

Duram Multithane UVHV

Duram

Chemwatch: 74-3219

Version No: 4.1.8.7

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

Chemwatch Hazard Alert Code: 2

Issue Date: 01/11/2019

Print Date: 22/06/2021

L.GHS.AUS.EN

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

Product Identifier

Product name	Duram Multithane UVHV
Chemical Name	Not Applicable
Synonyms	Not Available
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	High viscosity liquid polyurethane waterproofing membrane for exposed areas. Use according to manufacturer's directions.
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Details of the supplier of the safety data sheet

Registered company name	Duram
Address	51 Prince William Drive Seven Hills NSW 2147 Australia
Telephone	+61 2 9624 4077
Fax	+61 2 9624 4079
Website	http://www.duram.com.au/
Email	mail@duram.com.au

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	Not Available
Other emergency telephone numbers	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

COMBUSTIBLE LIQUID, regulated for storage purposes only

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, Reproductive Toxicity Category 2, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Acute Aquatic Hazard Category 2, Chronic Aquatic Hazard Category 3, Flammable Liquid Category 4
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	
Signal word	Danger

Hazard statement(s)

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.
H361fd	Suspected of damaging fertility. Suspected of damaging the unborn child.
H335	May cause respiratory irritation.
H401	Toxic to aquatic life.

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H412	Harmful to aquatic life with long lasting effects.
H227	Combustible liquid.

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.
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

P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
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.
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.

Precautionary statement(s) Storage

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

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

Mixtures

CAS No	%[weight]	Name
1317-65-3	20-40	<u>limestone</u>
68515-48-0	10-30	<u>diisononyl phthalate</u>
64742-94-5	<5	<u>solvent naphtha petroleum, heavy aromatic</u>
64742-95-6	<5	<u>naphtha petroleum, light aromatic solvent</u>
13463-67-7	<5	<u>titanium dioxide</u>
5873-54-1	<5	<u>2,4'-diphenylmethane diisocyanate</u>
95-63-6	<5	<u>1,2,4-trimethyl benzene</u>
101-68-8	<5	<u>4,4'-diphenylmethane diisocyanate (MDI)</u>
1305-78-8	<5	<u>calcium oxide</u>
4098-71-9	<0.5	<u>isophorone diisocyanate</u>
140921-24-0	<0.5	<u>carbamic acid, complex ester</u>
25686-28-6	<0.2	<u>MDI homopolymer</u>
4083-64-1	<0.2	<u>p-toluenesulfonyl isocyanate</u>
91-20-3	<0.2	<u>naphthalene</u>
Not Available	balance	Ingredients determined not to be hazardous

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	
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Continued...

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	<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.
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

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 naphthalene intoxication: Naphthalene requires hepatic and microsomal activation prior to the production of toxic effects. Liver microsomes catalyse the initial synthesis of the reactive 1,2-epoxide intermediate which is subsequently oxidised to naphthalene dihydrodiol and alpha-naphthol. The 2-naphthoquinones are thought to produce haemolysis, the 1,2-naphthoquinones are thought to be responsible for producing cataracts in rabbits, and the glutathione-adducts of naphthalene-1,2-oxide are probably responsible for pulmonary toxicity. Suggested treatment regime:

- ▶ Induce emesis and/or perform gastric lavage with large amounts of warm water where oral poisoning is suspected.
- ▶ Instill a saline cathartic such as magnesium or sodium sulfate in water (15 to 30g).
- ▶ Demulcents such as milk, egg white, gelatin, or other protein solutions may be useful after the stomach is emptied but oils should be avoided because they promote absorption.
- ▶ If eyes/skin contaminated, flush with warm water followed by the application of a bland ointment.
- ▶ Severe anaemia, due to haemolysis, may require small repeated blood transfusions, preferably with red cells from a non-sensitive individual.
- ▶ Where intravascular haemolysis, with haemoglobinuria occurs, protect the kidneys by promoting a brisk flow of dilute urine with, for example, an osmotic diuretic such as mannitol. It may be useful to alkalise the urine with small amounts of sodium bicarbonate but many researchers doubt whether this prevents blockage of the renal tubules.
- ▶ Use supportive measures in the case of acute renal failure. GOSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products, 5th Ed.

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.

For acute or short term repeated exposures to xylene:

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

BIOLOGICAL EXPOSURE INDEX - BEI

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

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

Continued...

SECTION 5 Firefighting measures

Extinguishing media

- ▶ Water spray or fog.
- ▶ Alcohol stable foam.
- ▶ Dry chemical powder.
- ▶ Carbon dioxide.

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. ▶ Wear full body protective clothing with breathing apparatus. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Use water delivered as a fine spray to control fire and cool adjacent area. ▶ Avoid spraying water onto liquid pools. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ Combustible. ▶ Slight fire hazard when exposed to heat or flame. ▶ Heating may cause expansion or decomposition leading to violent rupture of containers. ▶ On combustion, may emit toxic fumes of carbon monoxide (CO). ▶ May emit acrid smoke. ▶ Mists containing combustible materials may be explosive. <p>Combustion products include: carbon dioxide (CO₂) isocyanates and minor amounts of hydrogen cyanide nitrogen oxides (NO_x) other pyrolysis products typical of burning organic material.</p>
HAZCHEM	Not Applicable

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"> ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Control personal contact with the substance, by using protective equipment. ▶ Contain and absorb spill with sand, earth, inert material or vermiculite. ▶ Wipe up. ▶ Place in a suitable, labelled container for waste disposal. 																																																																											
Major Spills	<p>Environmental hazard - contain spillage. Chemical Class: aromatic hydrocarbons For release onto land: recommended sorbents listed in order of priority.</p> <table border="1"> <thead> <tr> <th>SORBENT TYPE</th> <th>RANK</th> <th>APPLICATION</th> <th>COLLECTION</th> <th>LIMITATIONS</th> </tr> </thead> <tbody> <tr> <td colspan="5">LAND SPILL - SMALL</td> </tr> <tr> <td>Feathers - pillow</td> <td>1</td> <td>throw</td> <td>pitchfork</td> <td>DGC, RT</td> </tr> <tr> <td>cross-linked polymer - particulate</td> <td>2</td> <td>shovel</td> <td>shovel</td> <td>R,W,SS</td> </tr> <tr> <td>cross-linked polymer- pillow</td> <td>2</td> <td>throw</td> <td>pitchfork</td> <td>R, DGC, RT</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>3</td> <td>shovel</td> <td>shovel</td> <td>R, I, P,</td> </tr> <tr> <td>treated clay/ treated natural organic - particulate</td> <td>3</td> <td>shovel</td> <td>shovel</td> <td>R, I</td> </tr> <tr> <td>wood fibre - pillow</td> <td>4</td> <td>throw</td> <td>pitchfork</td> <td>R, P, DGC, RT</td> </tr> <tr> <td colspan="5">LAND SPILL - MEDIUM</td> </tr> <tr> <td>cross-linked polymer -particulate</td> <td>1</td> <td>blower</td> <td>skiploader</td> <td>R, W, SS</td> </tr> <tr> <td>treated clay/ treated natural organic - particulate</td> <td>2</td> <td>blower</td> <td>skiploader</td> <td>R, I</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>3</td> <td>blower</td> <td>skiploader</td> <td>R, I, P</td> </tr> <tr> <td>polypropylene - particulate</td> <td>3</td> <td>blower</td> <td>skiploader</td> <td>W, SS, DGC</td> </tr> <tr> <td>feathers - pillow</td> <td>3</td> <td>throw</td> <td>skiploader</td> <td>DGC, RT</td> </tr> <tr> <td>expanded mineral - particulate</td> <td>4</td> <td>blower</td> <td>skiploader</td> <td>R, I, W, P, DGC</td> </tr> </tbody> </table> <p>Legend DGC: Not effective where ground cover is dense</p>	SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS	LAND SPILL - SMALL					Feathers - pillow	1	throw	pitchfork	DGC, RT	cross-linked polymer - particulate	2	shovel	shovel	R,W,SS	cross-linked polymer- pillow	2	throw	pitchfork	R, DGC, RT	sorbent clay - particulate	3	shovel	shovel	R, I, P,	treated clay/ treated natural organic - particulate	3	shovel	shovel	R, I	wood fibre - pillow	4	throw	pitchfork	R, P, DGC, RT	LAND SPILL - MEDIUM					cross-linked polymer -particulate	1	blower	skiploader	R, W, SS	treated clay/ treated natural organic - particulate	2	blower	skiploader	R, I	sorbent clay - particulate	3	blower	skiploader	R, I, P	polypropylene - particulate	3	blower	skiploader	W, SS, DGC	feathers - pillow	3	throw	skiploader	DGC, RT	expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC
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R: Not reusable

I: Not incinerable

P: Effectiveness reduced when rainy

RT: Not effective where terrain is rugged

SS: Not for use within environmentally sensitive sites

W: Effectiveness reduced when windy

Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;

R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988

For isocyanate spills of less than 40 litres (2 m²):

- ▶ 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.

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

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.

- ▶ 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.

Moderate hazard.

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

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling

- ▶ **DO NOT allow clothing wet with material to stay in contact with skin**

Continued...

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	<ul style="list-style-type: none"> ▶ 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 exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ Avoid smoking, naked lights or ignition sources. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
Other information	<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. - 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. <ul style="list-style-type: none"> ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Metal can or drum ▶ Packaging as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<ul style="list-style-type: none"> ▶ Avoid contamination with water, alkalies 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. ▶ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	limestone	Calcium carbonate	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
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	2,4'-diphenylmethane diisocyanate	Isocyanates, all (as-NCO)	0.02 mg/m3	0.07 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	calcium oxide	Calcium oxide	2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	isophorone diisocyanate	Isophorone diisocyanate	0.02 mg/m3	0.07 mg/m3	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

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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	naphthalene	Naphthalene	10 ppm / 52 mg/m3	79 mg/m3 / 15 ppm	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
limestone	45 mg/m3	210 mg/m3	1,300 mg/m3
naphtha petroleum, light aromatic solvent	1,200 mg/m3	6,700 mg/m3	40,000 mg/m3
titanium dioxide	30 mg/m3	330 mg/m3	2,000 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
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
calcium oxide	6 mg/m3	110 mg/m3	660 mg/m3
isophorone diisocyanate	0.02 ppm	0.14 ppm	0.6 ppm
naphthalene	15 ppm	83 ppm	500 ppm

Ingredient	Original IDLH	Revised IDLH
limestone	Not Available	Not Available
diisononyl phthalate	Not Available	Not Available
solvent naphtha petroleum, heavy aromatic	Not Available	Not Available
naphtha petroleum, light aromatic solvent	Not Available	Not Available
titanium dioxide	5,000 mg/m3	Not Available
2,4'-diphenylmethane diisocyanate	Not Available	Not Available
1,2,4-trimethyl benzene	Not Available	Not Available
4,4'-diphenylmethane diisocyanate (MDI)	75 mg/m3	Not Available
calcium oxide	25 mg/m3	Not Available
isophorone diisocyanate	Not Available	Not Available
carbamic acid, complex ester	Not Available	Not Available
MDI homopolymer	Not Available	Not Available
p-toluenesulfonyl isocyanate	Not Available	Not Available
naphthalene	250 ppm	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
diisononyl phthalate	E	≤ 0.1 ppm
1,2,4-trimethyl benzene	E	≤ 0.1 ppm
carbamic acid, complex ester	D	> 0.1 to ≤ 1 ppm
MDI homopolymer	E	≤ 0.1 ppm

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

MATERIAL DATA

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

<p>Appropriate engineering controls</p>	<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> <p>Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. An approved self contained breathing apparatus (SCBA) may be required in some situations.</p> <p>Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.</p>
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	<p>Type of Contaminant:</p> <p>solvent, vapours, degreasing etc., evaporating from tank (in still air).</p> <p>aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)</p> <p>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</p> <p>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</p> <p>Within each range the appropriate value depends on:</p> <table border="1" data-bbox="384 488 1118 651"> <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 (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p>	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	<p>Air Speed:</p> <p>0.25-0.5 m/s (50-100 f/min.)</p> <p>0.5-1 m/s (100-200 f/min.)</p> <p>1-2.5 m/s (200-500 f/min.)</p> <p>2.5-10 m/s (500-2000 f/min.)</p>
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											
<p>Personal protection</p>												
<p>Eye and face protection</p>	<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] 											
<p>Skin protection</p>	<p>See Hand protection below</p>											
<p>Hands/feet protection</p>	<ul style="list-style-type: none"> ▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber <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. · 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>											

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Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ P.V.C apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash unit.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

The effect(s) of the following substance(s) are taken into account in the **computer-generated** selection:

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Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVA	C
PVC	C
PVDC/PE/PVDC	C
TEFLON	C
VITON	C

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.

Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

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

^ - Full-face

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

- ▶ Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- ▶ The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- ▶ Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used
- ▶ In certain circumstances, personal protection of the individual employee is necessary. Personal protective devices should be regarded as being supplementary to substitution and engineering control and should not be used in preference to them as they do nothing to eliminate the hazard.
- ▶ However, in some situations, minimising exposure to isocyanates by enclosure and ventilation is not possible, and occupational exposure standards may be exceeded, particularly during on-site mixing of paints, spray-painting, foaming and maintenance of machine and ventilation systems. In these situations, air-line respirators or self-contained breathing apparatus complying with the appropriate national standard must be used.
- ▶ **Organic vapour respirators with particulate pre-filters and powered, air-purifying respirators are NOT suitable.**
- ▶ Personal protective equipment must be appropriately selected, individually fitted and workers trained in their correct use and maintenance. Personal protective equipment must be regularly checked and maintained to ensure that the worker is being protected.
- ▶ Air-line respirators or self-contained breathing apparatus complying with the appropriate national standard should be used during the clean-up of spills and the repair or clean-up of contaminated equipment and similar situations which cause emergency exposures to hazardous atmospheric concentrations of isocyanate.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Coloured liquid with a characteristic odour; not miscible with water.		
Physical state	Liquid	Relative density (Water = 1)	1.39
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)	161	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	75	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available

Continued...

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Flammability	Combustible.	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Miscible	pH as a solution (%)	Not Available
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 be harmful. 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>
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. Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis). Accidental ingestion of the material may be damaging to the health of the individual. Phthalates (aromatic dicarboxylic acid esters), in general, exhibit low toxicity, partly because of poor absorption but mainly as a result of rapid metabolism in which the esters are saponified to phthalic acid (which is rapidly excreted) and the parent alcohol (which is subsequently metabolised). The pathology of these compounds seems to be related to the released alcohol and its biological effects. The rate of absorption of ingested phthalate esters is influenced by the content of dietary fat. Ingested phthalate esters may to a lesser degree be absorbed as the monoester derivatives or in the case of di(2-ethylhexyl)phthalate, as the diester. Cumulative toxicity of the phthalates has been observed on repeated administration. Both di-n-octyl phthalate and di(2-ethylhexyl)phthalate were found to have 22-28 times greater toxicity (based on LD50s) following repeated administration to animals. The liver has been shown to be the target organ affected by the phthalates. In general phthalates have induced liver enlargement; this increase in liver weight has been attributed to rapid cell division (hyperplasia) along with the detachment of cells (hypertrophy). The increase in liver weight caused by phthalates has been found to reverse to normal or even below normal levels on prolonged exposure. Exposure to phthalates, in general, has been found to be associated with a reduction in circulating cholesterol and serum triglyceride levels which accounted for a reduction in liver steroidogenesis. The phthalates also effect carbohydrate metabolism in the liver producing depleted glycogen electron transport inhibitors following interaction with mitochondria. Testicular atrophy produced in rats during feeding studies depends on the length and structure of the alcohol; in general the lower molecular weight esters produce the more severe effects. The toxicity of phthalic acid isomers decreases in the order o-phthalic acid, isophthalic acid and terephthalic acid. Phthalic acid is not metabolised but is excreted, unchanged, in the urine and faeces. Terephthalic acid appears to potentiate the biological effects of substances such as antibiotics, thiamine and sulfonamides.</p>
Skin Contact	<p>The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either</p> <ul style="list-style-type: none"> ▶ produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or ▶ produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. <p>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. Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. Open cuts, abraded or irritated skin should not be exposed to this material</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> <p>Exposure to naphthalene and its congeners has produced cataracts in animals and workers. In one study, eight of twenty-one workers, exposed to naphthalene for 5-years, showed opacities of the lens.</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. 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. Practical experience shows that skin contact with the material is capable of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway</p>

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.

Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion 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 are not a secondary non-specific consequence of other toxic effects.

Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects.

On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.

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

In a two-year inhalation study, groups of mice were exposed at 0, 10 or 30 ppm naphthalene, 6 hours/day, 5 days/week for 103 weeks. Female mice showed an increase of pulmonary alveolar/bronchiolar adenomas at 30 ppm. There was no increase in the incidence of tumours in male mice. Naphthalene inhalation was associated with an increase in the incidence and severity of chronic inflammation, metaplasia of the olfactory epithelium, and hyperplasia of the respiratory epithelium in the nose, and chronic inflammation of the lungs of both sexes.

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

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

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

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

The various phthalates have different uses, chemical structures and toxicity profiles. It is therefore difficult to generalise about the safety of all phthalates as a group. The main health concern associated with some phthalates is that animal studies have shown that high regular doses can affect the reproductive system in developing young, particularly males. While there is no significant risk to the general population, young children may experience higher exposures than the general population if they chew or suck on phthalate-containing toys, or if they ingest phthalates over a long period from other products containing high levels of phthalates.

In animal tests, phthalates have been shown to "feminise" male animals, increasing the likelihood of small or undeveloped testes, undescended testicles, and low sperm counts. A 2005 study also linked higher foetal exposure to phthalates through the mother's blood with increased risk of developmental abnormalities in male infants. Higher phthalate levels are also associated with lower testosterone production and reduced sperm count in men.

One study suggested that high levels of phthalates may be connected to the current obesity epidemic in children. It was found that obese children show greater exposure to phthalates than non-obese children. It was reported that the obesity risk increases according to the level of the chemical found in the children's bloodstream. In a national cross-section of U.S. men, concentrations of several prevalent phthalate metabolites showed statistically significant correlations with abnormal obesity and insulin resistance. A further study found that people with elevated phthalate levels had roughly twice the risk of developing diabetes compared with those with lower levels. This study also found that phthalates were associated with disrupted insulin production.

Much of the current research on effects of phthalate exposure has been focused towards children and men's health, however, women may be at higher risk for potential adverse health effects of phthalates due to increased cosmetic use. According to in vivo and observational studies there is an association between phthalate exposure and endocrine disruption leading to development of breast cancer. This finding may be associated with the demethylation of the oestrogen receptor complex in breast cancer cells.

A Russian study describes exposure by workers to mixed phthalates (and other plasticisers) - pain, numbness and spasms in the upper and lower extremities were related to duration of exposures. Symptoms usually developed after the sixth or seventh year of work. Neurological studies revealed the development of polyneuritis in about 30% of the workers involved in this study. About 30% of the workforce showed depression of the vestibular receptors. Because the study described mixed exposures it is difficult to determine what, if any, unique role was played by the phthalates. Increased incidences of anovulatory reproductive cycles and low oestrogen concentrations were reported among Russian women working with phthalate plasticisers; the abnormal cycles were associated with spontaneous abortion. The specific phthalates implicated, dose levels and other data were not reported. It has been alleged that the phthalates mimic or interfere with sex packaging) and are used as ingredients in paints, inks and adhesives. Their potential for entering the human body is marked. They have been added to a list of chemicals (including alkyl phenolics, polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs) and dioxins) which are implicated in reducing sperm counts and fertility in males a phenomenon which has apparently arisen since the mid 1960s.

Phthalates are generally considered to be in a class of endocrine disruptors known as "xenoestrogens," for their ability to mimic the effect of oestrogen on the body.

Although the human foetus is "bathed" in naturally occurring oestrogens during pregnancy it is suggested that it has developed a protective mechanism against natural oestrogens but is not safe from synthetic variants. These tend to accumulate in body fats which sets them apart from the natural product. During early pregnancy, fats are broken down and may flood the body with concentrated pollutants

Human phthalate exposure during pregnancy results in decreased anogenital distance among baby boys. Boys born to mothers with the highest levels of phthalates were 7 times more likely to have a shortened anogenital distance.

While anogenital distance is routinely used as a measure of foetal exposure to endocrine disruptors in animals, this parameter is rarely assessed in humans, and its significance is unknown

One study also found that female animals exposed to higher levels of phthalates experienced increased risk of miscarriage, a common symptom of excessive estrogen levels in human women, and stillbirth. Prematurity may also be linked to phthalate exposure.

Another study found a link between exposure to phthalates and increased rates of childhood obesity.

In adult human men, phthalates have been linked to greater waist circumference and higher insulin resistance, a common precursor to type 2 (adult onset) diabetes. They have been linked to thyroid irregularities, asthma, and skin allergies in both sexes. Though the exact mechanism is unclear, studies have linked higher rates of respiratory infections and other symptoms in children living in houses with vinyl floors. One possible explanation is inhalation of dust tainted by phthalates, which are used in cosmetics such as nail polishes and hand creams precisely because of their ability to bind to human tissues.

Animal studies have shown increased risks of certain birth defects (including the genital abnormalities and, in rats, extra ribs) and low birth rates in rats whose mothers were fed higher levels of phthalates.

	<p>These effects on foetal development are of particular concern because young women of childbearing age often have higher than average phthalate levels in the body thanks to their use of cosmetics, many of which contain phthalates.</p> <p>The EU has applied limitations to the use of several phthalates in general food contact applications (packaging and closures) and medical device applications. The USA has introduced regulation of phthalate esters as components of children's toys and childcare articles for children under the age of 12 that could be 'placed in the mouth'.</p> <p>Endocrine disruptors such as phthalates can be added to the effects of other endocrine disruptors, so even very small amounts can interact with other chemicals to have cumulative, adverse "cocktail effects"</p> <p>Large amounts of specific phthalates fed to rodents have been shown to damage their liver and testes, and initial rodent studies also indicated hepatocarcinogenicity. Later studies on primates showed that the mechanism is specific to rodents - humans are resistant to the effect</p> <p>Studies conducted on mice exposed to phthalates in utero did not result in metabolic disorder in adults. However, "At least one phthalate, monoethylhexyl phthalate (MEHP) has been found to interact with all three peroxisome proliferator-activated receptors (PPARs) PPARs are members of the nuclear receptor superfamily involved in lipid and carbohydrate metabolism.</p> <p>Prenatal exposure to phthalates may affect children's mental, motor and behavioral development during the preschool year.</p> <p>A 2009 study found that prenatal phthalate exposure was related to low birth weight in infants. Low birth weight is the leading cause of death in children under 5 years of age and increases the risk of cardiovascular and metabolic disease in adulthood. Another study found that women who deliver prematurely have, on average, up to three times the phthalate level in their urine compared to women who carry to term.</p> <p>Several findings point to a statistically significant correlation between urine phthalate concentrations in children and symptoms of attention deficit hyperactivity disorder (ADHD)</p>	
Duram Multithane UVHV	TOXICITY Not Available	IRRITATION Not Available
limestone	TOXICITY Oral(Rat) LD50; 6450 mg/kg ^[2]	IRRITATION Eye: no adverse effect observed (not irritating) ^[1] Skin (rabbit): 500 mg/24h-moderate Skin: no adverse effect observed (not irritating) ^[1]
diisononyl phthalate	TOXICITY Dermal (rabbit) LD50: >3160 mg/kg ^[2] Inhalation(Rat) LC50; >4.4 mg/l4h ^[1] Oral(Rat) LD50; 2550 mg/kg ^[2]	IRRITATION Not Available
solvent naphtha petroleum, heavy aromatic	TOXICITY Dermal (rabbit) LD50: >2000 mg/kg ^[2] Inhalation(Rat) LC50; >0.003 mg/L4h ^[1] Oral(Rat) LD50; 512 mg/kg ^[1]	IRRITATION Eye (rabbit): Irritating Eye: no adverse effect observed (not irritating) ^[1] Skin: adverse effect observed (irritating) ^[1]
naphtha petroleum, light aromatic solvent	TOXICITY Dermal (rabbit) LD50: >1900 mg/kg ^[1] Inhalation(Rat) LC50; >4.42 mg/L4h ^[1] Oral(Rat) LD50; >4500 mg/kg ^[1]	IRRITATION Eye: no adverse effect observed (not irritating) ^[1] Skin: adverse effect observed (irritating) ^[1]
titanium dioxide	TOXICITY dermal (hamster) LD50: >=10000 mg/kg ^[2] Inhalation(Rat) LC50; >2.28 mg/l4h ^[1] Oral(Rat) LD50; >=2000 mg/kg ^[1]	IRRITATION Eye: no adverse effect observed (not irritating) ^[1] Skin (human): 0.3 mg /3D (int)-mild * Skin: no adverse effect observed (not irritating) ^[1]
2,4'-diphenylmethane diisocyanate	TOXICITY Dermal (rabbit) LD50: >9400 mg/kg ^[1] Inhalation(Rat) LC50; 0.368 mg/L4h ^[1] Oral(Rat) LD50; >2000 mg/kg ^[1]	IRRITATION Not Available
1,2,4-trimethyl benzene	TOXICITY Dermal (rabbit) LD50: >3160 mg/kg ^[2] Inhalation(Rat) LC50; 10.2 mg/L4h ^[1] Oral(Rat) LD50; 6000 mg/kg ^[1]	IRRITATION Not Available
4,4'-diphenylmethane diisocyanate (MDI)	TOXICITY Dermal (rabbit) LD50: >6200 mg/kg ^[2] Inhalation(Rat) LC50; 0.368 mg/L4h ^[1] Oral(Rat) LD50; >2000 mg/kg ^[1]	IRRITATION Dermal Sensitiser * Eye: no adverse effect observed (not irritating) ^[1] Skin (rabbit): 500 mg /24 hours Skin: adverse effect observed (irritating) ^[1]
calcium oxide	TOXICITY dermal (rat) LD50: >2000 mg/kg ^[1]	IRRITATION Eye: adverse effect observed (irreversible damage) ^[1]

Duram Multithane UVHV

	Inhalation(Rat) LC50; >3 mg/l4h ^[1] Oral(Rat) LD50; >2000 mg/kg ^[1]	Skin: adverse effect observed (irritating) ^[1]
isophorone diisocyanate	TOXICITY	IRRITATION
	dermal (rat) LD50: >=2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation(Rat) LC50; 0.031 mg/L4h ^[1]	Skin: adverse effect observed (irritating) ^[1]
	Oral(Rat) LD50; >=2000 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
carbamic acid, complex ester	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Not Available
	Oral(Rat) LD50; >2000 mg/kg ^[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]	
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]	
naphthalene	TOXICITY	IRRITATION
	dermal (rat) LD50: >2500 mg/kg ^[2]	Eye (rabbit): 100 mg - mild
	Inhalation(Rat) LC50; >0.4 mg/l4h ^[1]	Skin (rabbit):495 mg (open) - mild
	Oral(Rat) LD50; >2000 mg/kg ^[1]	

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

LIMESTONE	Eye (rabbit) 0.75: mg/24h - No evidence of carcinogenic properties. No evidence of mutagenic or teratogenic effects.
	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. 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.
DIISONONYL PHTHALATE	<p>[HLS] The effects of DINP on fertility-related parameters such as reduced testosterone content and production and altered reproductive organ weights (with or without histopathologies) have been demonstrated in rats. Although quantitatively being less potent, DINP has exhibited adverse effects on the male reproductive system and sexual differentiation during development in a number of rodent studies (e.g. increased nipple retention, testicular pathology and decreased AGD/AGI in male offspring), which are components of the antiandrogenic pattern observed with diethylhexyl phthalate (DEHP) (a known reproductive toxicant). Foetal expression of genes involved in androgen synthesis such as StAR and Cyp11a were also reduced. There was also a report of increased gene expression levels of Insl3 (a foetal Leydig cell product critical for testis descent) that may infer the impaired testicular steroidogenesis following exposure to DINP at high doses (e.g. = 750 mg/kg bw/d). Reduced Insl3 was also reported in numerous studies with DEHP. Considering the chemical composition of DINP, which is represented as mixed phthalates with side-chains made up of 5?10% methylethylhexyl, limited evidence of the toxicological properties of transitional phthalates may be expected at high doses of DINP tested The reduced pup weight was observed at approximately 100 mg/kg bw/d in both sexes, both in one- and two-generation reproductive studies in rats, in the absence of overt maternal toxicity. The pup weight reduction was also sustained and not considered solely related to low birth weight. In a post-natal toxicity study, reduced pup weight was also reduced at = 250 mg/kg bw/d. Therefore, this adverse effect of DINP is assessed as the most sensitive endpoint on offspring development. Overall, the available human data do not provide sufficient evidence for a causal relationship between exposure to DINP and adverse health effects in humans. There is also insufficient information to examine the mode of action of DINP on male reproductive tract development and sexual function in comparison with transitional phthalates. However, elements of the plausible mode of action for DINP effects on the male reproductive system, offspring growth and sexual differentiation are considered likely to be parallel in rats and humans if the exposure to DINP is high and within a critical window of development. Therefore, the effects observed in animal studies are regarded as relevant to a human risk assessment.</p> <p>High Molecular Weight Phthalate Esters (HMWPEs) Category as defined by the Phthalate Esters Panel HPV Testing Group (2001) and OECD (2004). The HMWPE group includes chemically similar substances produced from alcohols having backbone carbon lengths of >= 7. Due to their similar chemical structure, category members are generally similar with respect to physicochemical, biological and toxicological properties or display an expected trend. Thus, read-across for toxicity endpoints is an appropriate approach to characterise selected endpoints for members of this category.</p> <p>In some cases the substances have ester side group constituents that span two subcategories (i.e., transitional and high molecular weight constituents). If the level of C4 to C6 constituents in the substance exceeded 10%,the substance was conservatively placed in the transitional subcategory.</p> <p>High molecular weight phthalates are used nearly exclusively as plasticisers of PVC.</p> <p>They are very poorly soluble in water, and have very low vapor pressure. The extant database demonstrates that these substances have few biological effects. A notable exception to this generalisation is that hepatocarcinogenicity has been observed for diisononyl phthalate (DINP). The hepatocarcinogenicity effects of DINP are by a mechanism (peroxisomal proliferation) to which rodents are particularly sensitive. However, it does not appear to be relevant to humans.</p> <p>The high molecular weight phthalates all demonstrate minimal acute toxicity, are not genotoxic, exhibit some liver and kidney effects at high doses, and are negative for reproductive and developmental effects. Further, the available data indicate that the toxicological activity of these molecules diminishes with increasing molecular weight.</p> <p>Studies on HMWPEs indicate that they are rapidly metabolised in the gastrointestinal tract to the corresponding monoester, absorbed and excreted primarily in the urine.</p> <p>Acute toxicity: The available data on phthalates spanning the carbon range from C8-C13 indicate that phthalate esters in the high molecular</p>

weight subcategory are not toxic by acute oral and dermal administration; LD50 values of all substances tested exceed the maximum amounts which can be administered to the animals. There are fewer data available on inhalation toxicity; only di-iso-nonyl phthalate (DINP) and di-iso-decyl phthalate (DIDP) have been tested. However, the phthalates in the high molecular weight subcategory have extremely low vapor pressures, and exposure by inhalation at potentially hazardous levels is not anticipated.

Repeat dose toxicity. Several substances ranging from C8-C11 have been tested for repeated dose toxicity in studies ranging from 21 days to two years. Ditridecyl phthalate (CAS 119-06-2) has been studied by the Japan Ministry of Health and Welfare (unpublished report) and data for this substance is used as read-across data for DTDP*. In addition results from repeat dose studies examining DINP (CAS 685 15-48-0) and DIDP (CAS 68515-49-1) are used as read across for the di C9-C11 phthalates (CAS 68515-43-5). The principal effects found are those associated with peroxisomal proliferation, including liver enlargement and induction of peroxisomal enzymes. As shown for example in a comparative study of liver effects, the strongest inducers of peroxisomal proliferation were DEHP, DINP, and DIDP with substances of shorter and longer ester side chains (e.g., 610P*, 711P*, and diundecyl phthalate - DUP) showing less pronounced effects. Thus, it is reasonable to conclude that other members of this subcategory would show effects similar to but not more pronounced than those associated with DINP and 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 humans. Thus, one would expect humans to be substantially less responsive than rodents to peroxisome proliferating agents. Empirical evidence supporting this postulation is provided by studies in primates in which repeated administration of DEHP and DINP had no effects on liver, kidney or testicular parameters.

In this regard it should also be noted that kidney enlargement is also commonly observed but normally without any pathological changes. There is a component of the kidney changes which is also PPARα-related. It has also been shown that in male rats, DINP induces an alpha 2u-globulin nephropathy which is male rat- specific but without relevance to humans. Thus, as was true for the liver changes, the relevance of the kidney changes to human health is also questionable

Finally, some of the lower molecular weight phthalates can induce testicular atrophy when administered to juvenile rats at high levels. However, the higher molecular weight phthalates including di-n-octyl phthalate (DnOP), DINP, DIDP, 610P, and 711P do not induce testicular atrophy. Further, the testis was not a target organ for DINP in either marmosets or cynomolgus monkeys. Thus, testicular atrophy is not an effect associated with phthalates in the high molecular weight subcategory

Reproductive toxicity: Reproductive toxicity tests in rats have been carried out with DINP, DIDP a linear C7-C9 phthalate (CAS 68515-41-3), a linear C9-C11 phthalate, and ditridecyl phthalate (Japan Ministry of Health and Welfare, unpublished report). None of these affected fertility or profoundly affected male reproductive development. A slight decrease in offspring viability was reported for both DIDP and ditridecyl phthalate at levels associated with maternal effects. DnOP was tested for effects on fertility in a continuous breeding protocol in mice, and, like the other members of this subcategory, did not reduce fertility. Thus, it can be concluded that the subcategory of high molecular weight phthalates do not affect fertility.

Developmental toxicity: Developmental toxicity tests in rats have been carried out with DINP; DIDP; C7-9 phthalate (CAS 68515-41-3); C9-11 phthalate (CAS 68515-43-5); and ditridecyl phthalate (CAS 119-06-2). None of the substances tested affected litter size, foetal survival or bodyweight, and none produced teratogenic effects. Increased frequencies of developmental variants including dilated renal pelvis, and supernumerary lumbar and cervical ribs were found at levels associated with maternal effects. The toxicological significance of these developmental variants is unclear. DnOP was not teratogenic in mice when tested at very high levels. Thus, it can be concluded that this subcategory of high molecular weight phthalates do not produce profound developmental effects in rodents

Genotoxicity: The majority of the substances in the subcategory of high molecular weight phthalates have been tested for genetic activity in the Salmonella assay, and all were inactive. One large program covering many of these substances was carried out by the National Institute of Environmental Health Sciences. Similarly, a range of substances covering the majority of the carbon numbers in this subcategory were found to be inactive in mouse lymphoma tests

Chromosomal Aberrations. Two representative members of the subcategory of high molecular weight phthalates (DINP and DIDP) have been tested for chromosomal mutation in the mouse micronucleus test, and both were inactive. Ditridecyl phthalate (CAS 119-06-2) induced neither structural chromosomal aberrations nor polyploidy in CHL cells up to the limit concentration of 4.75 mg/ ml, in the absence or presence of an exogenous metabolic activation system (Japan Ministry of Health and Welfare, unpublished report). Further, all of the low molecular weight and transitional phthalates that have been tested were inactive.

*610P - mixed decyl, hexyl and octyl esters (CAS Rn: 68648-93-1)

*711P - C7,C11, branched and linear esters (CAS Rn: 111381-90-9)

* DTDP - di-C11-14, C13 rich ester (CAS 68515-47-9)

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. 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.

Inhalation (rat) TCLo: 1320 ppm/6h/90D-I * [Devoe]

For Low Boiling Point Naphthas (LBPNS):

Acute toxicity:

LBPNS generally have low acute toxicity by the oral (median lethal dose [LD50] in rats > 2000 mg/kg-bw), inhalation (LD50 in rats > 5000 mg/m3) and dermal (LD50 in rabbits > 2000 mg/kg-bw) routes of exposure

Most LBPNS are mild to moderate eye and skin irritants in rabbits, with the exception of heavy catalytic cracked and heavy catalytic reformed naphthas, which have higher primary skin irritation indices.

Sensitisation:

LBPNS do not appear to be skin sensitizers, but a poor response in the positive control was also noted in these studies

Repeat dose toxicity:

The lowest-observed-adverse-effect concentration (LOAEC) and lowest-observed-adverse-effect level (LOAEL) values identified following short-term (2-89 days) and subchronic (greater than 90 days) exposure to the LBPNS substances. These values were determined for a variety of endpoints after considering the toxicity data for all LBPNS in the group. Most of the studies were carried out by the inhalation route of exposure.

Renal effects, including increased kidney weight, renal lesions (renal tubule dilation, necrosis) and hyaline droplet formation, observed in male rats exposed orally or by inhalation to most LBPNS, were considered species- and sex-specific. These effects were determined to be due to a mechanism of action not relevant to humans -specifically, the interaction between hydrocarbon metabolites and alpha-2-microglobulin, an enzyme not produced in substantial amounts in female rats, mice and other species, including humans. The resulting nephrotoxicity and subsequent carcinogenesis in male rats were therefore not considered in deriving LOAEC/LOAEL values.

Only a limited number of studies of short-term and subchronic duration were identified for site-restricted LBPNS. The lowest LOAEC identified in these studies, via the inhalation route, is 5475 mg/m3, based on a concentration-related increase in liver weight in both male and female rats following a 13-week exposure to light catalytic cracked naphtha. Shorter exposures of rats to this test substance resulted in nasal irritation at 9041 mg/m3

No systemic toxicity was reported following dermal exposure to light catalytic cracked naphtha, but skin irritation and accompanying histopathological changes were increased, in a dose-dependent manner, at doses as low as 30 mg/kg-bw per day when applied 5 days per week for 90 days in rats

No non-cancer chronic toxicity studies (= 1 year) were identified for site-restricted LBPNS and very few non-cancer chronic toxicity studies were identified for other LBPNS. An LOAEC of 200 mg/m3 was noted in a chronic inhalation study that exposed mice and rats to unleaded gasoline (containing 2% benzene). This inhalation LOAEC was based on ocular discharge and ocular irritation in rats. At the higher concentration of 6170 mg/m3, increased kidney weight was observed in male and female rats (increased kidney weight was also observed in males only at 870 mg/m3). Furthermore, decreased body weight in male and female mice was also observed at 6170 mg/m3

A LOAEL of 714 mg/kg-bw was identified for dermal exposure based on local skin effects (inflammatory and degenerative skin changes) in mice following application of naphtha for 105 weeks. No systemic toxicity was reported.

**NAPHTHA PETROLEUM,
LIGHT AROMATIC SOLVENT**

Genotoxicity:

Although few genotoxicity studies were identified for the site-restricted LBPNS, the genotoxicity of several other LBPNS substances has been evaluated using a variety of in vivo and in vitro assays. While in vivo genotoxicity assays were negative overall, the in vitro tests exhibited mixed results.

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

While the majority of in vivo genotoxicity results for LBPNS substances are negative, the potential for genotoxicity of LBPNS as a group cannot be discounted based on the mixed in vitro genotoxicity results.

Carcinogenicity:

Although a number of epidemiological studies have reported increases in the incidence of a variety of cancers, the majority of these studies are considered to contain incomplete or inadequate information. Limited data, however, are available for skin cancer and leukemia incidence, as well as mortality among petroleum refinery workers. It was concluded that there is limited evidence supporting the view that working in petroleum refineries entails a carcinogenic risk (Group 2A carcinogen). IARC (1989a) also classified gasoline as a Group 2B carcinogen; it considered the evidence for carcinogenicity in humans from gasoline to be inadequate and noted that published epidemiological studies had several limitations, including a lack of exposure data and the fact that it was not possible to separate the effects of combustion products from those of gasoline itself. Similar conclusions were drawn from other reviews of epidemiological studies for gasoline (US EPA 1987a, 1987b). Thus, the evidence gathered from these epidemiological studies is considered to be inadequate to conclude on the effect of human exposure to LBPNS substances.

No inhalation studies assessing the carcinogenicity of the site-restricted LBPNS were identified. Only unleaded gasoline has been examined for its carcinogenic potential, in several inhalation studies. In one study, rats and mice were exposed to 0, 200, 870 or 6170 mg/m³ of a 2% benzene formulation of the test substance, via inhalation, for approximately 2 years. A statistically significant increase in hepatocellular adenomas and carcinomas, as well as a non-statistical increase in renal tumours, were observed at the highest dose in female mice. A dose-dependent increase in the incidence of primary renal neoplasms was also detected in male rats, but this was not considered to be relevant to humans, as discussed previously. Carcinogenicity was also assessed for unleaded gasoline, via inhalation, as part of initiation/promotion studies. In these studies, unleaded gasoline did not appear to initiate tumour formation, but did show renal cell and hepatic tumour promotion ability, when rats and mice were exposed, via inhalation, for durations ranging from 13 weeks to approximately 1 year using an initiation/promotion protocol. However, further examination of data relevant to the composition of unleaded gasoline demonstrated that this is a highly-regulated substance; it is expected to contain a lower percentage of benzene and has a discrete component profile when compared to other substances in the LBPNS group.

Both the European Commission and the International Agency for Research on Cancer (IARC) have classified LBPNS substances as carcinogenic. All of these substances were classified by the European Commission (2008) as Category 2 (R45: may cause cancer) (benzene content = 0.1% by weight). IARC has classified gasoline, an LBPNS, as a Group 2B carcinogen (possibly carcinogenic to humans) and "occupational exposures in petroleum refining" as Group 2A carcinogens (probably carcinogenic to humans).

Several studies were conducted on experimental animals to investigate the dermal carcinogenicity of LBPNS. The majority of these studies were conducted through exposure of mice to doses ranging from 694-1351 mg/kg-bw, for durations ranging from 1 year to the animals' lifetime or until a tumour persisted for 2 weeks. Given the route of exposure, the studies specifically examined the formation of skin tumours. Results for carcinogenicity via dermal exposure are mixed. Both malignant and benign skin tumours were induced with heavy catalytic cracked naphtha, light catalytic cracked naphtha, light

straight-run naphtha and naphtha. Significant increases in squamous cell carcinomas were also observed when mice were dermally treated with Stoddard solvent, but the latter was administered as a mixture (90% test substance), and the details of the study were not available. In contrast, insignificant increases in tumour formation or no tumours were observed when light alkylate naphtha, heavy catalytic reformed naphtha, sweetened naphtha, light catalytically cracked naphtha

or unleaded gasoline was dermally applied to mice. Negative results for skin tumours were also observed in male mice dermally exposed to sweetened naphtha using an initiation/promotion protocol.

Reproductive/ Developmental toxicity:

No reproductive or developmental toxicity was observed for the majority of LBPNS substances evaluated. Most of these studies were carried out by inhalation exposure in rodents.

NOAEC values for reproductive toxicity following inhalation exposure ranged from 1701 mg/m³ (CAS RN 8052-41-3) to 27 687 mg/m³ (CAS RN 64741-63-5) for the LBPNS group evaluated, and from 7690 mg/m³ to 27 059 mg/m³ for the site-restricted light catalytic cracked and full-range catalytic reformed naphthas. However, a decreased number of pups per litter and higher frequency of post-implantation loss were observed following inhalation exposure of female rats to hydrotreated heavy naphtha (CAS RN 64742-48-9) at a concentration of 4679 mg/m³, 6 hours per day, from gestational days 7-20. For dermal exposures, NOAEL values of 714 mg/kg-bw (CAS RN 8030-30-6) and 1000 mg/kg-bw per day (CAS RN 68513-02-0) were noted. For oral exposures, no adverse effects on reproductive parameters were reported when rats were given site-restricted light catalytic cracked naphtha at 2000 mg/kg on gestational day 13.

For most LBPNS, no treatment-related developmental effects were observed by the different routes of exposure. However, developmental toxicity was observed for a few naphthas. Decreased foetal body weight and an increased incidence of ossification variations were observed when rat dams were exposed to light aromatized solvent naphtha, by gavage, at 1250 mg/kg-bw per day. In addition, pregnant rats exposed by inhalation to hydrotreated heavy naphtha at 4679 mg/m³ delivered pups with higher birth weights. Cognitive and memory impairments were also observed in the offspring.

Low Boiling Point Naphthas [Site-Restricted]

For C9 aromatics (typically trimethylbenzenes - TMBs)

Acute Toxicity

Acute toxicity studies (oral, dermal and inhalation routes of exposure) have been conducted in rats using various solvent products containing predominantly mixed C9 aromatic hydrocarbons (CAS RN 64742-95-6). Inhalation LC50's range from 6,000 to 10,000 mg/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 rat dermal LD50 for the C9 aromatic naphtha is >4 ml/kg bw. These data indicate that C9 aromatic solvents show that LD50/LC50 values are greater than limit doses for acute toxicity studies established under OECD test guidelines.

Irritation and Sensitization

Several irritation studies, including skin, eye, and lung/respiratory system, have been conducted on members of the category. The results indicate that C9 aromatic hydrocarbon solvents are mildly to moderately irritating to the skin, minimally irritating to the eye, and have the potential to irritate the respiratory tract and cause depression of respiratory rates in mice. Respiratory irritation is a key endpoint in the current occupational exposure limits established for C9 aromatic hydrocarbon solvents and trimethylbenzenes. No evidence of skin sensitization was identified.

Repeated Dose Toxicity

Inhalation: The results from a subchronic (3 month) neurotoxicity study and a one-year chronic study (6 hr/day, 5 days/week) indicate that effects from inhalation exposure to C9 Aromatic Hydrocarbon Solvents on systemic toxicity are slight. A battery of neurotoxicity and neurobehavioral endpoints were evaluated in the 3-month inhalation study on C9 aromatic naphtha tested at concentrations of 0, 101, 452, or 1320 ppm (0, 500, 2,220, or 6,500 mg/m³). In this study, other than a transient weight reduction in the high exposure group (not statistically significant at termination of exposures), no effects were reported on neuropathology or neuro/behavioral parameters. The NOAEL for systemic and/or neurotoxicity was

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6,500 mg/m³, the highest concentration tested. In an inhalation study of a commercial blend, rats were exposed to C9 aromatic naphtha concentrations of 0, 96, 198, or 373 ppm (0, 470, 970, 1830 mg/m³) for 6 hr/day, 5 days/week, for 12 months. Liver and kidney weights were increased in the high exposure group but no accompanying histopathology was observed in these organs.

The NOAEL was considered to be the high exposure level of 373 ppm, or 1830 mg/m³. In two subchronic rat inhalation studies, both of three months duration, rats were exposed to the individual TMB isomers (1,2,4- and 1,3,5-) to nominal concentrations of 0, 25, 100, or 250 ppm (0, 123, 492, or 1230 mg/m³). Respiratory irritation was observed at 492 (100 ppm) and 1230 mg/m³ (250 ppm) and no systemic toxicity was observed in either study. For both pure isomers, the NOELs are 25 ppm or 123 mg/m³ for respiratory irritation and 250 ppm or 1230 mg/m³ for systemic effects.

Oral: The C9 aromatic naphtha has not been tested via the oral route of exposure. Individual TMB isomers have been evaluated in a series of repeated-dose oral studies ranging from 14 days to 3 months over a wide range of doses. The effects observed in these studies included increased liver and kidney weights, changes in blood chemistry, increased salivation, and decreased weight gain at higher doses. Organ weight changes appeared to be adaptive as they were not accompanied by histopathological effects. Blood changes appeared sporadic and without pattern. One study reported hyaline droplet nephropathy in male rats at the highest dose (1000 mg/kg bw-day), an effect that is often associated with alpha-2mu-globulin-induced nephropathy and not considered relevant to humans. The doses at which effects were detected were 100 mg/kg-bw day or above (an exception was the pilot 14 day oral study - LOAEL 150 mg/kg bw-day - but the follow up three month study had a LOAEL of 600 mg/kg/bw-day with a NOAEL of 200 mg/kg bw-day). Since effects generally were not severe and could be considered adaptive or spurious, oral exposure does not appear to pose a high toxicity hazard for pure trimethylbenzene isomers.

Mutagenicity

In vitro genotoxicity testing of a variety of C9 aromatics has been conducted in both bacterial and mammalian cells. In vitro point mutation tests were conducted with *Salmonella typhimurium* and *Escherichia coli* bacterial strains, as well as with cultured mammalian cells such as the Chinese hamster cell ovary cells (HGPRT assay) with and without metabolic activation. In addition, several types of in vitro chromosomal aberration tests have been performed (chromosome aberration frequency in Chinese hamster ovary and lung cells, sister chromatid exchange in CHO cells). Results were negative both with and without metabolic activation for all category members. For the supporting chemical 1,2,3-TMB, a single in vitro chromosome aberration test was weakly positive. In vivo bone marrow cytogenetics test, rats were exposed to C9 aromatic naphtha at concentrations of 0, 153, 471, or 1540 ppm (0, 750, 2,310, or 7,560 mg/m³) 6 hr/day, for 5 days. No evidence of in vivo somatic cell genotoxicity was detected. Based on the cumulative results of these assays, genetic toxicity is unlikely for substances in the C9 Aromatic

Hydrocarbon Solvents Category

Reproductive and Developmental Toxicity

Results from the three-generation reproduction inhalation study in rats indicate limited effects from C9 aromatic naphtha. In each of three generations (F0, F1 and F2), rats were exposed to High Flash Aromatic Naphtha (CAS RN 64742-95-6) via whole body inhalation at target concentrations of 0, 100, 500, or 1500 ppm (actual mean concentrations throughout the full study period were 0, 103, 495, or 1480 ppm, equivalent to 0, 505, 2430, or 7265 mg/m³, respectively). In each generation, both sexes were exposed for 10 weeks prior to and two weeks during mating for 6 hrs/day, 5 days/wks. Female rats in the F0, F1, and F2 generation were then exposed during gestation days 0-20 and lactation days 5-21 for 6 hrs/day, 7 days/wk. The age at exposure initiation differed among generations; F0 rats were exposed starting at 9 weeks of age, F1 exposure began at 5-7 weeks, and F2 exposure began at postnatal day (PND) 22. In the F0 and F1 parental generations, 30 rats/sex/group were exposed and mated. However, in the F2 generation, 40/sex/group were initially exposed due to concerns for toxicity, and 30/sex/group were randomly selected for mating, except that all survivors were used at 1480 ppm. F3 litters were not exposed directly and were sacrificed on lactation day 21.

Systemic Effects on Parental Generations:

The F0 males showed statistically and biologically significantly decreased mean body weight by ~15% at 1480 ppm when compared with controls. Seven females died or were sacrificed in extremis at 1480 ppm. The F0 female rats in the 495 ppm exposed group had a 13% decrease in body weight gain when adjusted for initial body weight when compared to controls. The F1 parents at 1480 ppm had statistically significantly decreased mean body weights (by ~13% (females) and 22% (males)), and locomotor activity. F1 parents at 1480 ppm had increased ataxia and mortality (six females). Most F2 parents (70/80) exposed to 1480 ppm died within the first week. The remaining animals survived throughout the rest of the exposure period. At week 4 and continuing through the study, F2 parents at 1480 ppm had statistically significant mean body weights much lower than controls (~33% for males; ~28% for females); body weights at 495 ppm were also reduced significantly (by 13% in males and 15% in females). The male rats in the 495 ppm exposed group had a 12% decrease in body weight gain when adjusted for initial body weight when compared to controls. Based on reduced body weight observed, the overall systemic toxicity LOAEC is 495 ppm (2430 mg/m³).

Reproductive Toxicity-Effects on Parental Generations: There were no pathological changes noted in the reproductive organs of any animal of the F0, F1, or F2 generation. No effects were reported on sperm morphology, gestational period, number of implantation sites, or post-implantation loss in any generation. Also, there were no statistically or biologically significant differences in any of the reproductive parameters, including: number of mated females, copulatory index, copulatory interval, number of females delivering a litter, number of females delivering a live litter, or male fertility in the F0 or in the F2 generation. Male fertility was statistically significantly reduced at 1480 ppm in the F1 rats. However, male fertility was not affected in the F0 or in the F2 generations; therefore, the biological significance of this change is unknown and may or may not be attributed to the test substance. No reproductive effects were observed in the F0 or F1 dams exposed to 1480 ppm (7265 mg/m³). Due to excessive mortality at the highest concentration (1480 ppm, only six dams available) in the F2 generation, a complete evaluation is precluded. However, no clear signs of reproductive toxicity were observed in the F2 generation. Therefore, the reproductive NOAEC is considered 495 ppm (2430 mg/m³), which excludes analysis of the highest concentration due to excessive mortality.

Developmental Toxicity - Effects on Pups: Because of significant maternal toxicity (including mortality) in dams in all generations at the highest concentration (1480 ppm), effects in offspring at 1480 ppm are not reported here. No significant effects were observed in the F1 and F2 generation offspring at 103 or 495 ppm. However, in F3 offspring, body weights and body weight gain were reduced by ~ 10-11% compared with controls at 495 ppm for approximately a week (PND 14 through 21). Maternal body weight was also depressed by ~ 12% throughout the gestational period compared with controls. The overall developmental LOAEC from this study is 495 ppm (2430 mg/m³) based on the body weights reductions observed in the F3 offspring.

Conclusion: No effects on reproductive parameters were observed at any exposure concentration, although a confident assessment of the group exposed at the highest concentration was not possible. A potential developmental effect (reduction in mean pup weight and weight gain) was observed at a concentration that was also associated with maternal toxicity.

TITANIUM DIOXIDE

* IUCLID

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.

For titanium dioxide:

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.

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.

No data were available on genotoxic effects in titanium dioxide-exposed humans.

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

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	<p>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>
1,2,4-TRIMETHYL BENZENE	Other Toxicity data is available for CHEMWATCH 12172 1,2,3-trimethylbenzene CHEMWATCH 2325 1,3,5-trimethylbenzene
4,4'-DIPHENYLMETHANE DIISOCYANATE (MDI)	Inhalation (human) TCLo: 0.13 ppm/30 mins Eye (rabbit): 0.10 mg moderate
MDI HOMOPOLYMER	as polymethylene polyphenyl isocyanate
P-TOLUENESULFONYL ISOCYANATE	<p>for p-toluenesulfonyl isocyanate</p> <p>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.</p> <p>.</p> <p>for p-toluenesulfonamide (PTSA):</p> <p>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.</p> <p>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.</p> <p>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.</p>
NAPHTHALENE	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
SOLVENT NAPHTHA PETROLEUM, HEAVY AROMATIC & NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT	<p>Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length, with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent than iso- or cyclo-paraffins.</p> <p>The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption, is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.</p> <p>for petroleum:</p> <p>Altered mental state, drowsiness, peripheral motor neuropathy, irreversible brain damage (so-called Petrol Sniffer's Encephalopathy), delirium, seizures, and sudden death have been reported from repeated overexposure to some hydrocarbon solvents, naphthas, and gasoline</p> <p>This product may contain benzene which is known to cause acute myeloid leukaemia and n-hexane which has been shown to metabolize to compounds which are neuropathic.</p> <p>This product contains toluene. There are indications from animal studies that prolonged exposure to high concentrations of toluene may lead to hearing loss.</p> <p>This product contains ethyl benzene and naphthalene from which there is evidence of tumours in rodents</p> <p>Carcinogenicity: Inhalation exposure to mice causes liver tumours, which are not considered relevant to humans. Inhalation exposure to rats causes kidney tumours which are not considered relevant to humans.</p> <p>Mutagenicity: There is a large database of mutagenicity studies on gasoline and gasoline blending streams, which use a wide variety of endpoints and give predominantly negative results. All in vivo studies in animals and recent studies in exposed humans (e.g. petrol service station attendants) have shown negative results in mutagenicity assays.</p> <p>Reproductive Toxicity: Repeated exposure of pregnant rats to high concentrations of toluene (around or exceeding 1000 ppm) can cause developmental effects, such as lower birth weight and developmental neurotoxicity, on the foetus. However, in a two-generation reproductive study in rats exposed to gasoline vapour condensate, no adverse effects on the foetus were observed.</p> <p>Human Effects: Prolonged/ repeated contact may cause defatting of the skin which can lead to dermatitis and may make the skin more</p>

	<p>susceptible to irritation and penetration by other materials.</p> <p>Lifetime exposure of rodents to gasoline produces carcinogenicity although the relevance to humans has been questioned. Gasoline induces kidney cancer in male rats as a consequence of accumulation of the alpha2-microglobulin protein in hyaline droplets in the male (but not female) rat kidney. Such abnormal accumulation represents lysosomal overload and leads to chronic renal tubular cell degeneration, accumulation of cell debris, mineralisation of renal medullary tubules and necrosis. A sustained regenerative proliferation occurs in epithelial cells with subsequent neoplastic transformation with continued exposure. The alpha2-microglobulin is produced under the influence of hormonal controls in male rats but not in females and, more importantly, not in humans.</p>
<p>NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT & 1,2,4-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-trimethylbenzene 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.</p> <p>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 trimethylbenzenes at 1700 ppm for 10 to 21 days.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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 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.</p> <p>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 developmental 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 trimethylbenzenes, 4-6 hours/day, 5 days/week over one generation.</p>
<p>TITANIUM DIOXIDE & 2,4'-DIPHENYLMETHANE DIISOCYANATE & 1,2,4- TRIMETHYL BENZENE & 4,4'-DIPHENYLMETHANE DIISOCYANATE (MDI) & CALCIUM OXIDE & ISOPHORONE DIISOCYANATE & MDI HOMOPOLYMER & 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>TITANIUM DIOXIDE & 2,4'-DIPHENYLMETHANE DIISOCYANATE & CARBAMIC ACID, COMPLEX ESTER</p>	<p>No significant acute toxicological data identified in literature search.</p>
<p>TITANIUM DIOXIDE & 4,4'-DIPHENYLMETHANE DIISOCYANATE (MDI)</p>	<p>The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
<p>TITANIUM DIOXIDE & NAPHTHALENE</p>	<p>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.</p> <p>WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.</p>
<p>2,4'-DIPHENYLMETHANE DIISOCYANATE & 4,4'-DIPHENYLMETHANE DIISOCYANATE (MDI) & ISOPHORONE DIISOCYANATE & CARBAMIC ACID, COMPLEX ESTER & MDI HOMOPOLYMER</p>	<p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>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.</p>

<p>2,4'-DIPHENYLMETHANE DIISOCYANATE & 4,4'-DIPHENYLMETHANE DIISOCYANATE (MDI) & ISOPHORONE DIISOCYANATE & MDI HOMOPOLYMER & P-TOLUENESULFONYL ISOCYANATE</p>	<p>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.</p>
<p>2,4'-DIPHENYLMETHANE DIISOCYANATE & 4,4'-DIPHENYLMETHANE DIISOCYANATE (MDI) & ISOPHORONE DIISOCYANATE & CARBAMIC ACID, COMPLEX ESTER & MDI HOMOPOLYMER & P-TOLUENESULFONYL ISOCYANATE</p>	<p>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.</p>
<p>2,4'-DIPHENYLMETHANE DIISOCYANATE & 4,4'-DIPHENYLMETHANE DIISOCYANATE (MDI) & ISOPHORONE DIISOCYANATE</p>	<p>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 to assume that both aromatic and aliphatic diisocyanates are respiratory sensitizers. Diisocyanates are moderate to strong dermal sensitizers in animal studies. Skin irritation studies performed on rabbits and guinea pigs indicate no difference in the effects of aromatic versus aliphatic diisocyanates. 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. There is also evidence that both aromatic and aliphatic diisocyanates are acutely toxic via the inhalation route 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'-methylenediphenyl 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. 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. 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 sensitizers. 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 sensitizer 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 sensitizers. 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 sensitizers in animal studies. There seems to be little or no difference in the level of reactivity between aromatic and aliphatic diisocyanates. 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-methylenbis-4-isocyanatocyclohexane), was found to be corrosive to the skin in guinea pigs.</p>
<p>4,4'-DIPHENYLMETHANE DIISOCYANATE (MDI) & MDI HOMOPOLYMER</p>	<p>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.</p>

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

Continued...

Duram Multithane UVHV

limestone	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	6h	Fish	4-320mg/l	4
	EC50	72h	Algae or other aquatic plants	>14mg/l	2
	LC50	96h	Fish	>165200mg/L	4
diisononyl phthalate	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	504h	Crustacea	>0.034mg/l	1
	EC50	72h	Algae or other aquatic plants	>88mg/l	2
	LC50	96h	Fish	>0.1mg/l	2
	EC50	48h	Crustacea	>0.086mg/l	1
	EC50	96h	Algae or other aquatic plants	>2.8mg/l	1
solvent naphtha petroleum, heavy aromatic	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	48h	Crustacea	0.95mg/l	1
	EC50	72h	Algae or other aquatic plants	<1mg/l	1
	LC50	96h	Fish	0.58mg/l	2
	EC50	48h	Crustacea	0.95mg/l	1
	EC50	96h	Algae or other aquatic plants	1mg/l	2
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
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
2,4'-diphenylmethane diisocyanate	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	504h	Crustacea	>=10mg/l	2
	EC50	72h	Algae or other aquatic plants	>1640mg/l	2
	LC50	96h	Fish	>1000mg/l	2
1,2,4-trimethyl benzene	Endpoint	Test Duration (hr)	Species	Value	Source
	BCF	1344h	Fish	31-207	7
	EC50(ECx)	96h	Algae or other aquatic plants	2.356mg/l	2
	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
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
calcium oxide	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>14mg/l	2
	LC50	96h	Fish	50.6mg/l	2
	EC50	48h	Crustacea	49.1mg/l	2
	EC10(ECx)	72h	Algae or other aquatic plants	>14mg/l	2
isophorone diisocyanate	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	96h	Crustacea	0.56mg/l	1
	EC50	72h	Algae or other aquatic plants	>70mg/l	2
	LC50	96h	Fish	>72mg/l	2

Continued...

Duram Multithane UVHV

	EC50	48h	Crustacea	27mg/l	2
carbamic acid, complex ester	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	29mg/l	2
	LC50	96h	Fish	199.2mg/l	2
	EC50	48h	Crustacea	193mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	12.5mg/l	2
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
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
naphthalene	Endpoint	Test Duration (hr)	Species	Value	Source
	BCF	1344h	Fish	23-146	7
	NOEC(ECx)	48h	Fish	0.013mg/L	4
	EC50	72h	Algae or other aquatic plants	~0.4~0.5mg/l	2
	LC50	96h	Fish	0.51mg/l	4
	EC50	48h	Crustacea	1.09-3.4mg/l	4

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*

On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and/or observed environmental fate and behaviour, the material may present a danger, immediate or long-term and/or delayed, to the structure and/or functioning of natural ecosystems. Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment. Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters. Wastes resulting from use of the product must be disposed of on site or at approved waste sites. **DO NOT discharge into sewer or waterways.**

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
diisononyl phthalate	HIGH	HIGH
titanium dioxide	HIGH	HIGH
2,4'-diphenylmethane diisocyanate	HIGH	HIGH
1,2,4-trimethyl benzene	LOW (Half-life = 56 days)	LOW (Half-life = 0.67 days)
4,4'-diphenylmethane diisocyanate (MDI)	LOW (Half-life = 1 days)	LOW (Half-life = 0.24 days)
isophorone diisocyanate	HIGH	HIGH
p-toluenesulfonyl isocyanate	HIGH	HIGH
naphthalene	HIGH (Half-life = 258 days)	LOW (Half-life = 1.23 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
diisononyl phthalate	LOW (BCF = 183.8)
solvent naphtha petroleum, heavy aromatic	LOW (BCF = 159)
titanium dioxide	LOW (BCF = 10)
2,4'-diphenylmethane diisocyanate	HIGH (LogKOW = 5.4481)
1,2,4-trimethyl benzene	LOW (BCF = 275)
4,4'-diphenylmethane diisocyanate (MDI)	LOW (BCF = 15)
isophorone diisocyanate	HIGH (LogKOW = 4.7519)
p-toluenesulfonyl isocyanate	LOW (LogKOW = 2.3424)
naphthalene	HIGH (BCF = 18000)

Mobility in soil

Duram Multithane UVHV

Ingredient	Mobility
diisononyl phthalate	LOW (KOC = 467200)
titanium dioxide	LOW (KOC = 23.74)
2,4'-diphenylmethane diisocyanate	LOW (KOC = 384000)
1,2,4-trimethyl benzene	LOW (KOC = 717.6)
4,4'-diphenylmethane diisocyanate (MDI)	LOW (KOC = 376200)
isophorone diisocyanate	LOW (KOC = 36450)
p-toluenesulfonyl isocyanate	LOW (KOC = 882.1)
naphthalene	LOW (KOC = 1837)

SECTION 13 Disposal considerations

Waste treatment methods

<p>Product / Packaging disposal</p>	<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. <p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> ▶ Reduction ▶ Reuse ▶ Recycling ▶ Disposal (if all else fails) <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> ▶ 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. ▶ DO NOT recycle spilled material. ▶ Consult State Land Waste Management Authority for disposal. ▶ Neutralise spill material carefully and decontaminate empty containers and spill residues with 10% ammonia solution plus detergent or a proprietary decontaminant prior to disposal. ▶ DO NOT seal or stopper drums being decontaminated as CO2 gas is generated and may pressurise containers. ▶ Puncture containers to prevent re-use. ▶ Bury or incinerate residues at an approved site.
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SECTION 14 Transport information

Labels Required

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

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

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

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

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

Not Applicable

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

Product name	Group
limestone	Not Available
diisononyl phthalate	Not Available
solvent naphtha petroleum, heavy aromatic	Not Available
naphtha petroleum, light aromatic solvent	Not Available
titanium dioxide	Not Available
2,4'-diphenylmethane diisocyanate	Not Available
1,2,4-trimethyl benzene	Not Available

Duram Multithane UVHV

Product name	Group
4,4'-diphenylmethane diisocyanate (MDI)	Not Available
calcium oxide	Not Available
isophorone diisocyanate	Not Available
carbamic acid, complex ester	Not Available
MDI homopolymer	Not Available
p-toluenesulfonyl isocyanate	Not Available
naphthalene	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
limestone	Not Available
diisononyl phthalate	Not Available
solvent naphtha petroleum, heavy aromatic	Not Available
naphtha petroleum, light aromatic solvent	Not Available
titanium dioxide	Not Available
2,4'-diphenylmethane diisocyanate	Not Available
1,2,4-trimethyl benzene	Not Available
4,4'-diphenylmethane diisocyanate (MDI)	Not Available
calcium oxide	Not Available
isophorone diisocyanate	Not Available
carbamic acid, complex ester	Not Available
MDI homopolymer	Not Available
p-toluenesulfonyl isocyanate	Not Available
naphthalene	Not Available

SECTION 15 Regulatory information

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

<p>limestone is found on the following regulatory lists</p> <ul style="list-style-type: none"> Australian Inventory of Industrial Chemicals (AIIC) 	
<p>diisononyl phthalate is found on the following regulatory lists</p> <ul style="list-style-type: none"> Australian Inventory of Industrial Chemicals (AIIC) 	<ul style="list-style-type: none"> Chemical Footprint Project - Chemicals of High Concern List
<p>solvent naphtha petroleum, heavy aromatic is found on the following regulatory lists</p> <ul style="list-style-type: none"> Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals Australian Inventory of Industrial Chemicals (AIIC) 	<ul style="list-style-type: none"> International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
<p>naphtha petroleum, light aromatic solvent is found on the following regulatory lists</p> <ul style="list-style-type: none"> Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals Australian Inventory of Industrial Chemicals (AIIC) 	<ul style="list-style-type: none"> Chemical Footprint Project - Chemicals of High Concern List International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
<p>titanium dioxide is found on the following regulatory lists</p> <ul style="list-style-type: none"> 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 	<ul style="list-style-type: none"> 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)
<p>2,4'-diphenylmethane diisocyanate is found on the following regulatory lists</p> <ul style="list-style-type: none"> Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring 	<ul style="list-style-type: none"> Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6 Australian Inventory of Industrial Chemicals (AIIC)
<p>1,2,4-trimethyl benzene is found on the following regulatory lists</p> <ul style="list-style-type: none"> Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5 	<ul style="list-style-type: none"> Australian Inventory of Industrial Chemicals (AIIC)
<p>4,4'-diphenylmethane diisocyanate (MDI) is found on the following regulatory lists</p> <ul style="list-style-type: none"> Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6 	<ul style="list-style-type: none"> Australian Inventory of Industrial Chemicals (AIIC) International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

calcium oxide is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

isophorone diisocyanate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

Australian Inventory of Industrial Chemicals (AIIC)

carbamic acid, complex ester is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

MDI homopolymer is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

p-toluenesulfonyl isocyanate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

Australian Inventory of Industrial Chemicals (AIIC)

naphthalene is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C

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

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (carbamic acid, complex ester)
Canada - DSL	Yes
Canada - NDSL	No (diisononyl phthalate; solvent naphtha petroleum, heavy aromatic; naphtha petroleum, light aromatic solvent; 2,4'-diphenylmethane diisocyanate; 1,2,4-trimethyl benzene; 4,4'-diphenylmethane diisocyanate (MDI); calcium oxide; isophorone diisocyanate; carbamic acid, complex ester; MDI homopolymer; p-toluenesulfonyl isocyanate; naphthalene)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (solvent naphtha petroleum, heavy aromatic; carbamic acid, complex ester)
Korea - KECI	No (carbamic acid, complex ester)
New Zealand - NZIoC	Yes
Philippines - PICCS	No (carbamic acid, complex ester)
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (2,4'-diphenylmethane diisocyanate; carbamic acid, complex ester; MDI homopolymer; p-toluenesulfonyl isocyanate)
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing (see specific ingredients in brackets)

SECTION 16 Other information

Revision Date	01/11/2019
Initial Date	25/01/2017

SDS Version Summary

Version	Date of Update	Sections Updated
3.1.1.1	31/01/2017	Acute Health (eye), Classification, Environmental, Exposure Standard, Toxicity and Irritation (Other)
4.1.1.1	01/11/2019	One-off system update. NOTE: This may or may not change the GHS classification
4.1.2.1	26/04/2021	Regulation Change
4.1.3.1	03/05/2021	Regulation Change
4.1.4.1	06/05/2021	Regulation Change
4.1.5.1	10/05/2021	Regulation Change
4.1.5.2	30/05/2021	Template Change
4.1.5.3	04/06/2021	Template Change
4.1.5.4	05/06/2021	Template Change
4.1.6.4	07/06/2021	Regulation Change
4.1.6.5	09/06/2021	Template Change

Version	Date of Update	Sections Updated
4.1.6.6	11/06/2021	Template Change
4.1.6.7	15/06/2021	Template Change
4.1.7.7	17/06/2021	Regulation Change
4.1.8.7	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
 AIC: 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
 KECl: Korea Existing Chemicals Inventory
 NZIoC: New Zealand Inventory of Chemicals
 PICCS: Philippine Inventory of Chemicals and Chemical Substances
 TSCA: Toxic Substances Control Act
 TCSI: Taiwan Chemical Substance Inventory
 INSQ: Inventario Nacional de Sustancias Químicas
 NCI: National Chemical Inventory
 FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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