Duroproof PUM grey 15L

Durotech Industries

Chemwatch Hazard Alert Code: **2**

Chemwatch: **5245-84**
Version No: **2.1.1.1**
Safety Data Sheet according to WHS and ADG requirements

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SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	Duroproof PUM grey 15L
Synonyms	Duroproof PUM grey, polyurethane waterproofing membrane for non-exposed areas
Other means of identification	Not Available

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

Relevant identified uses	Use according to manufacturer's directions.

Details of the supplier of the safety data sheet

Registered company name	Durotech Industries
Address	14 Essex Street Minto NSW 2566 Australia
Telephone	02 9603 1177
Fax	02 9475 5059
Website	www.durotechindustries.com.au
Email	accounts@durotechindustries.com.au

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	0421 670 636
Other emergency telephone numbers	Not Available

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

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

COMBUSTIBLE LIQUID, regulated for storage purposes only

Poisons Schedule	S6
Classification [1]	Flammable Liquid Category 4, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Respiratory Sensitizer Category 1, Skin Sensitizer Category 1, Carcinogenicity Category 2, Reproductive Toxicity Category 2, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Acute Aquatic Hazard Category 2
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HSIS ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI*

Label elements

GHS label elements	
SIGNAL WORD	**DANGER**

Hazard statement(s)

H227	Combustible liquid
H315	Causes skin irritation.
H319	Causes serious eye irritation.
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H317	May cause an allergic skin reaction.
H351	Suspected of causing cancer.

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H361	Suspected of damaging fertility or the unborn child.
H335	May cause respiratory irritation.
H401	Toxic to aquatic life

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat/sparks/open flames/hot surfaces. - 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/face protection.
P281	Use personal protective equipment as required.
P285	In case of inadequate ventilation wear respiratory protection.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.
P308+P313	IF exposed or concerned: Get medical advice/attention.
P342+P311	If experiencing respiratory symptoms: Call a POISON CENTER or doctor/physician.
P362	Take off contaminated clothing and wash before reuse.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam for extinction.
P302+P352	IF ON SKIN: Wash with plenty of soap and 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 or doctor/physician 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.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

P501	Dispose of contents/container in accordance with local regulations.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
Not Available	<60	filler
68515-48-0	<30	diisononyl phthalate
154099-10-2	<30	MDI/ castor oil/ glycerol, propoxylated
101-68-8	<5	4,4'-diphenylmethane diisocyanate (MDI)
13463-67-7	<5	titanium dioxide
25686-28-6	<5	MDI homopolymer
5873-54-1	<5	2,4'-diphenylmethane diisocyanate
4083-64-1	<1	p-toluenesulfonyl isocyanate
	balance	Ingredients determined not to be hazardous

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Wash out immediately with fresh running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation. For thermal burns:

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	▸ Decontaminate area around burn. ▸ Consider the use of cold packs and topical antibiotics. For first-degree burns (affecting top layer of skin) ▸ Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides. ▸ Use compresses if running water is not available. ▸ Cover with sterile non-adhesive bandage or clean cloth. ▸ Do NOT apply butter or ointments; this may cause infection. ▸ Give over-the counter pain relievers if pain increases or swelling, redness, fever occur. For second-degree burns (affecting top two layers of skin) ▸ Cool the burn by immerse in cold running water for 10-15 minutes. ▸ Use compresses if running water is not available. ▸ Do NOT apply ice as this may lower body temperature and cause further damage. ▸ Do NOT break blisters or apply butter or ointments; this may cause infection. ▸ Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape. To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort): ▸ Lay the person flat. ▸ Elevate feet about 12 inches. ▸ Elevate burn area above heart level, if possible. ▸ Cover the person with coat or blanket. ▸ Seek medical assistance. For third-degree burns Seek immediate medical or emergency assistance. In the mean time: ▸ Protect burn area cover loosely with sterile, nonstick bandage or, for large areas, a sheet or other material that will not leave lint in wound. ▸ Separate burned toes and fingers with dry, sterile dressings. ▸ Do not soak burn in water or apply ointments or butter; this may cause infection. ▸ To prevent shock see above. ▸ For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway. ▸ Have a person with a facial burn sit up. ▸ Check pulse and breathing to monitor for shock until emergency help arrives.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay. 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.
Ingestion	▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Seek medical advice.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.
For sub-chronic and chronic exposures to isocyanates:

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

[Ellenhorn and Barceloux; Medical Toxicology]

NOTE: Isocyanates cause airway restriction in naive individuals with the degree of response dependant on the concentration and duration of exposure. They induce smooth muscle contraction which leads to bronchoconstrictive episodes. Acute changes in lung function, such as decreased FEV1, may not represent sensitivity.
[Karol & Jin, Frontiers in Molecular Toxicology, pp 56-61, 1992]
Personnel who work with isocyanates, isocyanate prepolymers or polyisocyanates should have a pre-placement medical examination and periodic examinations thereafter, including a pulmonary function test. Anyone with a medical history of chronic respiratory disease, asthmatic or bronchial attacks, indications of allergic responses, recurrent eczema or sensitisation conditions of the skin should not handle or work with isocyanates. Anyone who develops chronic respiratory distress when working with isocyanates should be removed from exposure and examined by a physician. Further exposure must be avoided if a sensitivity to isocyanates or polyisocyanates has developed.

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

- Small quantities of water in contact with hot liquid may react violently with generation of a large volume of rapidly expanding hot sticky semi-solid foam.
- Presents additional hazard when fire fighting in a confined space.
- Cooling with flooding quantities of water reduces this risk.
- Water spray or fog may cause frothing and should be used in large quantities.
- Foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.
- Water spray or fog - Large fires only.

Special hazards arising from the substrate or mixture

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

Advice for firefighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Use water delivered as a fine spray to control fire and cool adjacent area. ▸ Avoid spraying water onto liquid pools. ▸ **DO NOT** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	▸ Combustible. ▸ Moderate fire hazard when exposed to heat or flame. ▸ When heated to high temperatures decomposes rapidly generating vapour which pressures and may then rupture containers with release of flammable and highly toxic isocyanate vapour. ▸ Burns with acrid black smoke and poisonous fumes. ▸ Combustion yields traces of highly toxic hydrogen cyanide HCN, plus toxic nitrogen oxides NOx and carbon monoxide. Combustion products include: , carbon dioxide (CO2) , isocyanates , and minor amounts of , hydrogen cyanide , nitrogen oxides (NOx) , metal oxides , other pyrolysis products typical of burning organic material. May emit corrosive fumes. When heated at high temperatures many isocyanates decompose rapidly generating a vapour which pressurises containers, possibly to the point of rupture. Release of toxic and/or flammable isocyanate vapours may then occur
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	Environmental hazard - contain spillage. ▸ Remove all ignition sources. ▸ Clean up all spills immediately. ▸ Avoid breathing vapours and contact with skin and eyes. ▸ Control personal contact with the substance, by using protective equipment. ▸ Contain and absorb spill with sand, earth, inert material or vermiculite. ▸ Wipe up. ▸ Place in a suitable, labelled container for waste disposal.
Major Spills	Environmental hazard - contain spillage. ▸ Liquid Isocyanates and high isocyanate vapour concentrations will penetrate seals on self contained breathing apparatus - SCBA should be used inside encapsulating suit where this exposure may occur. 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:**

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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, 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.

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
- 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

for commercial quantities of isocyanates:

- Isocyanates should be stored in adequately bunded 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.

- Store in original containers.
- Keep containers securely sealed.
- No smoking, naked lights or ignition sources.
- Store in a cool, dry, well-ventilated area.
- Store away from incompatible materials and foodstuff containers.
- Protect containers against physical damage and check regularly for leaks.
- Observe manufacturer's storage and handling recommendations contained within this SDS.

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Conditions for safe storage, including any incompatibilities

Suitable container	▸ Metal can or drum ▸ Packaging as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▸ Avoid reaction with water, alcohols and detergent solutions. ▸ Isocyanates and thioisocyanates are incompatible with many classes of compounds, reacting exothermically to release toxic gases. Reactions with amines, strong bases, aldehydes, alcohols, alkali metals, ketones, mercaptans, strong oxidisers, hydrides, phenols, and peroxides can cause vigorous releases of heat. Acids and bases initiate polymerisation reactions in these materials. ▸ Isocyanates easily form adducts with carbodiimides, isothiocyanates, ketenes, or with substrates containing activated CC or CN bonds. ▸ Some isocyanates react with water to form amines and liberate carbon dioxide. This reaction may also generate large volumes of foam and heat. Foaming in confined spaces may produce pressure in confined spaces or containers. Gas generation may pressurise drums to the point of rupture. ▸ Do NOT reseal container if contamination is expected ▸ Open all containers with care ▸ Base-catalysed reactions of isocyanates with alcohols should be carried out in inert solvents. Such reactions in the absence of solvents often occur with explosive violence, ▸ Isocyanates will attack and embrittle some plastics and rubbers. ▸ A range of exothermic decomposition energies for isocyanates is given as 20-30 kJ/mol. ▸ The relationship between energy of decomposition and processing hazards has been the subject of discussion; it is suggested that values of energy released per unit of mass, rather than on a molar basis (J/g) be used in the assessment. ▸ For example, in "open vessel processes" (with man-hole size openings, in an industrial setting), substances with exothermic decomposition energies below 500 J/g are unlikely to present a danger, whilst those in "closed vessel processes" (opening is a safety valve or bursting disk) present some danger where the decomposition energy exceeds 150 J/g. BRETHERICK: Handbook of Reactive Chemical Hazards, 4th Edition ▸ 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	4,4'-diphenylmethane diisocyanate (MDI)	Isocyanates, all (as-NCO)	0.02 mg/m3	0.07 mg/m3	Not Available	Sen
Australia Exposure Standards	titanium dioxide	Titanium dioxide	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	2,4'-diphenylmethane diisocyanate	Isocyanates, all (as-NCO)	0.02 mg/m3	0.07 mg/m3	Not Available	Sen
Australia Exposure Standards	p-toluenesulfonyl isocyanate	Isocyanates, all (as-NCO)	0.02 mg/m3	0.07 mg/m3	Not Available	Sen

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
4,4'-diphenylmethane diisocyanate (MDI)	Methylene diphenyl diisocyanate; (Diphenylmethane diisocyanate; MDI)	0.45 mg/m3	Not Available	Not Available
4,4'-diphenylmethane diisocyanate (MDI)	Methylenebis(isocyanato-benzene), 1,1'-; (Diphenyl methane diisocyanate)	29 mg/m3	40 mg/m3	240 mg/m3
titanium dioxide	Titanium oxide; (Titanium dioxide)	30 mg/m3	330 mg/m3	2,000 mg/m3
2,4'-diphenylmethane diisocyanate	Isocyanate-bearing waste (as CNs N.O.S.)	6 mg/m3	8.3 mg/m3	50 mg/m3
p-toluenesulfonyl isocyanate	Isocyanate-bearing waste (as CNs N.O.S.)	6 mg/m3	8.3 mg/m3	50 mg/m3

Ingredient	Original IDLH	Revised IDLH
filler	Not Available	Not Available
diisononyl phthalate	Not Available	Not Available
MDI/ castor oil/ glycerol, propoxylated	Not Available	Not Available
4,4'-diphenylmethane diisocyanate (MDI)	100 mg/m3	75 mg/m3
titanium dioxide	N.E. mg/m3 / N.E. ppm	5,000 mg/m3
MDI homopolymer	Not Available	Not Available
2,4'-diphenylmethane diisocyanate	Not Available	Not Available
p-toluenesulfonyl isocyanate	Not Available	Not Available

MATERIAL DATA

Exposure controls

Appropriate engineering controls	▸ All processes in which isocyanates are used should be enclosed wherever possible. ▸ Total enclosure, accompanied by good general ventilation, should be used to keep atmospheric concentrations below the relevant exposure standards. ▸ If total enclosure of the process is not feasible, local exhaust ventilation may be necessary. Local exhaust ventilation is essential where lower molecular weight isocyanates (such as TDI or HDI) is used or where isocyanate or polyurethane is sprayed. ▸ Where other isocyanates or pre-polymers are used and aerosol formation cannot occur, local exhaust ventilation may not be necessary if the atmospheric concentration can be kept below the relevant exposure standards. ▸ Where local exhaust ventilation is installed, exhaust vapours should not be vented to the exterior in such a manner as to create a hazard. Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk.

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Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
Employers may need to use multiple types of controls to prevent employee overexposure.

- Spraying of material or material in admixture with other components must be carried out in conditions conforming to local state regulations (AS/NZS 4114, UNI EN 12215:2010, ANSI/AIHA Z9.3–2007 or national equivalent).
- Local exhaust ventilation with full face positive-pressure air supplied breathing apparatus (hood or helmet type) is required.
- Spraying should be performed in a spray booth fitted with an effective exhaust system which complies with local environmental legislation.
- The spray booth area must be isolated from unprotected personnel whilst spraying is in progress and until all spraying mist has cleared.

NOTE: Isocyanate vapours will not be adequately absorbed by organic vapour respirators. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

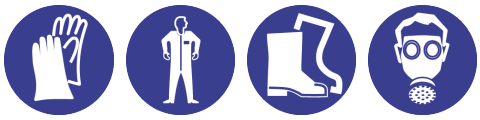
Type of Contaminant:	Air Speed:
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only	2: Contaminants of high toxicity
3: Intermittent, low production.	3: High production, heavy use
4: Large hood or large air mass in motion	4: Small hood-local control only

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 4-10 m/s (800-2000 f/min.) for extraction of solvents generated by spraying at a point 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

Personal protection



Eye and face protection

- Safety glasses with side shields.
- Chemical goggles.
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]

Skin protection

See Hand protection below

Hands/feet protection

NOTE:

- The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.
- Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturizer is recommended.
Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:

- frequency and duration of contact,
- chemical resistance of glove material,
- glove thickness and
- dexterity

Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).

- When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
- Contaminated gloves should be replaced.

For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.
It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.
Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task.
Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:

- Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.
- Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential

Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

- Do NOT wear natural rubber (latex gloves).
- Isocyanate resistant materials include Teflon, Viton, nitrile rubber and some PVA gloves.
- Protective gloves and overalls should be worn as specified in the appropriate national standard.
- Contaminated garments should be removed promptly and should not be re-used until they have been decontaminated.

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	▸ NOTE: Natural rubber, neoprene, PVC can be affected by isocyanates ▸ **DO NOT** use skin cream unless necessary and then use only minimum amount. ▸ Isocyanate vapour may be absorbed into skin cream and this increases hazard.
Body protection	See Other protection below
Other protection	All employees working with isocyanates must be informed of the hazards from exposure to the contaminant and the precautions necessary to prevent damage to their health. They should be made aware of the need to carry out their work so that as little contamination as possible is produced, and of the importance of the proper use of all safeguards against exposure to themselves and their fellow workers. Adequate training, both in the proper execution of the task and in the use of all associated engineering controls, as well as of any personal protective equipment, is essential. Employees exposed to contamination hazards should be educated in the need for, and proper use of, facilities, clothing and equipment and thereby maintain a high standard of personal cleanliness. Special attention should be given to ensuring that all personnel understand instructions, especially newly recruited employees and those with local-language difficulties, where they are known. ▸ Overalls. ▸ P.V.C. apron. ▸ Barrier cream. ▸ Skin cleansing cream. ▸ Eye wash unit.
Thermal hazards	Not Available

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:
Duroproof PUM grey 15L

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

* 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.
For spraying or operations which might generate aerosols:
Full face respirator with supplied air.

- 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 nationals 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; does not mix 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

Continued...

Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	78	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Combustible.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water (g/L)	Immiscible	**pH as a solution (1%)**	Not Applicable
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 STABILITY AND REACTIVITY

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

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual. A significant number of individuals exposed to mixed trimethylbenzenes complained of nervousness, tension, anxiety and asthmatic bronchitis. Peripheral blood showed a tendency to hypochromic anaemia and a deviation from normal in coagulability of the blood. Hydrocarbon concentrations ranged from 10 to 60 ppm. Contamination of the mixture with benzene may have been responsible for the blood dyscrasias. High concentrations of mesitylene vapour (5000 to 9000 ppm) caused central nervous system depression in mice. Similar exposures of pseudocumene also produced evidence of CNS involvement. The vapour/mist may be highly irritating to the upper respiratory tract and lungs; the response may be severe enough to produce bronchitis 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 for several hours after exposure. Sensitized people can react to very low doses, and should not be allowed to work in situations allowing exposure to this material. Continued exposure of sensitised persons may lead to possible long term respiratory impairment. Inhalation hazard is increased at higher temperatures. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal. The acute toxicity of inhaled alkylbenzenes is best described by central nervous system depression. As a rule, these compounds may also act as general anaesthetics. Systemic poisoning produced by general anaesthesia is characterised by lightheadedness, nervousness, apprehension, euphoria, confusion, dizziness, drowsiness, tinnitus, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness and respiratory depression and arrest. Cardiac arrest may result from cardiovascular collapse. Bradycardia, and hypotension may also be produced. Inhaled alkylbenzene vapours cause death in animals at air levels that are relatively similar (typically LC50s are in the range 5000 -8000 ppm for 4 to 8 hour exposures). It is likely that acute inhalation exposure to alkylbenzenes resembles that to general anaesthetics. Alkylbenzenes are not generally toxic other than at high levels of exposure. This may be because their metabolites have a low order of toxicity and are easily excreted. There is little or no evidence to suggest that metabolic pathways can become saturated leading to spillover to alternate pathways. Nor is there evidence that toxic reactive intermediates, which may produce subsequent toxic or mutagenic effects, are formed
Ingestion	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

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	potentiate the biological effects of substances such as antibiotics, thiamine and sulfonamides. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. The material may accentuate any pre-existing dermatitis condition Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	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.
Chronic	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 either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. 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. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Persons with a history of asthma or other respiratory problems or are known to be sensitised, should not be engaged in any work involving the handling of isocyanates. [CCTRADE-Bayer, APMF] 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. 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. 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'.

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Endocrine disruptors such as phthalates can be add to the effects of other endocrine disruptors, so even very small amounts can interact with other chemicals to have cumulative, adverse "cocktail effects"
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
Studies conducted on mice exposed to phthalates in utero did not result in metabolic disorder in adults. However, "At least one phthalate, monoethyhexyl 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 carbohdrate metabolism.
Prenatal exposure to phthalates may affect children's mental, motor and behavioral development during the preschool year.
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.
Several findings point to a statistically significant correlation between urine phthalate concentrations in children and symptoms of attention deficit hyperactivity disorder (ADHD)

Limited evidence of a carcinogenic effect.

Duroproof PUM grey 15L	TOXICITY	IRRITATION
	Not Available	Not Available

diisononyl phthalate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >3160 mg/kg[1]	Not Available
	Oral (rat) LD50: >10000 mg/kg[2]	

MDI/ castor oil/ glycerol, propoxylated	TOXICITY	IRRITATION
	Not Available	Not Available

4,4'-diphenylmethane diisocyanate (MDI)	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >6200 mg/kg[2]	Dermal Sensitiser *
	Inhalation (rat) LC50: 0.49 mg/l/4hr[1]	Skin (rabbit): 500 mg /24 hours
	Oral (rat) LD50: >2000 mg/kg[1]	

titanium dioxide	TOXICITY	IRRITATION
	Inhalation (rat) LC50: >2.28 mg/l/4hr[1]	Skin (human): 0.3 mg /3D (int)-mild *
	Inhalation (rat) LC50: >3.56 mg/l/4hr[1]	
	Inhalation (rat) LC50: >6.82 mg/l/4hr[1]	
	Inhalation (rat) LC50: 3.43 mg/l/4hr[1]	
	Inhalation (rat) LC50: 5.09 mg/l/4hr[1]	
	Oral (rat) LD50: >2000 mg/kg[1]	

MDI homopolymer	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >9400 mg/kg[1]	Not Available
	Inhalation (rat) LC50: 0.49 mg/l/4hr[1]	
	Oral (rat) LD50: >5000 mg/kg[1]	

2,4'-diphenylmethane diisocyanate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >9400 mg/kg[1]	Not Available
	Oral (rat) LD50: >2000 mg/kg[1]	

p-toluenesulfonyl isocyanate	TOXICITY	IRRITATION
	Inhalation (rat) LC50: >640 ppm/1hr[2]	Not Available
	Oral (rat) LD50: 2234 mg/kg[2]	

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

DIISONONYL PHTHALATE

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.
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.
High molecular weight phthalates are used nearly exclusively as plasticisers of PVC.
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 hepatocarcinogencity 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.
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

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	increasing molecular weight. Studies on HMWPEs indicate that they are rapidly metabolised in the gastrointestinal tract to the corresponding monoester, absorbed and excreted primarily in the urine. **Acute toxicity:** The available data on phthalates spanning the carbon range from C8-C13 indicate that phthalate esters in the high molecular 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 (PPARa;), and that levels of PPARa 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 PPARa-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 71 1P 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/ rnl, 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. [Huls] 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.
MDI/ CASTOR OIL/ GLYCEROL, PROPOXYLATED	Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air. Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture . On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing. Allergic Contact Dermatitis—Formation, Structural Requirements,and Reactivity of Skin Sensitizers. Ann-Therese Karlberg et al; Chem. Res. Toxicol.2008,21,53-69
4,4'-DIPHENYLMETHANE DIISOCYANATE (MDI)	Inhalation (human) TCLo: 0.13 ppm/30 mins Eye (rabbit): 0.10 mg moderate

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TITANIUM DIOXIDE	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. 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 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. 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. 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. **Animal carcinogenicity data** 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. 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. 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. 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. **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. * IUCLID
MDI HOMOPOLYMER	as polymethylene polyphenyl isocyanate
P-TOLUENESULFONYL ISOCYANATE	for p-toluenesulfonyl isocyanate The acute oral toxicity (LD50) of PTSI is 2600 mg/kg. Based on the rapid hydrolysis of PTSI to PTSA (and carbon dioxide), repeated dose, reproductive, and developmental toxicity, as well as genotoxicity are best described by PTSA. . for p-toluenesulfonamide (PTSA): PTSA was studied for oral toxicity in rats in a single dose toxicity test at doses of 889, 1333, 2000 and 3000 mg/kg in females and 2000 mg/kg in males, and in an OECD combined repeat dose and reproductive/developmental toxicity screening test at doses of 0, 120, 300 and 750 mg/kg/day in both sexes .PTSA was also tested for mutagenicity with assays for reverse mutation in bacteria and chromosomal aberrations in cultured Chinese hamster (CHL) cells. The single dose toxicity test revealed LD50 values of above 2000 mg/kg for both sexes. For repeat dose toxicity caused, daily administration of 300 mg/kg or more in males and females displayed an increase in salivation and a reduction in body weight gain, as well as a suppression of food consumption. No compound-related deaths were observed. Haematuria was observed within 3 days administration of 750 mg/kg in 4/13 males. Hematological examination and blood chemistry measurements in males showed a decrease in white blood cell count with an increase in lymphocyte count, increases in blood urea nitrogen and chloride, and slight elevation in GOT in medium and high dose groups and a decrease in potassium concentration,and increased GPT levels in the high dose group. Histopathological examination showed cytoplasmic changes in the epithelium of the urinary bladder in both sexes and an accelerated involution in the thymus especially in females. Signs of toxicity, such as salivation and urinary bladder changes, were observed in animals given 120 mg/kg and above. The NOEL for repeat dose toxicity was less than 120 mg/kg/day. For reproductive/developmental toxicity, females given 750 mg/kg/day demonstrated possible delivery or lactation state dysfunction and developmental suppression of embryos. NOELs for reproductive performance and offspring development were both 300 mg/kg/day. No teratogenic effects were observed. The mutagenicity tests performed were all negative. PTSA was not mutagenic for bacteria either with or without an exogenous metabolic activation system up to 5000 ug/plate. No chromosomal aberrations or polyploidy were induced in CHL cells up to 1.7 mg/ml with metabolic activation and 1.3 mg/ml without metabolic activation.
MDI/ CASTOR OIL/ GLYCEROL, PROPOXYLATED & 4,4'-DIPHENYLMETHANE DIISOCYANATE (MDI) & MDI HOMOPOLYMER & 2,4'-DIPHENYLMETHANE DIISOCYANATE	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.
MDI/ CASTOR OIL/ GLYCEROL, PROPOXYLATED & 4,4'-DIPHENYLMETHANE DIISOCYANATE (MDI) & 2,4'-DIPHENYLMETHANE DIISOCYANATE & P-TOLUENESULFONYL	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.

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ISOCYANATE	Unprotected or sensitised persons should not be allowed to work in situations allowing exposure to this material.
MDI/ CASTOR OIL/ GLYCEROL, PROPOXYLATED & MDI HOMOPOLYMER & 2,4'-DIPHENYLMETHANE DIISOCYANATE	No significant acute toxicological data identified in literature search.
4,4'-DIPHENYLMETHANE DIISOCYANATE (MDI) & 2,4'-DIPHENYLMETHANE DIISOCYANATE & P-TOLUENESULFONYL ISOCYANATE	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.
4,4'-DIPHENYLMETHANE DIISOCYANATE (MDI) & 2,4'-DIPHENYLMETHANE DIISOCYANATE & P-TOLUENESULFONYL ISOCYANATE	Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens).
4,4'-DIPHENYLMETHANE DIISOCYANATE (MDI) & 2,4'-DIPHENYLMETHANE DIISOCYANATE & P-TOLUENESULFONYL ISOCYANATE	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.
4,4'-DIPHENYLMETHANE DIISOCYANATE (MDI) & 2,4'-DIPHENYLMETHANE DIISOCYANATE & P-TOLUENESULFONYL ISOCYANATE	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.
4,4'-DIPHENYLMETHANE DIISOCYANATE (MDI) & TITANIUM DIOXIDE	The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
4,4'-DIPHENYLMETHANE DIISOCYANATE (MDI) & 2,4'-DIPHENYLMETHANE DIISOCYANATE	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 sensitisers. Diisocyanates are moderate to strong dermal sensitisers in animal studies. Skin irritation studies performed on rabbits and guinea pigs indicate no difference in the effects of aromatic versus aliphatic diisocyanates. 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 sensitisers. Aliphatic diisocyanates are generally not active in animal models for respiratory sensitization. However, HDI and possibly isophorone diisocyanate (IPDI), are reported to be associated with respiratory sensitization in humans. Symptoms resulting from occupational exposure to HDI include shortness of breath, increased bronchoconstriction reaction to histamine challenges, asthmatic reactions, wheezing and coughing. Two case reports of human exposure to IPDI by inhalation suggest IPDI is a respiratory sensitiser in humans. In view of the information from case reports in humans, it would be prudent at this time to assume that both aromatic and aliphatic diisocyanates are respiratory sensitisers. Studies in both human and mice using TDI, HDI, MDI and dicyclohexylmethane-4,4'-diisocyanate (HMDI) suggest cross-reactivity with the other diisocyanates, irrespective of whether the challenge compound was an aliphatic or aromatic diisocyanate. Diisocyanates are moderate to strong dermal sensitisers in animal studies. There seems to be little or no difference in the level of reactivity between aromatic and aliphatic diisocyanates. **Dermal Irritation:** Skin irritation studies performed on rabbits and guinea pigs indicate no difference in the effects of aromatic versus aliphatic diisocyanates. The level of irritation ranged from slightly to severely irritating to the skin. One chemical, hydrogenated MDI (1,1-methylenebis-4-isocyanatocyclohexane), was found to be corrosive to the skin in guinea pigs.
4,4'-DIPHENYLMETHANE DIISOCYANATE (MDI) & MDI HOMOPOLYMER	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.

Acute Toxicity	⊘	**Carcinogenicity**	✔
Skin Irritation/Corrosion	✔	**Reproductivity**	✔

Continued...

Serious Eye Damage/Irritation	✔	**STOT - Single Exposure**	✔
Respiratory or Skin sensitisation	✔	**STOT - Repeated Exposure**	⊘
Mutagenicity	⊘	**Aspiration Hazard**	⊘

Legend: ✘ *– Data available but does not fill the criteria for classification*
✔ *– Data available to make classification*
⊘ *– Data Not Available to make classification*

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
diisononyl phthalate	LC50	96	Fish	>0.1mg/L	2
diisononyl phthalate	EC50	48	Crustacea	>0.06mg/L	2
diisononyl phthalate	EC50	96	Algae or other aquatic plants	>2.8mg/L	1
diisononyl phthalate	EC50	504	Crustacea	>0.0036mg/L	2
diisononyl phthalate	NOEC	504	Crustacea	0.0036mg/L	2
4,4'-diphenylmethane diisocyanate (MDI)	LC50	96	Fish	>0.500mg/L	6
titanium dioxide	LC50	96	Fish	9.214mg/L	3
titanium dioxide	EC50	48	Crustacea	>10mg/L	2
titanium dioxide	EC50	72	Algae or other aquatic plants	5.83mg/L	4
titanium dioxide	EC20	72	Algae or other aquatic plants	1.81mg/L	4
titanium dioxide	NOEC	336	Fish	0.089mg/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.
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.
Hydrolysis would represents the primary fate mechanism for the majority of the commercial isocyanate monomers, but, is tempered somewhat by the lack of water solubility. In the absence of hydrolysis, sorption to solids (e.g., sludge and sediments) will be the primary mechanism of removal. Biodegradation is minimal for most compounds and volatilisation is negligible. Atmospheric degradation is not expected with removal from air occurring by washout or dry deposition. Volatilisation from surface waters (e.g., lakes and rivers) is expected to take years. In wastewater treatment this process is not expected to be significant.
Review of the estimated properties of the isocyanates suggest that sorption is the primary removal mechanism in the ambient environment and in wastewater treatment in the absence of significant hydrolysis. Sorption to solids in wastewater treatment is considered strong to very strong for most compounds. Sorption to sediments and soils in the ambient environment is very strong in most instances. Migration to groundwater and surface waters is not expected due to sorption or hydrolysis.
Hydrolysis of the N=C=O will occur in less than hours in most instances and within minutes for more than 90% of the commercial isocyanates. However, the low to very low solubility of these substances will generally lessen the effectiveness of hydrolysis as a fate pathway. But hydrolysis should be considered one of the two major fate processes for the isocyanates.
Aerobic and/or anaerobic biodegradation of the isocyanates is not expected to occur at significant levels. Most of the substances take several months to degrade.
Degradation of the hydrolysis products will occur at varying rates depending on the moiety formed.
for phthalate esters:
Environmental fate;
Under aerobic and anaerobic conditions, studies reveal that many phthalate esters are degraded by a wide range of bacteria and actinomycetes. Standardized aerobic biodegradation tests with sewage sludge inocula show that within 28 days approximately 50% ultimate degradation occurs. Biodegradation is, therefore, expected to be the dominant pathway in surface soils and sediments.
In the atmosphere, photodegradation via free radical attack is the anticipated dominant pathway. The half-life of many phthalate esters is ca. 1 day in the air, from < 1 day to 2 weeks in surface and marine waters, and from < 1 week to several months in soils.
Phthalates are high molecular weight chemicals, and are not expected to partition significantly to air. However for the minor amount that may partition to air, modelled predictions indicate that they would be rapidly oxidised: with a predicted atmospheric oxidation half-life of around 0.52 days . They are expected to react appreciably with other photo oxidative species in the atmosphere, such as O3. Therefore, it is expected that reactions with hydroxyl radicals will be the most important fate process in the atmosphere for phthalates.
Bioaccumulation of phthalate esters in the aquatic and terrestrial food chain is limited by biotransformation.
Most phthalates have experimental bioaccumulation factor (BCFs) and bioconcentration factor (BAFs) below 5000 L/kg, as they are readily metabolised by fish
A study of 18 commercial phthalate esters with alkyl chains ranging from one to 13 carbons found an eight order of magnitude increase in octanol-water coefficients (Kow) and a four order of magnitude decrease in vapor pressure with increasing length. This increase in Kow and decrease in vapor pressure results in increased partitioning of the phthalate esters to suspended solids, soils, sediments, and aerosols
The phthalate esters are distributed throughout the environment ubiquitously. They are found complexed with fulvic acid components of the humic substances in soil and marine and estuarine waters. Fulvic acid appears to act as a solubiliser for the otherwise insoluble ester and serves to mediate its transport and mobilisation in water or immobilisation in soil. Phthalate esters have been found in open ocean environments, in deep sea jelly fish, Atlantic herring and in mackerel. Phthalic ester plasticisers are clearly recognised as general contaminants of almost every soil and water ecosystem. In general they have low acute toxicity but the weight of evidence supporting their carcinogenicity is substantial. Other subtle chronic effects have also been reported. As little as 4 ug/ml in culture medium is lethal to chick embryo heart cells. This concentration is similar to that reached in human blood stored in vinyl plastic bags for as little as one day. As phthalates are present in drinking water and food, concerns have been raised about their long term effects on humans.
Ecotoxicity:
Some phthalates (notably di-2-ethylhexyl phthalate and dibutyl phthalate) may be detrimental to the reproduction of the water flea (Daphnia magna), zebra fish and guppies
While phthalates may have very low true water solubilities, they possess the ability to form suspensions which may cause adverse effects through physical contact with *Daphnia* at very low concentrations.
Available toxicity and water solubility information suggest that the high molecular weight phthalates, form these suspensions and are able to elicit chronic toxic effects at concentrations of approximately 0.05 mg/L . Therefore, these substances are considered to have the potential to harm aquatic organisms at relatively low concentrations
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
diisononyl phthalate	HIGH	HIGH

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4,4'-diphenylmethane diisocyanate (MDI)	LOW (Half-life = 1 days)	LOW (Half-life = 0.24 days)
titanium dioxide	HIGH	HIGH
2,4'-diphenylmethane diisocyanate	HIGH	HIGH
p-toluenesulfonyl isocyanate	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
diisononyl phthalate	LOW (BCF = 183.8)
4,4'-diphenylmethane diisocyanate (MDI)	LOW (BCF = 15)
titanium dioxide	LOW (BCF = 10)
2,4'-diphenylmethane diisocyanate	HIGH (LogKOW = 5.4481)
p-toluenesulfonyl isocyanate	LOW (LogKOW = 2.3424)

Mobility in soil

Ingredient	Mobility
diisononyl phthalate	LOW (KOC = 467200)
4,4'-diphenylmethane diisocyanate (MDI)	LOW (KOC = 376200)
titanium dioxide	LOW (KOC = 23.74)
2,4'-diphenylmethane diisocyanate	LOW (KOC = 384000)
p-toluenesulfonyl isocyanate	LOW (KOC = 882.1)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ **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.

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

Continued...

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

DIISONONYL PHTHALATE(68515-48-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

MDI/ CASTOR OIL/ GLYCEROL, PROPOXYLATED(154099-10-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Not Applicable

4,4'-DIPHENYLMETHANE DIISOCYANATE (MDI)(101-68-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Hazardous Substances Information System - Consolidated Lists
Australia Inventory of Chemical Substances (AICS)
Australia Work Health and Safety Regulations 2016 - Hazardous chemicals (other than lead) requiring health monitoring

TITANIUM DIOXIDE(13463-67-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Inventory of Chemical Substances (AICS)

MDI HOMOPOLYMER(25686-28-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists
Australia Inventory of Chemical Substances (AICS)

2,4'-DIPHENYLMETHANE DIISOCYANATE(5873-54-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Hazardous Substances Information System - Consolidated Lists
Australia Inventory of Chemical Substances (AICS)
Australia Work Health and Safety Regulations 2016 - Hazardous chemicals (other than lead) requiring health monitoring

P-TOLUENESULFONYL ISOCYANATE(4083-64-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Hazardous Substances Information System - Consolidated Lists
Australia Inventory of Chemical Substances (AICS)
Australia Work Health and Safety Regulations 2016 - Hazardous chemicals (other than lead) requiring health monitoring

National Inventory	Status
Australia - AICS	N (MDI/ castor oil/ glycerol, propoxylated)
Canada - DSL	N (MDI/ castor oil/ glycerol, propoxylated)
Canada - NDSL	N (diisononyl phthalate; MDI homopolymer; 4,4'-diphenylmethane diisocyanate (MDI); 2,4'-diphenylmethane diisocyanate; p-toluenesulfonyl isocyanate)
China - IECSC	N (MDI/ castor oil/ glycerol, propoxylated)
Europe - EINEC / ELINCS / NLP	N (MDI/ castor oil/ glycerol, propoxylated)
Japan - ENCS	N (diisononyl phthalate; MDI/ castor oil/ glycerol, propoxylated)
Korea - KECI	N (MDI/ castor oil/ glycerol, propoxylated)
New Zealand - NZIoC	N (MDI/ castor oil/ glycerol, propoxylated)
Philippines - PICCS	N (MDI/ castor oil/ glycerol, propoxylated)
USA - TSCA	Y
Legend:	*Y = All ingredients are on the inventory* *N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 OTHER INFORMATION

Other information

Ingredients with multiple cas numbers

Name	CAS No
diisononyl phthalate	68515-48-0, 28553-12-0
4,4'-diphenylmethane diisocyanate (MDI)	101-68-8, 26447-40-5
titanium dioxide	13463-67-7, 1317-70-0, 1317-80-2, 12188-41-9, 1309-63-3, 100292-32-8, 101239-53-6, 116788-85-3, 12000-59-8, 12701-76-7, 12767-65-6, 12789-63-8, 1344-29-2, 185323-71-1, 185828-91-5, 188357-76-8, 188357-79-1, 195740-11-5, 221548-98-7, 224963-00-2, 246178-32-5, 252962-41-7, 37230-92-5, 37230-94-7, 37230-95-8, 37230-96-9, 39320-58-6, 39360-64-0, 39379-02-7, 416845-43-7, 494848-07-6, 494848-23-6, 494851-77-3, 494851-98-8, 55068-84-3, 55068-85-4, 552316-51-5, 62338-64-1, 767341-00-4, 97929-50-5, 98084-96-9

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

Continued...

STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit。
IDLH: Immediately Dangerous to Life or Health Concentrations
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