone and</p>

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butyrate group. Phthalate esters containing >10% C4 to C6 molecules were conservatively included in this subcategory. Branched C7 and C8 isomers (di-iso-heptyl, di-iso-octyl and diethylhexyl phthalates) in contrast to linear dihexyl and dioctyl phthalate are members of this family. Transitional phthalates have varied uses, but are largely used as plasticisers for PVC. Physicochemical properties also vary in that the lower molecular weight transitional phthalates are more water-soluble than higher molecular weight transitional phthalates, but none would be characterised as highly water soluble. Transitional phthalates have lower water solubility than the low molecular weight phthalates and except for butylbenzyl phthalate (BBP), existing data suggest they do not exhibit acute or chronic aquatic toxicity. What distinguishes some of the transitional phthalates from others is their greater mammalian toxicity potential, particularly with regard to reproductive and developmental effects, compared to either the low or high molecular weight phthalate subcategories.

Acute Toxicity. The available data on phthalates spanning the carbon range from C4 to C6 indicate that phthalate esters in the transitional subcategory are minimally toxic by acute oral and dermal administration. The oral LD50 value for BBP exceeds 2 g/ kg, and for materials with higher molecular weights, the LD50 values exceed the maximum amounts which can be administered to the animals in a manner consistent with the principles of responsible animal use.

One member of this subcategory, diethylhexyl phthalate (DEHP), has been tested for acute inhalation toxicity. It did not cause an effect at the highest concentration tested. Further, considering the low volatility of these substances, inhalation exposure at toxicologically significant levels is not anticipated.

Repeated Dose Toxicity. Several substances in the C4 to C6 range, including BBP, have been tested for repeated dose toxicity in studies ranging from 3 weeks to 2 years. The principal effects found in these studies were those associated with peroxisome proliferation including liver enlargement and induction of peroxisomal enzymes. As shown in a comparative study of liver effects, the strongest inducers of peroxisome proliferation are diisononyl phthalate (DINP) and di-iso-decyl phthalate (DIDP) with substances of shorter chain length (e.g., BBP) showing much less pronounced effects. Thus it is reasonable to conclude that other members of this subcategory would show effects similar to BBP and less pronounced than DINP or DIDP. It should also be noted that the relevance of these findings to human health is, at best, questionable. It has been shown that these effects are mediated through the peroxisome proliferation-activated receptor alpha (PPARα) and that levels of PPARα are much higher in rodents than they are in humans. Thus one would expect humans to be substantially less responsive than rodents to peroxisome proliferating agents. Empirical evidence that this is true is provided by studies in primates in which repeated administration of DINP had no effects on liver, kidney or testicular parameters.

Several of the substances in the transitional phthalate esters subcategory, however, have been shown to produce testicular atrophy when given to juvenile rats at high levels. Testicular atrophy has been associated with BBP and other substances with C4 to C6 linear carbon chains. However, molecules with fewer than 4 or more than 6 carbons did not produce testicular atrophy in these studies. Although the relevance of these data are uncertain, as the testes is not a target organ for diethylhexyl phthalate (DEHP) in primates, these data do provide one of the distinguishing toxicological characteristics of this subcategory and are one of the underlying reasons supporting the differentiation of phthalate esters on the basis of length of the linear region of the carbon chain.

Genetic Toxicity (Salmonella). A number of the substances in this subcategory including the reference substance BBP has been assessed in the Salmonella and mouse lymphoma assays. All of these substances were inactive in these assays.

Chromosomal Aberrations. BBP and dihexyl phthalate (DHP) were inactive in micronucleus assays in mice. DEHP was inactive in a cytogenetics assay in rat bone marrow. Diisooheptyl phthalate was inactive in CHO cells, in vitro.

Reproductive toxicity: A series of studies assessed the structure-activity relationship of the effects of phthalate esters on fertility using a continuous breeding protocol. The test substances included in these studies were diethyl-, dipropyl-, dibutyl-, dipentyl-, d-n-hexyl-, di-2(ethylhexyl)-, and di-n-octyl phthalates. The most profound effects were on fertility (i.e., number of females delivering/ number mated) and number of live births. The substance showing the greatest activity was DEHP which produced effects at dietary levels of 0.1 % with a no effect level of 0.01 %. The next most active compounds were di-n-hexyl- and di-n-pentyl phthalate which showed effects in the range of 0.3 to 0.5 %; no effect levels were not experimentally defined. Dipropyl phthalate had an effect on live birth index at 2.5 % but produced no effects at 1.25 %. Diethyl phthalate and di-n-octyl phthalate were inactive at the highest levels tested, 2.5 % and 5.0 %, respectively. These data demonstrated that molecules with linear alkyl chains of 4 to 6 carbons profoundly affect fertility in rodents, with DEHP being the most active. Molecules with longer or shorter side chains are essentially inactive in these assays. These data were also a basis for the separation of phthalates into three categories based on length of side chain.

In addition to these data there are reproductive toxicity studies on BBP and DEHP.

A 2-generation reproductive study was conducted in rats in which BBP was administered via the diet. Parental effects were limited to changes in body weight, weight gain, and increased absolute and relative liver weights. In the F1 parents, treatment with BBP affected mating and fertility indices and sperm number and motility. The F1 male offspring exhibited shortened anogenital distance, delayed acquisition of puberty and retention of nipples and areolae as well as reproductive effects. The NOAEL of the study was reported to be 3750 mg/ kg for reproductive effects. However, for male F1 and F2 offspring, the NOEL for reproductive effects was reported to be 50 mg/ kg based on reductions in anogenital distance. These studies along with previous data provide a good basis to assess the reproductive effects of C4 to C6 phthalate esters. Although several substances (diheptyl, heptyl nonyl, heptyl undecyl) have ester side chain constituents that predominately fall in the high molecular weight subcategory, these substances are conservatively assumed to exhibit reproductive effects similar to other transitional phthalates.

Developmental toxicity: There have been extensive studies of the developmental toxicity of BBP and DEHP. These substances produce structural malformations and also affect male reproductive development. No effect levels are in the range of 50 to 300 mg/ kg bw/ day. There is also an unpublished developmental toxicity study of di-isoheptyl phthalate (DIHP). The results of these studies are broadly consistent with the structure-activity relationships previously described, i.e., that phthalate esters with linear carbon chains of C4 to C6 carbons produce much more profound effects than either shorter or longer molecules.

Phthalate esters with >10% C4 to C6 isomers were conservatively placed in the transitional subcategory. This conclusion is supported by developmental test data on "711P" (which showed structural malformations in rats at 1000 mg/ kg/ day with a NOAEL of 200 mg/ kg/ day. "711P" is an equal composition mixture of six phthalate esters consisting of linear and methyl-branched C7, C9, and C11 ester side chains. This test substance is considered by EPA under the following CAS Numbers.: 68515-44-6 (di C7), 68515-45-7 (di C9), 3648-20-2 (di C11), 111381-89-6 (C7, C9), 111381-90-9 (C7, C11), and 111381-91-0 (C9, C11). The overall content of C4 to C6 isomers in "711P" is approximately 10%, based on the contribution from methyl-branched C7 isomers e.g., di C7 (30% C4-C6); C7, C9 (15% C4-C6); and C7, C11 (15 % C4-C6). Test data on 711P were used selectively as read-across data to the C7-containing substances in the mixture, based on the C4 to C6 content of each substance in the mixture.

NOTE: Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.

Oral (rat) NOAEL: 28.9-36.1 mg/kg/day Gastrointestinal changes, respiratory system changes, somnolence, haemorrhage, necrotic changes in GI tract, lowered blood pressure, liver, endocrine tumours, foetotoxicity, paternal effects, maternal effects, specific developmental abnormalities (hepatobiliary system, musculoskeletal system, cardiovascular system, urogenital system, central nervous system, eye/ear), foetolethality recorded.

MDI HOMOPOLYMER

as polymethylene polyphenyl isocyanate

4,4'-DIPHENYLMETHANE DIISOCYANATE (MDI)

Inhalation (human) TCLo: 0.13 ppm/30 mins Eye (rabbit): 0.10 mg moderate for diisocyanates:

In general, there appears to be little or no difference between aromatic and aliphatic diisocyanates as toxicants. In addition, there are insufficient data available to make any major distinctions between polymeric (<1000 MW) and monomeric diisocyanates. Based on repeated dose studies in animals by the inhalation route, both aromatic and aliphatic diisocyanates appear to be of high concern for pulmonary toxicity at low exposure levels. Based upon a very limited data set, it appears that diisocyanate prepolymers exhibit the same respiratory tract effects as the monomers in repeated dose studies. There is also evidence that both aromatic and aliphatic diisocyanates are acutely toxic via the inhalation route. Most members of the diisocyanate category have not been tested for carcinogenic potential. Though the aromatic diisocyanates tested positive and the one aliphatic diisocyanate tested negative in one species, it is premature to make any generalizations about the carcinogenic potential of aromatic versus aliphatic diisocyanates. In the absence of more human data, it would be prudent at this time

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	<p>to assume that both aromatic and aliphatic diisocyanates are respiratory sensitisers. Diisocyanates are moderate to strong dermal sensitisers in animal studies. Skin irritation studies performed on rabbits and guinea pigs indicate no difference in the effects of aromatic versus aliphatic diisocyanates.</p> <p>For monomers, effects on the respiratory tract (lungs and nasal cavities) were observed in animal studies at exposure concentrations of less than 0.005 mg/L. The experimental animal data available on prepolymeric diisocyanates show similar adverse effects at levels that range from 0.002 mg/L to 0.026 mg/L.</p> <p>There is also evidence that both aromatic and aliphatic diisocyanates are acutely toxic via the inhalation route</p> <p>Oncogenicity: Most members of the diisocyanate category have not been tested for carcinogenic potential. Commercially available Poly-MDI was tested in a 2-year inhalation study in rats. The tested material contained 47% aromatic 4,4'-methylenebisphenyl diisocyanate (MDI) and 53% higher molecular weight oligomers. Interim sacrifices at one year showed that males and females in the highest dose group (6 mg/m³) had treatment related histological changes in the nasal cavity, lungs and mediastinal lymph nodes. The incidence and severity of degeneration and basal cell hyperplasia of the olfactory epithelium and Bowman's gland hyperplasia were increased in males at the mid and high doses and in females at the high dose following the two year exposure period. Pulmonary adenomas were found in 6 males and 2 females, and pulmonary adenocarcinoma in one male in the high dose group. However, aliphatic hexamethylene diisocyanate (HDI) was found not to be carcinogenic in a two year repeated dose study in rats by the inhalation route. HDI has not been tested in mice by the inhalation route.</p> <p>Though the oral route is not an expected route of exposure to humans, it should be noted that in two year repeated dose studies by the oral route, aromatic toluene diisocyanate (TDI) and 3,3'-dimethoxy-benzidine-4,4'-diisocyanate (dianisidine diisocyanate, DADI) were found to be carcinogenic in rodents. TDI induced a statistically significant increase in the incidence of liver tumors in rats and mice as well as dose-related hemangiosarcomas of the circulatory system and has been classified by the Agency as a B2 carcinogen. DADI was found to be carcinogenic in rats, but not in mice, with a statistically increase in the incidence of pancreatic tumors observed.</p> <p>Respiratory and Dermal Sensitization: Based on the available toxicity data in animals and epidemiologic studies of humans, aromatic diisocyanates such as TDI and MDI are strong respiratory sensitisers. Aliphatic diisocyanates are generally not active in animal models for respiratory sensitization. However, HDI and possibly isophorone diisocyanate (IPDI), are reported to be associated with respiratory sensitization in humans. Symptoms resulting from occupational exposure to HDI include shortness of breath, increased bronchoconstriction reaction to histamine challenges, asthmatic reactions, wheezing and coughing. Two case reports of human exposure to IPDI by inhalation suggest IPDI is a respiratory sensitiser in humans. In view of the information from case reports in humans, it would be prudent at this time to assume that both aromatic and aliphatic diisocyanates are respiratory sensitisers. Studies in both human and mice using TDI, HDI, MDI and dicyclohexylmethane-4,4'-diisocyanate (HMDI) suggest cross-reactivity with the other diisocyanates, irrespective of whether the challenge compound was an aliphatic or aromatic diisocyanate. Diisocyanates are moderate to strong dermal sensitisers in animal studies. There seems to be little or no difference in the level of reactivity between aromatic and aliphatic diisocyanates.</p> <p>Dermal Irritation: Skin irritation studies performed on rabbits and guinea pigs indicate no difference in the effects of aromatic versus aliphatic diisocyanates. The level of irritation ranged from slightly to severely irritating to the skin. One chemical, hydrogenated MDI (1,1-methylenebis-4-isocyanatocyclohexane), was found to be corrosive to the skin in guinea pigs.</p>
1,3,5-TRIMETHYL BENZENE	CHEMWATCH 12171 1,2,4-trimethylbenzene
CARBON BLACK	Inhalation (rat) TCLo: 50 mg/m ³ /6h/90D-I Nil reported
SILICA AMORPHOUS	<p>Reports indicate high/prolonged exposures to amorphous silicas induced lung fibrosis in experimental animals; in some experiments these effects were reversible. [PATTYS]</p> <p>For silica amorphous:</p> <p>Derived No Adverse Effects Level (NOAEL) in the range of 1000 mg/kg/d.</p> <p>In humans, synthetic amorphous silica (SAS) is essentially non-toxic by mouth, skin or eyes, and by inhalation. Epidemiology studies show little evidence of adverse health effects due to SAS. Repeated exposure (without personal protection) may cause mechanical irritation of the eye and drying/cracking of the skin.</p> <p>When experimental animals inhale synthetic amorphous silica (SAS) dust, it dissolves in the lung fluid and is rapidly eliminated. If swallowed, the vast majority of SAS is excreted in the faeces and there is little accumulation in the body. Following absorption across the gut, SAS is eliminated via urine without modification in animals and humans. SAS is not expected to be broken down (metabolised) in mammals.</p> <p>After ingestion, there is limited accumulation of SAS in body tissues and rapid elimination occurs. Intestinal absorption has not been calculated, but appears to be insignificant in animals and humans. SASs injected subcutaneously are subjected to rapid dissolution and removal. There is no indication of metabolism of SAS in animals or humans based on chemical structure and available data. In contrast to crystalline silica, SAS is soluble in physiological media and the soluble chemical species that are formed are eliminated via the urinary tract without modification.</p> <p>Both the mammalian and environmental toxicology of SASs are significantly influenced by the physical and chemical properties, particularly those of solubility and particle size. SAS has no acute intrinsic toxicity by inhalation. Adverse effects, including suffocation, that have been reported were caused by the presence of high numbers of respirable particles generated to meet the required test atmosphere. These results are not representative of exposure to commercial SASs and should not be used for human risk assessment. Though repeated exposure of the skin may cause dryness and cracking, SAS is not a skin or eye irritant, and it is not a sensitiser.</p> <p>Repeated-dose and chronic toxicity studies confirm the absence of toxicity when SAS is swallowed or upon skin contact.</p> <p>Long-term inhalation of SAS caused some adverse effects in animals (increases in lung inflammation, cell injury and lung collagen content), all of which subsided after exposure.</p> <p>Numerous repeated-dose, subchronic and chronic inhalation toxicity studies have been conducted with SAS in a number of species, at airborne concentrations ranging from 0.5 mg/m³ to 150 mg/m³. Lowest-observed adverse effect levels (LOAELs) were typically in the range of 1 to 50 mg/m³. When available, the no-observed adverse effect levels (NOAELs) were between 0.5 and 10 mg/m³. The difference in values may be explained by different particle size, and therefore the number of particles administered per unit dose. In general, as particle size decreases so does the NOAEL/LOAEL.</p> <p>Neither inhalation nor oral administration caused neoplasms (tumours). SAS is not mutagenic in vitro. No genotoxicity was detected in in vivo assays. SAS does not impair development of the foetus. Fertility was not specifically studied, but the reproductive organs in long-term studies were not affected.</p> <p>For Synthetic Amorphous Silica (SAS)</p> <p>Repeated dose toxicity</p> <p>Oral (rat), 2 weeks to 6 months, no significant treatment-related adverse effects at doses of up to 8% silica in the diet.</p> <p>Inhalation (rat), 13 weeks, Lowest Observed Effect Level (LOEL) = 1.3 mg/m³ based on mild reversible effects in the lungs. Inhalation (rat), 90 days, LOEL = 1 mg/m³ based on reversible effects in the lungs and effects in the nasal cavity.</p> <p>For silane treated synthetic amorphous silica:</p> <p>Repeated dose toxicity: oral (rat), 28-d, diet, no significant treatment-related adverse effects at the doses tested.</p> <p>There is no evidence of cancer or other long-term respiratory health effects (for example, silicosis) in workers employed in the manufacture of SAS. Respiratory symptoms in SAS workers have been shown to correlate with smoking but not with SAS exposure, while serial pulmonary function values and chest radiographs are not adversely affected by long-term exposure to SAS.</p>
CUMENE	<p>Cumene is reasonably anticipated to be a human carcinogen based on sufficient evidence of carcinogenicity from studies in experimental animals. Cumene caused tumours at several tissue sites, including lung and liver in mice and kidney in male rats. Several proposed mechanisms of carcinogenesis support the relevance to humans of lung and liver tumours in experimental animals. Specifically, there is evidence that humans and experimental animals metabolise cumene through similar metabolic pathways. There is also evidence that cumene is genotoxic in some tissues, based on findings of DNA damage in rodent lung and liver. Furthermore, mutations of the K-ras oncogene and p53 tumor-suppressor gene observed in cumene-induced lung tumours in mice, along with altered expression of many other genes, resemble molecular alterations found in human lung and other cancers. The relevance of the kidney tumors to cancer in humans is uncertain; there is evidence that a species-specific mechanism not relevant to humans contributes to their induction, but it is possible that other mechanisms relevant to humans, such as genotoxicity, may also contribute to kidney-tumour formation in male rats.</p> <p>For aromatic terpenes:</p> <p>Acute toxicity: Mammalian LD50 for p-cymene have shown it to have low toxic potential. Similar studies with cumene have concurred with</p>

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these results

In general, the studies indicate that p-cymene (p-methylisopropylbenzene) or cumene (isopropylbenzene) is rapidly absorbed by oral or inhalation routes. They undergo oxidation (hydroxylation) of the side chain isopropyl substituent and, in the case of p-cymene, the methyl substituent to yield polar oxygenated metabolites. These metabolites are either excreted unchanged in the urine or undergo Phase II conjugation with glucuronic acid and/or glycine followed by excretion in the urine. Unchanged p-cymene or cumene were not detected in the urine or faeces.

Humans (5 males and 5 females/group) exposed to an atmosphere containing 49, 98, or 147 ppm cumene for 7 hours showed 64% absorption at 0.5 hours and 45% at 7 hours. Maximum excretion is observed at 6 to 8 hours and is essentially complete at 48 hours. Approximately 35% of the dose inhaled was excreted as 2-phenyl-2-propanol

Repeat Dose Toxicity: Subacute Studies: Groups of 7 to 12 male rats were exposed to 0, 50, or 250 ppm of p-cymene for 6 hours/day, 5 days/week for 4 weeks with an 8-week recovery period. there was no overt toxicity in the treated rats and no effect on body weight or terminal weight of the brain, cerebellum or whole brain. There was also no effect on regional enzyme activities, regional protein synthesis or regional neurotransmitter concentrations.

Cumene has been tested by the National Toxicology Program (NTP) in both rats and mice. Animals were exposed to up to 4,000 ppm cumene by whole-body inhalation for 12-13 days over a period of 16-17 days. In rats, all animals died at 4,000 ppm, and about half the animals died at the next exposure concentration (2,000 ppm). Varying degrees of ataxia were reported in surviving rats exposed to 500 to 2,000 ppm cumene. Increased relative liver and kidney weights were reported in rats exposed to cumene. In exposed male rats, hyaline droplets in the renal cortical tubules were reported. At 2,000 ppm, suppurative inflammation of the lung was reported in 40% of the rats. In mice, all animals died at the 2 highest exposures (2,000 and 4,000 ppm). At 1,000 ppm, 80% of the female mice died and male mice showed varying degrees of ataxia. Increased relative liver and kidney weights were reported in mice exposed to cumene. Decreased thymus weight was reported in male mice exposed to 1,000 ppm of cumene. No histopathological findings accompanied the organ weight changes. A NOAEL of 1,000 ppm was determined for female rats and male mice and a NOAEL of 500 ppm was determined for female mice based on mortality and histopathological findings.

Chronic toxicity: The US EPA concluded that there is some evidence that suggests that cumene is not likely to produce a carcinogenic response (i.e., numerous genotoxic tests, including gene mutation, chromosomal aberration, and primary DNA damage tests, all but one of which were negative or not reproducible) In addition, EPA noted that cumene does not appear to metabolise to highly reactive chemical species and in terms of metabolism, cumene is analogous to methyl benzene for which a 2-year inhalation study was conducted by NTP and no evidence of carcinogenic activity was reported in either rats or mice.

Given that the only structural difference between p-cymene and cumene is the presence of a second alkyl substituent (isopropylbenzene versus p-methylisopropylbenzene), similar conclusions can be drawn for p-cymene, particularly since the pharmacokinetic, metabolic and toxicologic data that are available support this conclusion.

Reproductive toxicity: Taking into consideration the rapid metabolism and excretion of cumene, the US EPA concluded, "cumene has low potential for reproductive toxicity."

Developmental toxicity: Even at maternally toxic concentrations exposure to cumene vapor did not produce developmental toxicity in rats.

However the US EPA determined that the changes in gestational parameters of the rabbits, though not significant, were consistent in indicating possible developmental effects and therefore set the NOAEL in rabbits for both developmental and maternal effects at 1,206 ppm and the LOAEL at 2,297 ppm, respectively (as reported in EPA, 1997). Since both cumene and p-cymene exhibit such similar pharmacokinetic and metabolic profiles, and show no evidence of toxicity at levels of exposure similar to those experienced by humans, further teratogenic or developmental testing is not recommended

Genotoxicity: The genotoxicity database on p-cymene and cumene shows no mutagenic potential in the Ames assay. In cytogenetic assays, there is no evidence of a genotoxic potential in vitro. In whole animals, the genotoxicity results for cumene are mixed showing weakly positive results in micronuclei induction in rats, but no evidence of genotoxicity in mice.

DIISOBUTYL KETONE

[Eastman; * for mixed isomer, ** for 2,6-dimethyl-4-heptanone] NOEL = 400 ppm (12 exposures rat) * LOEL = 250 ppm (30 exposures, rat) ** NOEL = 125 ppm (" " ") ** - target organ; kidney LOEL = 2000 mg/kg/day (oral neurotoxicity; minor target organs - liver, kidney, stomach) ** NOEL = 2000 mg/kg (for neurotoxicity) ** Skin sensitisation (g.pig) - moderate *

For diisobutyl ketone (DIBK)

There is very little data on DIBK exposure available. For the risk characterisation a selection of the data on methyl isobutyl ketone (MIBK) and methyl ethyl ketone, (MEK) was used. MEK and MIBK were selected because they are comparable to DIBK in effects and use.

There is no specific data on the metabolism of diisobutyl ketone (DIBK) however it is expected to undergo the metabolic change typical of many ketones, that is reduction to the corresponding secondary alcohol and elimination as a glucuronic acid conjugate. Data available for the related ketone methyl isobutyl ketone (MIBK) indicate that it is metabolised to the corresponding secondary alcohol 4-methyl-2-pentanol and 4-hydroxy-4-methyl-2-pentanone (major metabolite). The structure of MIBK and DIBK precludes metabolism to the neurotoxic metabolite 2,5-hexanedione formed from both hexane and methyl n-butyl ketone.

From the available data it is concluded that DIBK is of low acute toxicity following oral, dermal and inhalational exposure. Signs of intoxication include irritation of the eyes and nose, salivation, lethargy, instability, respiratory difficulty, unsteady gait and narcosis.

Following dermal administration slight skin irritation has been observed. Gross pathological examination of animals exposed orally or dermally to 2000 mg/kg or inhalationally to 5 mg/l DIBK (non-lethal doses) showed no treatment related findings

Exposure to near saturated vapours (7.5 to 16 hours) induced only minor histopathological changes in the lung, kidney, liver, spleen and adrenals. Autopsies following administration of oral doses revealed congested and haemorrhagic lungs, mottled liver, pale kidneys and some damage to the intestinal tract.

1-DECENE HOMOPOLYMER, HYDROGENATED

(estimated) * Evidence of conjunctival changes ** No evidence of tissue damage [Inland Vacuum Industries] ^ US EPA HPV Challenge program October 2002

P-TOLUENESULFONYL ISOCYANATE

for p-toluenesulfonyl isocyanate

The acute oral toxicity (LD50) of PTSI is 2600 mg/kg. Based on the rapid hydrolysis of PTSI to PTSA (and carbon dioxide), repeated dose, reproductive, and developmental toxicity, as well as genotoxicity are best described by PTSA.

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for p-toluenesulfonamide (PTSA):

PTSA was studied for oral toxicity in rats in a single dose toxicity test at doses of 889, 1333, 2000 and 3000 mg/kg in females and 2000 mg/kg in males, and in an OECD combined repeat dose and reproductive/developmental toxicity screening test at doses of 0, 120, 300 and 750 mg/kg/day in both sexes. PTSA was also tested for mutagenicity with assays for reverse mutation in bacteria and chromosomal aberrations in cultured Chinese hamster (CHL) cells. The single dose toxicity test revealed LD50 values of above 2000 mg/kg for both sexes.

For repeat dose toxicity caused, daily administration of 300 mg/kg or more in males and females displayed an increase in salivation and a reduction in body weight gain, as well as a suppression of food consumption. No compound-related deaths were observed. Haematuria was observed within 3 days administration of 750 mg/kg in 4/13 males. Hematological examination and blood chemistry measurements in males showed a decrease in white blood cell count with an increase in lymphocyte count, increases in blood urea nitrogen and chloride, and slight elevation in GOT in medium and high dose groups and a decrease in potassium concentration, and increased GPT levels in the high dose group. Histopathological examination showed cytoplasmic changes in the epithelium of the urinary bladder in both sexes and an accelerated involution in the thymus especially in females. Signs of toxicity, such as salivation and urinary bladder changes, were observed in animals given 120 mg/kg and above. The NOEL for repeat dose toxicity was less than 120 mg/kg/day. For reproductive/developmental toxicity, females given 750 mg/kg/day demonstrated possible delivery or lactation state dysfunction and developmental suppression of embryos. NOELs for reproductive performance and offspring development were both 300 mg/kg/day. No teratogenic effects were observed.

The mutagenicity tests performed were all negative. PTSA was not mutagenic for bacteria either with or without an exogenous metabolic activation system up to 5000 ug/plate. No chromosomal aberrations or polyploidy were induced in CHL cells up to 1.7 mg/ml with metabolic activation and 1.3 mg/ml without metabolic activation.

DURAM ROCKTUFF (BLACK, DARK GREY, LIGHT GREY, YELLOW)

<p>C7-9 BRANCHED ALKYL-3,5-DI-TERT-BUTYL-4-HYDROXYHYDROCINNAMATE</p>	<p>Non-sensitising to guinea pig skin * Everspring Chemical MSDS For hindered phenols: Available data shows that acute toxicity of these substances is low. Mutagenicity. Data from bacterial reverse mutation assays and <i>in vitro</i> and <i>in vivo</i> 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</p>
<p>DIBUTYL TIN DILAURYL MERCAPTIDE</p>	<p>* Arkema Canada MSDS for substance in 40% mineral oil ** Geleste MSDS</p>
<p>NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT & 1,2,4-TRIMETHYL BENZENE & TITANIUM DIOXIDE & MDI HOMOPOLYMER & 4,4'-DIPHENYLMETHANE DISOCYANATE (MDI) & 1,3,5-TRIMETHYL BENZENE & CUMENE & DIISOBUTYL KETONE & P-TOLUENESULFONYL ISOCYANATE</p>	<p>Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.</p>
<p>NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT & 1,2,4-TRIMETHYL BENZENE & 1,3,5-TRIMETHYL BENZENE</p>	<p>For trimethylbenzenes: Absorption of 1,2,4-trimethylbenzene occurs after oral, inhalation, or dermal exposure. Occupationally, inhalation and dermal exposures are the most important routes of absorption although systemic intoxication from dermal absorption is not likely to occur due to the dermal irritation caused by the chemical prompting quick removal. Following oral administration of the chemical to rats, 62.6% of the dose was recovered as urinary metabolites indicating substantial absorption . 1,2,4-Trimethylbenzene is lipophilic and may accumulate in fat and fatty tissues. In the blood stream, approximately 85% of the chemical is bound to red blood cells Metabolism occurs by side-chain oxidation to form alcohols and carboxylic acids which are then conjugated with glucuronic acid, glycine, or sulfates for urinary excretion . After a single oral dose to rats of 1200 mg/kg, urinary metabolites consisted of approximately 43.2% glycine, 6.6% glucuronic, and 12.9% sulfuric acid conjugates . The two principle metabolites excreted by rabbits after oral administration of 438 mg/kg/day for 5 days were 2,4-dimethylbenzoic acid and 3,4-dimethylhippuric acid . The major routes of excretion of 1,2,4-trimethyl- benzene are exhalation of parent compound and elimination of urinary metabolites. Half-times for urinary metabolites were reported as 9.5 hours for glycine, 22.9 hours for glucuronide, and 37.6 hours for sulfuric acid conjugates. Acute Toxicity Direct contact with liquid 1,2,4-trimethylbenzene is irritating to the skin and breathing the vapor is irritating to the respiratory tract causing pneumonitis. Breathing high concentrations of the chemical vapor causes headache, fatigue, and drowsiness. In humans liquid 1,2,4-trimethylbenzene is irritating to the skin and inhalation of vapor causes chemical pneumonitis . High concentrations of vapor (5000-9000 ppm) cause headache, fatigue, and drowsiness . The concentration of 5000 ppm is roughly equivalent to a total of 221 mg/kg assuming a 30 minute exposure period (see end note 1). 2. Animals - Mice exposed to 8130-9140 ppm 1,2,4-trimethylbenzene (no duration given) had loss of righting response and loss of reflexes Direct dermal contact with the chemical (no species given) causes vasodilation, erythema, and irritation (U.S. EPA). Seven of 10 rats died after an oral dose of 2.5 mL of a mixture of trimethylbenzenes in olive oil (average dose approximately 4.4 g/kg) . Rats and mice were exposed by inhalation to a coal tar distillate containing about 70% 1,3,5- and 1,2,4-trimethylbenzene; no pathological changes were noted in either species after exposure to 1800-2000 ppm for up to 48 continuous hours, or in rats after 14 exposures of 8 hours each at the same exposure levels . No effects were reported for rats exposed to a mixture of trimethyl- benzenes at 1700 ppm for 10 to 21 days Neurotoxicity 1,2,4-Trimethylbenzene depresses the central nervous system. Exposure to solvent mixtures containing the chemical causes headache, fatigue, nervousness, and drowsiness. Occupationally, workers exposed to a solvent containing 50% 1,2,4-trimethylbenzene had nervousness, headaches, drowsiness, and vertigo (U.S. EPA). Headache, fatigue, and drowsiness were reported for workers exposed (no dose given) to paint thinner containing 80% 1,2,4- and 1,3,5-trimethylbenzenes Results of the developmental toxicity study indicate that the C9 fraction caused adverse neurological effects at the highest dose (1500 ppm) tested. Subchronic/Chronic Toxicity Long-term exposure to solvents containing 1,2,4-trimethylbenzene may cause nervousness, tension, and bronchitis. Painters that worked for several years with a solvent containing 50% 1,2,4- and 30% 1,3,5-trimethylbenzene showed nervousness, tension and anxiety, asthmatic bronchitis, anemia, and alterations in blood clotting; haematological effects may have been due to trace amounts of benzene Rats given 1,2,4-trimethylbenzene orally at doses of 0.5 or 2.0 g/kg/day, 5 days/week for 4 weeks. All rats exposed to the high dose died and 1 rat in the low dose died (no times given); no other effects were reported. Rats exposed by inhalation to 1700 ppm of a trimethylbenzene isomeric mixture for 4 months had decreased weight gain, lymphopenia and neutrophilia . Genotoxicity: Results of mutagenicity testing, indicate that the C9 fraction does not induce gene mutations in prokaryotes (Salmonella typhimurium/mammalian microsome assay); or in mammalian cells in culture (in Chinese hamster ovary cells with and without activation). The C9 fraction does not does not induce chromosome mutations in Chinese hamster ovary cells with and without activation; does not induce chromosome aberrations in the bone marrow of Sprague-Dawley rats exposed by inhalation (6 hours/day for 5 days); and does not induce sister chromatid exchange in Chinese hamster ovary cells with and without activation. Developmental/Reproductive Toxicity: A three-generation reproductive study on the C9 fraction was conducted CD rats (30/sex/group) were exposed by inhalation to the C9 fraction at concentrations of 0, 100, 500, or 1500 ppm (0, 100, 500, or 1500 mg/kg/day) for 6 hours/day, 5 days/week. There was evidence of parental and reproductive toxicity at all dose levels. Indicators of parental toxicity included reduced body weights, increased salivation, hunched posture, aggressive behavior, and death. Indicators of adverse reproductive system effects included reduced litter size and reduced pup body weight. The LOEL was 100 ppm; a no-observed-effect level was not established Developmental toxicity, including possible develop- mental neurotoxicity, was evident in rats in a 3-generation reproductive study No effects on fecundity or fertility occurred in rats treated dermally with up to 0.3 mL/rat/day of a mixture of trimethyl- benzenes, 4-6 hours/day, 5 days/week over one generation</p>
<p>POLYETHYLENE & 1-DECENE HOMOPOLYMER, HYDROGENATED</p>	<p>for poly-alpha-olefins (PAOs): PAOs are highly branched isoparaffinic chemicals produced by oligomerisation of 1-octene, 1-decene, and/or 1-dodecene. The crude polyalphaolefin mixture is then distilled into appropriate product fractions to meet specific viscosity specifications and hydrogenated. Read across data exist for health effects endpoints from the following similar <i>hydrogenated</i> long chain branched alkanes derived from a C8, C10, and/or C12 alpha olefins:</p>

DURAM ROCKTUFF (BLACK, DARK GREY, LIGHT GREY, YELLOW)

- ▶ Decene homopolymer
- ▶ Decene/dodecene copolymer
- ▶ Octene/decene/dodecene copolymer
- ▶ Dodecene trimer

The data for these structural analogs demonstrated no evidence of health effects. In addition, there is evidence in the literature that alkanes with 30 or more carbon atoms are unlikely to be absorbed when administered orally. The physicochemical data suggest that it is unlikely that significant absorption will occur. If a substance of the size and structure of a typical PAO is absorbed, then the principal mechanisms of absorption after oral administration are likely to be passive diffusion and absorption by way of the lymphatic system. The former requires both good lipid solubility and good water solubility as the substance has to partition from an aqueous environment through a lipophilic membrane into another aqueous environment during absorption. Absorption by way of the lymphatics occurs by mechanisms analogous to those that absorb fatty acids and is limited by the size of the molecule. Lipophilicity generally enhances the ability of chemicals to cross biological membranes. Biotransformation by mixed function oxidases often increases the water solubility of a substance; however, existing data suggest that these substances will not undergo oxidation to more hydrophilic metabolites. Finally, a chemical must have an active functional group that can interact chemically or physically with the target cell or receptor upon reaching it; there are no moieties in PAOs that represent a functional group that may have biological activity. The water solubilities of a C10 dimer PAO and a C12 trimer PAO were determined to be <1 ppb and <1 ppt respectively. The partition coefficient for a C12 trimer PAO was determined to be log Kow of >7. Given the very low water solubility it is extremely unlikely that PAOs will be absorbed by passive diffusion following oral administration, and the size of the molecules suggest that the extent of lymphatic absorption is likely to be very low. Although PAOs are relatively large lipophilic compounds, and molecular size may be a critical limiting determinant for absorption, there is some evidence that these substances are absorbed. However, the lack of observed toxicity in the studies with PAOs suggests that these products are absorbed poorly, if at all. Furthermore, a review of the literature regarding the absorption and metabolism of long chain alkanes indicates that alkanes with 30+ carbon atoms are unlikely to be absorbed. For example the absorption of squalane, an analogous C30 product, administered orally to male CD rats was examined - essentially all of the squalane was recovered unchanged in the faeces. At the same time, the hydrophobic properties of PAOs suggest that, should they be absorbed, they would undergo limited distribution in the aqueous systemic circulation and reach potential target organs in limited concentrations.

In addition to the general considerations discussed above, the low volatility of PAOs indicates that, under normal conditions of use or transportation, exposure by the inhalation route is unlikely. In particular, the high viscosity of these substances suggests that it would be difficult to generate a high concentration of respirable particles in the air.

Acute toxicity: PAOs (decene/dodecene copolymer, octene/decene/dodecene homo-polymer, and dodecene trimer) have been adequately tested for acute oral toxicity. There were no deaths when the test materials were administered at doses of 5,000 mg/kg (decene/dodecene copolymer and dodecene trimer) and at 2,000 mg/kg (octene/decene/dodecene copolymer) in rats. Overall, the acute oral LD50 for these substances was greater than the 2000 mg/kg limit dose, indicating a relatively low order of toxicity.

PAOs (decene/dodecene copolymer, octene/decene/dodecene copolymer, and dodecene trimer) have been tested for acute dermal toxicity. No mortality was observed for any substance when administered at the limit dose of 2000 or 5000 mg/kg. Overall, the acute dermal LD50 for these substances was greater than the 2000 mg/kg limit dose, indicating a relatively low order of toxicity.

1-Decene, homopolymer, is absorbed (unexpectedly for a high molecular weight polymer) to a moderate degree in rat skin and is eliminated slowly

PAOs (decene homopolymer, decene/dodecene copolymer, and decene trimer) have been tested for acute inhalation toxicity. Rats were exposed to aerosols of the substances at nominal atmospheric concentrations of 2.5, 5.0, and 5.06 mg/L, respectively, for four hours. These levels were the maximum attainable concentrations under the conditions of the tests, due to the low volatility and high viscosity of the test material. No mortality was noted, and all animals fully recovered following depuration. The lack of mortality at concentrations at or above the limit dose of 2.0 mg/L indicates a relatively low order of toxicity for these substances.

Repeat dose toxicity: Eight repeated-dose toxicity studies using two different animal species, rats and mice, and oral and dermal routes of administration have been conducted with three structural analogs. These data suggest that the structural analogs exhibit a low order of toxicity following repeated applications, due to their similarity in chemical structures and physicochemical properties.

One 28-day oral toxicity study in rats, one 90-day dermal and two 90-day dietary studies in rats, and a dermal carcinogenicity study in mice exist for decene homopolymer. A rat oral combined reproductive toxicity and 91-day systemic toxicity study was also conducted with decene homopolymer. In addition, 28-day rat oral toxicity studies exist for two structurally analogous substances (dodecene trimer and octene/decene/dodecene copolymer); and a 90-day rat dermal toxicity study exists for octene/decene/dodecene copolymer. Results from these studies show a low order of repeated dose toxicity. The dermal NOAEL for systemic toxicity studies was equal to or greater than 2000 mg/kg/day. The oral NOAEL for 1-decene homopolymer is between 5,000 and 20,000 mg/kg/day in Sprague-Dawley rats.

Rats exposed repeatedly by dermal exposure at doses of 2000 mg/kg decene/dodecene copolymer showed increased incidences of hyperplasia of the sebaceous glands, hyperplasia/hyperkeratosis of the epidermis and dermal inflammation. These symptoms generally subsided within 2 weeks. Males showed decreased body weight gain and altered serum chemistry.

In a 90-day feeding study rats receiving 20000 ppm of 1-decene, homopolymer, hydrogenated did not exhibit any clinical signs of systemic toxicity. Marginal effects on clinical chemistry (glucose and ALT in males; sodium, phosphorus and calcium in females) were seen.

Reproductive toxicity: Data are available for decene homopolymer. Results from these studies show a low order of reproductive/developmental toxicity. The NOAEL for reproductive toxicity was 1000 mg/kg/day, the highest concentration tested. The lack of effects on fertility in this study or effects on reproductive organs in this or other subchronic studies with closely related chemicals indicates that PAOs are unlikely to exert effects on reproduction.

Developmental toxicity: Decene homopolymer (with 10 ppm of an antioxidant) was administered once daily on gestation days 0-19 via dermal application to presumed-pregnant rats at doses of 0, 800, and 2000 mg/kg/day. Dermal administration of the test material did not adversely affect parameters of reproductive performance during gestation, nor did it adversely affect *in utero* survival and development of the offspring. The NOAEL in this study for developmental parameters was 2000 mg/kg/day.

Genotoxicity: Information for the following PAOs (decene homopolymer, octene/decene/dodecene copolymer, dodecene trimer; and decene/dodecene copolymer [prepared from 10% C12 and 90% C10 alpha olefins; approx. 33% trimer and 51% tetramer, 16% pentamer and higher]) is available. Either bacterial or mammalian gene mutation assays, *in vitro* chromosomal aberration assays, or *in vivo* chromosomal aberration assays have been conducted for these substances. Neither mutagenicity nor clastogenicity were exhibited by any of these substances in the referenced *in vivo* or *in vitro* tests, with or without metabolic activation.

Carcinogenicity: While alpha-olefin polymers have similar properties to mineral oils, they do not contain polycyclic aromatic hydrocarbons, or other known possible carcinogens.

Decene homopolymer produced no treatment-related tumors in C3H mice treated with a 50 ul/application twice weekly for 104 weeks. In addition, survival (56%) was greater than in any other group, including the untreated control.

POLYETHYLENE & MDI
HOMOPOLYMER &
4,4'-DIPHENYLMETHANE
DIISOCYANATE (MDI) & SILICA
AMORPHOUS

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.

1,2,4-TRIMETHYL BENZENE &
1,3,5-TRIMETHYL BENZENE

Other Toxicity data is available for CHEMWATCH 12172 1,2,3-trimethylbenzene

TITANIUM DIOXIDE & CARBON
BLACK & METHYL 1,2,2,6,6-
PENTAMETHYL-4-PIPERIDYL
SEBACATE

No significant acute toxicological data identified in literature search.

DURAM ROCKTUFF (BLACK, DARK GREY, LIGHT GREY, YELLOW)

TITANIUM DIOXIDE & 4,4'-DIPHENYLMETHANE DIISOCYANATE (MDI)	The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
TITANIUM DIOXIDE & DI-SEC-OCTYL PHTHALATE & DIISOBUTYL KETONE	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
TITANIUM DIOXIDE & DI-SEC-OCTYL PHTHALATE & CARBON BLACK & CUMENE	WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
DI-SEC-OCTYL PHTHALATE & 1,3,5-TRIMETHYL BENZENE & DIISOBUTYL KETONE	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
DI-SEC-OCTYL PHTHALATE & CUMENE	Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [National Toxicology Program: U.S. Dep. of Health & Human Services 2002]
MDI HOMOPOLYMER & 4,4'-DIPHENYLMETHANE DIISOCYANATE (MDI) & BIS(1,2,2,6,6-PENTAMETHYL-4-PIPERIDYL)SEBACATE & METHYL 1,2,2,6,6-PENTAMETHYL-4-PIPERIDYL SEBACATE	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.
MDI HOMOPOLYMER & 4,4'-DIPHENYLMETHANE DIISOCYANATE (MDI) & P-TOLUENESULFONYL ISOCYANATE	Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens). Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis. Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure. Isocyanate vapours/mists are irritating to the upper respiratory tract and lungs; the response may be severe enough to produce bronchitis with wheezing, gasping and severe distress, even sudden loss of consciousness, and pulmonary oedema. Possible neurological symptoms arising from isocyanate exposure include headache, insomnia, euphoria, ataxia, anxiety neurosis, depression and paranoia. Gastrointestinal disturbances are characterised by nausea and vomiting. Pulmonary sensitisation may produce asthmatic reactions ranging from minor breathing difficulties to severe allergic attacks; this may occur following a single acute exposure or may develop without warning after a period of tolerance. A respiratory response may occur following minor skin contact. Skin sensitisation is possible and may result in allergic dermatitis responses including rash, itching, hives and swelling of extremities. Isocyanate-containing vapours/ mists may cause inflammation of eyes and nasal passages. Onset of symptoms may be immediate or delayed for several hours after exposure. Sensitised people can react to very low levels of airborne isocyanates. Unprotected or sensitised persons should not be allowed to work in situations allowing exposure to this material.
1,3,5-TRIMETHYL BENZENE & CUMENE	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

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

DURAM ROCKTUFF (BLACK, DARK GREY, LIGHT GREY, YELLOW)	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
naphtha petroleum, light aromatic solvent	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	72h	Algae or other aquatic plants	1mg/l	1
	EC50	72h	Algae or other aquatic plants	19mg/l	1
	EC50	48h	Crustacea	6.14mg/l	1
	EC50	96h	Algae or other aquatic plants	64mg/l	2
polyethylene	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available

DURAM ROCKTUFF (BLACK, DARK GREY, LIGHT GREY, YELLOW)

1,2,4-trimethyl benzene	Endpoint	Test Duration (hr)	Species	Value	Source
	BCF	1344h	Fish	31-207	7
	EC50(ECx)	96h	Algae or other aquatic plants	2.356mg/l	2
	LC50	96h	Fish	3.41mg/l	2
	EC50	96h	Algae or other aquatic plants	2.356mg/l	2
	EC50	48h	Crustacea	ca.6.14mg/l	1
titanium dioxide	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	3.75-7.58mg/l	4
	BCF	1008h	Fish	<1.1-9.6	7
	EC50	48h	Crustacea	1.9mg/l	2
	LC50	96h	Fish	1.85-3.06mg/l	4
	NOEC(ECx)	504h	Crustacea	0.02mg/l	4
	EC50	96h	Algae or other aquatic plants	179.05mg/l	2
di-sec-octyl phthalate	Endpoint	Test Duration (hr)	Species	Value	Source
	ErC50	72h	Algae or other aquatic plants	>130mg/l	1
	EC50	72h	Algae or other aquatic plants	>130mg/l	1
	EC50	48h	Crustacea	>0.16mg/l	1
	LC50	96h	Fish	>0.16mg/l	2
	BCF	1344h	Fish	<0.7-29.7	7
	NOEC(ECx)	1680h	Fish	0.007mg/l	1
	EC50	96h	Algae or other aquatic plants	>0.1mg/l	1
MDI homopolymer	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>1640mg/l	2
	LC50	96h	Fish	>1000mg/l	2
	NOEC(ECx)	504h	Crustacea	>=10mg/l	2
4,4'-diphenylmethane diisocyanate (MDI)	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>1640mg/l	2
	LC50	96h	Fish	>1000mg/l	2
	NOEC(ECx)	504h	Crustacea	>=10mg/l	2
	BCF	672h	Fish	61-150	7
C.I. Pigment Yellow 74	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	504h	Crustacea	>1mg/l	2
1,3,5-trimethyl benzene	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	5.216mg/l	2
	EC50	48h	Crustacea	13mg/L	5
	BCF	1680h	Fish	23-342	7
	NOEC(ECx)	384h	Crustacea	0.257mg/l	2
	EC50	96h	Algae or other aquatic plants	3.084mg/l	2
carbon black	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>0.2mg/l	2
	LC50	96h	Fish	>100mg/l	2
	EC50	48h	Crustacea	33.076-41.968mg/l	4
	NOEC(ECx)	24h	Crustacea	3200mg/l	1
silica amorphous	Endpoint	Test Duration (hr)	Species	Value	Source
	EC0(ECx)	24h	Crustacea	>=10000mg/l	1
	EC50	72h	Algae or other aquatic plants	14.1mg/l	2
	LC50	96h	Fish	1033.016mg/l	2
	EC50	48h	Crustacea	>86mg/l	2
	EC50	96h	Algae or other aquatic plants	217.576mg/l	2
cumene	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	96h	Crustacea	0.4mg/l	1
	EC50	72h	Algae or other aquatic plants	1.29mg/l	2

DURAM ROCKTUFF (BLACK, DARK GREY, LIGHT GREY, YELLOW)

	LC50	96h	Fish	2.7mg/l	2
	EC50	48h	Crustacea	4mg/l	1
diisobutyl ketone	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	26.3mg/l	2
	LC50	96h	Fish	30mg/l	2
	EC50	48h	Crustacea	250mg/l	1
	NOEC(ECx)	96h	Algae or other aquatic plants	46mg/l	1
	EC50	96h	Algae or other aquatic plants	100mg/l	1
1-decene homopolymer, hydrogenated	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC0(ECx)	24h	Crustacea	<10mg/l	1
	LC50	96h	Fish	0.34mg/l	1
p-toluenesulfonyl isocyanate	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	72h	Algae or other aquatic plants	10mg/l	2
	EC50	72h	Algae or other aquatic plants	25mg/l	2
	LC50	96h	Fish	>45mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
C7-9 branched alkyl-3,5-di-tert-butyl-4-hydroxyhydrocinnamate	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	>0.001mg/l	2
	EC50	72h	Algae or other aquatic plants	<0.001mg/l	2
	EC50	48h	Crustacea	>0.008mg/l	2
	EC50(ECx)	72h	Algae or other aquatic plants	<0.001mg/l	2
dibutyltin dilauryl mercaptide	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>=1.6mg/l	2
	EC50	48h	Crustacea	0.023mg/l	2
	EC50(ECx)	48h	Crustacea	0.023mg/l	2
methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available

Legend: *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*

DO NOT discharge into sewer or waterways.

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
polyethylene	LOW	LOW
1,2,4-trimethyl benzene	LOW (Half-life = 56 days)	LOW (Half-life = 0.67 days)
titanium dioxide	HIGH	HIGH
di-sec-octyl phthalate	HIGH (Half-life = 389 days)	LOW (Half-life = 1.21 days)
4,4'-diphenylmethane diisocyanate (MDI)	LOW (Half-life = 1 days)	LOW (Half-life = 0.24 days)
C.I. Pigment Yellow 74	HIGH	HIGH
1,3,5-trimethyl benzene	HIGH	HIGH
silica amorphous	LOW	LOW
cumene	HIGH	HIGH
diisobutyl ketone	HIGH	HIGH
1-decene homopolymer, hydrogenated	LOW	LOW
p-toluenesulfonyl isocyanate	HIGH	HIGH
dibutyltin dilauryl mercaptide	HIGH	HIGH

Bioaccumulative potential

Continued...

DURAM ROCKTUFF (BLACK, DARK GREY, LIGHT GREY, YELLOW)

Ingredient	Bioaccumulation
polyethylene	LOW (LogKOW = 1.2658)
1,2,4-trimethyl benzene	LOW (BCF = 275)
titanium dioxide	LOW (BCF = 10)
di-sec-octyl phthalate	HIGH (BCF = 24500)
4,4'-diphenylmethane diisocyanate (MDI)	LOW (BCF = 15)
C.I. Pigment Yellow 74	LOW (LogKOW = 2.9756)
1,3,5-trimethyl benzene	LOW (BCF = 342)
silica amorphous	LOW (LogKOW = 0.5294)
cumene	LOW (BCF = 35.5)
diisobutyl ketone	LOW (LogKOW = 2.5646)
1-decene homopolymer, hydrogenated	HIGH (LogKOW = 5.116)
p-toluenesulfonyl isocyanate	LOW (LogKOW = 2.3424)
dibutyltin dilauryl mercaptide	LOW (LogKOW = 16.42)

Mobility in soil

Ingredient	Mobility
polyethylene	LOW (KOC = 14.3)
1,2,4-trimethyl benzene	LOW (KOC = 717.6)
titanium dioxide	LOW (KOC = 23.74)
di-sec-octyl phthalate	LOW (KOC = 165400)
4,4'-diphenylmethane diisocyanate (MDI)	LOW (KOC = 376200)
C.I. Pigment Yellow 74	LOW (KOC = 88.95)
1,3,5-trimethyl benzene	LOW (KOC = 703)
silica amorphous	LOW (KOC = 23.74)
cumene	LOW (KOC = 817.2)
diisobutyl ketone	LOW (KOC = 60.12)
1-decene homopolymer, hydrogenated	LOW (KOC = 1724)
p-toluenesulfonyl isocyanate	LOW (KOC = 882.1)
dibutyltin dilauryl mercaptide	LOW (KOC = 6187000000)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ Containers may still present a chemical hazard/ danger when empty. ▶ Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> ▶ 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. ▶ 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.
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SECTION 14 Transport information

Labels Required

	
Marine Pollutant	NO
HAZCHEM	*3Y

Land transport (ADG)

DURAM ROCKTUFF (BLACK, DARK GREY, LIGHT GREY, YELLOW)

UN number	1263	
UN proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)	
Transport hazard class(es)	Class	3
	Subrisk	Not Applicable
Packing group	III	
Environmental hazard	Not Applicable	
Special precautions for user	Special provisions	163 223 367
	Limited quantity	5 L

Air transport (ICAO-IATA / DGR)

UN number	1263	
UN proper shipping name	Paint (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base); Paint related material (including paint thinning or reducing compounds)	
Transport hazard class(es)	ICAO/IATA Class	3
	ICAO / IATA Subrisk	Not Applicable
	ERG Code	3L
Packing group	III	
Environmental hazard	Not Applicable	
Special precautions for user	Special provisions	A3 A72 A192
	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)

UN number	1263	
UN proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)	
Transport hazard class(es)	IMDG Class	3
	IMDG Subrisk	Not Applicable
Packing group	III	
Environmental hazard	Not Applicable	
Special precautions for user	EMS Number	F-E , S-E
	Special provisions	163 223 367 955
	Limited Quantities	5 L

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

Not Applicable

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

Product name	Group
naphtha petroleum, light aromatic solvent	Not Available
polyethylene	Not Available
1,2,4-trimethyl benzene	Not Available
titanium dioxide	Not Available
di-sec-octyl phthalate	Not Available
MDI homopolymer	Not Available
4,4'-diphenylmethane diisocyanate (MDI)	Not Available
C.I. Pigment Yellow 74	Not Available
1,3,5-trimethyl benzene	Not Available
carbon black	Not Available
silica amorphous	Not Available
cumene	Not Available

Continued...

DURAM ROCKTUFF (BLACK, DARK GREY, LIGHT GREY, YELLOW)

Product name	Group
diisobutyl ketone	Not Available
1-decene homopolymer, hydrogenated	Not Available
bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate	Not Available
p-toluenesulfonyl isocyanate	Not Available
C7-9 branched alkyl-3,5-di-tert-butyl-4-hydroxyhydrocinnamate	Not Available
dibutyltin dilauryl mercaptide	Not Available
methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
naphtha petroleum, light aromatic solvent	Not Available
polyethylene	Not Available
1,2,4-trimethyl benzene	Not Available
titanium dioxide	Not Available
di-sec-octyl phthalate	Not Available
MDI homopolymer	Not Available
4,4'-diphenylmethane diisocyanate (MDI)	Not Available
C.I. Pigment Yellow 74	Not Available
1,3,5-trimethyl benzene	Not Available
carbon black	Not Available
silica amorphous	Not Available
cumene	Not Available
diisobutyl ketone	Not Available
1-decene homopolymer, hydrogenated	Not Available
bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate	Not Available
p-toluenesulfonyl isocyanate	Not Available
C7-9 branched alkyl-3,5-di-tert-butyl-4-hydroxyhydrocinnamate	Not Available
dibutyltin dilauryl mercaptide	Not Available
methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate	Not Available

SECTION 15 Regulatory information

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

naphtha petroleum, light aromatic solvent 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
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

polyethylene 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

1,2,4-trimethyl benzene 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

Australian Inventory of Industrial Chemicals (AIIC)

titanium dioxide is found on the following regulatory lists

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

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

di-sec-octyl phthalate is found on the following regulatory lists

DURAM ROCKTUFF (BLACK, DARK GREY, LIGHT GREY, YELLOW)

<p>Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals</p> <p>Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C</p> <p>Australian Inventory of Industrial Chemicals (AIIC)</p>	<p>Chemical Footprint Project - Chemicals of High Concern List</p> <p>International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs</p> <p>International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans</p>
<p>MDI homopolymer is found on the following regulatory lists</p>	
<p>Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals</p>	<p>Australian Inventory of Industrial Chemicals (AIIC)</p>
<p>4,4'-diphenylmethane diisocyanate (MDI) is found on the following regulatory lists</p>	
<p>Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals</p> <p>Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring</p> <p>Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6</p>	<p>Australian Inventory of Industrial Chemicals (AIIC)</p> <p>International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs</p>
<p>C.I. Pigment Yellow 74 is found on the following regulatory lists</p>	
<p>Australian Inventory of Industrial Chemicals (AIIC)</p>	
<p>1,3,5-trimethyl benzene is found on the following regulatory lists</p>	
<p>Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals</p> <p>Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5</p>	<p>Australian Inventory of Industrial Chemicals (AIIC)</p>
<p>carbon black is found on the following regulatory lists</p>	
<p>Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals</p> <p>Australian Inventory of Industrial Chemicals (AIIC)</p> <p>Chemical Footprint Project - Chemicals of High Concern List</p>	<p>International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs</p> <p>International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans</p> <p>International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)</p>
<p>silica amorphous is found on the following regulatory lists</p>	
<p>Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals</p> <p>Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C</p> <p>Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4</p>	<p>Australian Inventory of Industrial Chemicals (AIIC)</p> <p>International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs</p> <p>International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)</p>
<p>cumene is found on the following regulatory lists</p>	
<p>Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals</p> <p>Australian Inventory of Industrial Chemicals (AIIC)</p> <p>Chemical Footprint Project - Chemicals of High Concern List</p>	<p>International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs</p> <p>International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans</p>
<p>diisobutyl ketone is found on the following regulatory lists</p>	
<p>Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals</p>	<p>Australian Inventory of Industrial Chemicals (AIIC)</p>
<p>1-decene homopolymer, hydrogenated is found on the following regulatory lists</p>	
<p>Australian Inventory of Industrial Chemicals (AIIC)</p>	
<p>bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate is found on the following regulatory lists</p>	
<p>Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals</p>	<p>Australian Inventory of Industrial Chemicals (AIIC)</p>
<p>p-toluenesulfonyl isocyanate is found on the following regulatory lists</p>	
<p>Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals</p> <p>Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring</p>	<p>Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6</p> <p>Australian Inventory of Industrial Chemicals (AIIC)</p>
<p>C7-9 branched alkyl-3,5-di-tert-butyl-4-hydroxyhydrocinnamate is found on the following regulatory lists</p>	
<p>Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals</p>	<p>Australian Inventory of Industrial Chemicals (AIIC)</p>
<p>dibutyltin dilauryl mercaptide is found on the following regulatory lists</p>	
<p>Australian Inventory of Industrial Chemicals (AIIC)</p>	<p>Chemical Footprint Project - Chemicals of High Concern List</p>
<p>methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate is found on the following regulatory lists</p>	
<p>Australian Inventory of Industrial Chemicals (AIIC)</p>	

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (naphtha petroleum, light aromatic solvent; polyethylene; 1,2,4-trimethyl benzene; di-sec-octyl phthalate; MDI homopolymer; 4,4'-diphenylmethane diisocyanate (MDI); C.I. Pigment Yellow 74; 1,3,5-trimethyl benzene; carbon black; cumene; diisobutyl ketone; 1-decene homopolymer, hydrogenated; bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate; p-toluenesulfonyl isocyanate; C7-9 branched alkyl-3,5-di-tert-butyl-4-hydroxyhydrocinnamate; dibutyltin dilauryl mercaptide; methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (polyethylene; C7-9 branched alkyl-3,5-di-tert-butyl-4-hydroxyhydrocinnamate)

DURAM ROCKTUFF (BLACK, DARK GREY, LIGHT GREY, YELLOW)

National Inventory	Status
Japan - ENCS	No (C7-9 branched alkyl-3,5-di-tert-butyl-4-hydroxyhydrocinnamate)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (MDI homopolymer; p-toluenesulfonyl isocyanate; methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate)
Vietnam - NCI	Yes
Russia - FBEPH	No (C7-9 branched alkyl-3,5-di-tert-butyl-4-hydroxyhydrocinnamate; methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate)
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

SECTION 16 Other information

Revision Date	07/03/2020
Initial Date	18/01/2017

SDS Version Summary

Version	Date of Update	Sections Updated
4.1.1.1	01/11/2019	One-off system update. NOTE: This may or may not change the GHS classification
5.1.1.1	07/03/2020	Classification change due to full database hazard calculation/update.
5.1.2.1	26/04/2021	Regulation Change
5.1.3.1	03/05/2021	Regulation Change
5.1.4.1	06/05/2021	Regulation Change
5.1.5.1	10/05/2021	Regulation Change
5.1.5.2	30/05/2021	Template Change
5.1.5.3	04/06/2021	Template Change
5.1.5.4	05/06/2021	Template Change
5.1.6.4	07/06/2021	Regulation Change
5.1.6.5	09/06/2021	Template Change
5.1.6.6	11/06/2021	Template Change
5.1.6.7	15/06/2021	Template Change
5.1.7.7	17/06/2021	Regulation Change
5.1.8.7	21/06/2021	Regulation Change

Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.

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

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
 PC—STEL: Permissible Concentration-Short Term Exposure Limit
 IARC: International Agency for Research on Cancer
 ACGIH: American Conference of Governmental Industrial Hygienists
 STEL: Short Term Exposure Limit
 TEEL: Temporary Emergency Exposure Limit.
 IDLH: Immediately Dangerous to Life or Health Concentrations
 ES: Exposure Standard
 OSF: Odour Safety Factor
 NOAEL :No Observed Adverse Effect Level
 LOAEL: Lowest Observed Adverse Effect Level
 TLV: Threshold Limit Value
 LOD: Limit Of Detection
 OTV: Odour Threshold Value
 BCF: BioConcentration Factors
 BEI: Biological Exposure Index
 AIIC: Australian Inventory of Industrial Chemicals
 DSL: Domestic Substances List
 NDSL: Non-Domestic Substances List
 IECSC: Inventory of Existing Chemical Substance in China
 EINECS: European INventory of Existing Commercial chemical Substances
 ELINCS: European List of Notified Chemical Substances
 NLP: No-Longer Polymers
 ENCS: Existing and New Chemical Substances Inventory
 KECI: Korea Existing Chemicals Inventory
 NZIoC: New Zealand Inventory of Chemicals
 PICCS: Philippine Inventory of Chemicals and Chemical Substances

DURAM ROCKTUFF (BLACK, DARK GREY, LIGHT GREY, YELLOW)

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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