Safety Data Sheet

Hazardous, NON-Dangerous Goods

1. MATERIAL AND SUPPLY COMPANY IDENTIFICATION

Product name: Vulkem 350R

Synonyms
Vulkem 350

Product Code

Recommended use: Polyurethane waterproofing membrane.Use according to manufacturer's directions.

Supplier: Tremco CPG Australia Pty Ltd
ABN: 25 000 024 064
Street Address: 12/4 Southridge Street
Eastern Creek NSW 2766
Telephone: 02 9638 2755
Facsimile: 02 9638 2955

Emergency Telephone number: **02 9037 2994**

2. HAZARDS IDENTIFICATION

This material is hazardous according to the criteria of Safe Work Australia GHS 7.

Signal Word
Danger

Hazard Classifications
Acute Toxicity - Oral - Category 4
Aspiration Hazard - Category 1
Skin Corrosion/Irritation - Category 2
Eye Damage/Irritation - Category 1
Sensitisation - Respiratory - Category 1
Sensitisation - Skin - Category 1
Germ Cell Mutagenicity - Category 2
Carcinogenicity - Category 1A
Reproductive Toxicity - Category 2
Specific Target Organ Toxicity (Single Exposure) - Category 3 Respiratory Tract Irritation
Specific Target Organ Toxicity (Repeated Exposure) - Category 2
Acute Hazard to the Aquatic Environment - Category 2

Hazard Statements

H302	Harmful if swallowed.
H304	May be fatal if swallowed and enters airways.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H318	Causes serious eye damage.
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H335	May cause respiratory irritation.
H341	Suspected of causing genetic defects .
H350	May cause cancer .
H361	Suspected of damaging fertility or the unborn child .
H373	May cause damage to organs through prolonged or repeated exposure.

H401	Toxic to aquatic life.

Prevention Precautionary Statements

P102	Keep out of reach of children.
P103	Read carefully and follow all instructions.
P202	Do not handle until all safety precautions have been read and understood.
P260	Do not breathe dust, fume, gas, mist, vapours or spray.
P264	Wash hands, face and all exposed skin thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P271	Use only outdoors or in a well-ventilated area.
P272	Contaminated work clothing should not be allowed out of the workplace.
P273	Avoid release to the environment.
P280	Wear protective gloves/protective clothing including eye/face protection.
P284	In case of inadequate ventilation wear respiratory protection.

Response Precautionary Statements

P101	If medical advice is needed, have product container or label at hand.
P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P304+P341	IF INHALED: If breathing is difficult, remove victim to fresh air and keep at rest in a position comfortable for breathing.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P310	Immediately call a POISON CENTER/doctor/insert appropriate source of emergency medical advice.
P330	Rinse mouth.
P331	Do NOT induce vomiting.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P342+P311	If experiencing respiratory symptoms: Call a POISON CENTRE or doctor/physician.
P362+P364	Take off contaminated clothing and wash it before reuse

Storage Precautionary Statements

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

Disposal Precautionary Statement

P501	Dispose of contents/container in accordance with local, regional, national and international regulations.

Poison Schedule:

DANGEROUS GOOD CLASSIFICATION

Not classified as Dangerous Goods by the criteria of the "Australian Code for the Transport of Dangerous Goods by Road & Rail" and the "New Zealand NZS5433: Transport of Dangerous Goods on Land".

3. COMPOSITION INFORMATION

CHEMICAL ENTITY	CAS NO	PROPORTION
Limestone	1317-65-3	35-40 %
MDI, propoxylated	9048-57-1	30-35 %
Naphtha, petroleum, hydrotreated heavy	64742-48-9	10-15 %
diisononyl phthalate	68515-48-0	5-10 %
MDI-glycerol, propoxylated. ethoxylated	59675-67-1	5-10 %
Titanium oxide (TiO2)	13463-67-7	<5 %
Calcium oxide	1305-78-8	<5 %
4, 4' - diphenylmethane diisocyanate (MDI)	101-68-8	<5 %
Silane	7803-62-5	<1 %
Ingredients determined to be Non-Hazardous		Balance

4. FIRST AID MEASURES

If poisoning occurs, contact a doctor or Poisons Information Centre (Phone Australia 131 126, New Zealand 0800 764 766).

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 administeredas 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.

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.

Eye contact: If this product comes in contact with the eyes:• Immediately hold eyelids apart and flush the eye continuously with running water.• Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.• Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes.• Transport to hospital or doctor without delay.• Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.

Ingestion: If swallowed do NOT induce vomiting.• If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.• Observe the patient carefully.• Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.• Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.• Seek medical advice.• Avoid giving milk or oils.• Avoid giving alcohol.• If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

PPE for First Aiders: Wear rubber boots, overalls, gloves, respirator. Use with adequate ventilation. If inhalation risk exists wear organic vapour/particulate respirator meeting the requirements of AS/NZS 1715 and AS/NZS 1716. Available information suggests that gloves made from nitrile rubber, polyvinyl alcohol (PVA), teflon should be suitable for intermittent contact. However, due to variations in glove construction and local conditions, the user should make a final assessment. Always wash hands before smoking, eating, drinking or using the toilet. Wash contaminated clothing and other protective equipment before storing or re-using.

Notes to physician: Treat symptomatically. Effects may be delayed. Can cause corneal burns. Indication of any immediate medical attention and special treatment neededAny material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it isconsidered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patientshould be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours. Treat symptomatically.For petroleum distillates• In case of ingestion, gastric lavage with activated charcoal can be used promptly to prevent absorption - decontamination (induced emesis or lavage) is controversial and should be considered on the merits of each individual case; of course the usual precautions of an endotracheal tube should be considered prior to lavage, to prevent aspiration.• Individuals intoxicated by petroleum distillates should be hospitalized immediately, with acute and continuing attention to neurologic and cardiopulmonary function.• Positive pressure ventilation may be necessary.• Acute central nervous system signs and symptoms may result from large ingestions of aspiration-induced hypoxia.• After the initial episode,individuals should be followed for changes in blood variables and the delayed appearance of pulmonary oedema and chemical pneumonitis. Such patients should be followed for several days or weeks for delayed effects, including bone marrow toxicity, hepatic and renal impairment Individuals with chronic pulmonary disease will be more seriously impaired, and recovery from inhalation exposure may be complicated.• Gastrointestinal symptoms are usually minor and pathological changes of the liver and kidneys are reported to be uncommon in acute intoxications.• Chlorinated and non-chlorinated hydrocarbons may sensitize the heart to epinephrine and other circulating catecholamines so that arrhythmias may occur.Careful consideration of this potential adverse effect should precede administration of

epinephrine or other cardiac stimulants and the selection of bronchodilators.BP America Product Safety & Toxicology Department 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.

5. FIRE FIGHTING MEASURES

Hazchem Code: Not applicable.

Suitable extinguishing media: If material is involved in a fire use water fog (or if unavailable fine water spray), alcohol resistant foam, standard foam, dry agent (carbon dioxide, dry chemical powder).

Specific hazards: Combustible material.

Fire fighting further advice: On burning or decomposing may emit toxic fumes. Fire fighters to wear self-contained breathing apparatus and suitable protective clothing if risk of exposure to vapour or products of combustion or decomposition.

6. ACCIDENTAL RELEASE MEASURES

SMALL 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.

LARGE 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:•Treat isocyanate spills with sufficient amounts of isocyanate decontaminant preparation ("neutralising fluid"). Isocyanates and polyisocyanatesare 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 hoursThree 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 Bliquid surfactant 0.2-2%concentrated ammonia 3-8%water to 100%Formulation Cethanol, 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.

Dangerous Goods - Initial Emergency Response Guide No: Not applicable

7. HANDLING AND STORAGE

Handling: 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. 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: • direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of

rapid air motion)Air Speed: 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 currents2: Contaminants of low toxicity or of nuisance value only 2: Contaminants of high toxicity3: Intermittent, low production. 3: High production, heavy use4: Large hood or large air mass in motion 4: Small hood-local control onlySimple 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 installedor used.The conductivity of this material may make it a static accumulator., A liquid is typically considered nonconductive if its conductivity is below 100 pS/m and is considered semi-conductive if its conductivity is below 10 000 pS/m., Whether a liquid is nonconductive or semi-conductive, the precautions are the same., A number of factors, for example liquid temperature, presence of contaminants, and anti-static additives can greatlyinfluence the conductivity of a liquid.• Containers, even those that have been emptied, may contain explosive vapours.• Do NOT cut, drill, grind, weld or perform similar operations on or near containers.• DO NOT allow clothing wet with material to stay in contact with skin• Overheating of ethoxylates/ alkoxylates in air should be avoided. When some ethoxylates are heated vigorously in the presence of air or oxygen, at temperatures exceeding 160 C, they may undergo exothermic oxidative degeneration resulting in self-heating and autoignition.• Nitrogen blanketing will minimise the potential for ethoxylate oxidation. Prolonged storage in the presence of air or oxygen may cause product degradation. Oxidation is not expected when stored under a nitrogen atmosphere. Inert gas blanket and breathing system needed to maintain color stability. Use dry inert gas having at least -40 C dew point.• Trace quantities of ethylene oxide may be present in the material. Although these may accumulate in the headspace of storage and transport vessels, concentrations are not expected to exceed levels which might produce a flammability or worker exposure hazard.• Electrostatic discharge may be generated during pumping - this may result in fire.• Ensure electrical continuity by bonding and grounding (earthing) all equipment.• Restrict line velocity during pumping in order to avoid generation of electrostatic discharge (<=1 m/sec until fill pipe submerged to twice its diameter, then <= 7 m/sec).• Avoid splash filling.• Do NOT use compressed air for filling discharging or handling operations.• Avoid all personal contact, including inhalation.• Wear protective clothing when risk of exposure occurs.• Use in a well-ventilated area.• Prevent concentration in hollows and sumps.• DO NOT enter confined spaces until atmosphere has been checked.• Avoid smoking, naked lights or ignition sources.• Avoid contact with incompatible materials.• When handling, DO NOT eat, drink or smoke.• Keep containers securely sealed when not in use.• Avoid physical damage to containers.• Always wash hands with soap and water after handling.• Work clothes should be laundered separately.• Use good occupational work practice.• Observe manufacturer's storage and handling recommendations contained within this SDS.• Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.

Storage: Consider storage under inert gas.• 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.Suitable container:• 15 Litre metal pail.• For ethoxylates suitable containers include carbon steel coated with baked phenolic.• Any moisture may cause rusting of carbon steel.• If product is moisture free, uncoated carbon steel tanks may be used.• Polyethylene or polypropylene container.• Packing as recommended by manufacturer.• Check all containers are clearly labelled and free from leaks.Storage incompatibility:• Avoid reaction with oxidising agents, bases and strong reducing agents.• Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.

8. EXPOSURE CONTROLS / PERSONAL PROTECTION

National occupational exposure limits:

	TWA		STEL		NOTICES
	ppm	mg/m3	ppm	mg/m3	
Calcium oxide	-	2	-	-	-
Methylene bisphenyl isocyanate (MDI)					
Silicon tetrahydride	5	6.6	-	-	-

Titanium dioxide	-	10	-	-	-

As published by Safe Work Australia.

TWA - The time-weighted average airborne concentration over an eight-hour working day, for a five-day working week over an entire working life.

STEL (Short Term Exposure Limit) - the average airborne concentration over a 15 minute period which should not be exceeded at any time during a normal eight-hour workday.

These Exposure Standards are guides to be used in the control of occupational health hazards. All atmospheric contamination should be kept to as low a level as is workable. These exposure standards should not be used as fine dividing lines between safe and dangerous concentrations of chemicals. They are not a measure of relative toxicity.

If the directions for use on the product label are followed, exposure of individuals using the product should not exceed the above standard. The standard was created for workers who are routinely, potentially exposed during product manufacture.

Biological Limit Values: As per the "National Model Regulations for the Control of Workplace Hazardous Substances (Safe Work Australia)" the ingredients in this material do not have a Biological Limit Allocated.

Engineering Measures: Ensure ventilation is adequate to maintain air concentrations below Exposure Standards. Use only in well ventilated areas. Use with local exhaust ventilation or while wearing appropriate respirator.

Personal Protection Equipment: RUBBER BOOTS, OVERALLS, GLOVES, RESPIRATOR.

Personal protective equipment (PPE) must be suitable for the nature of the work and any hazard associated with the work as identified by the risk assessment conducted.

Wear rubber boots, overalls, gloves, respirator. Use with adequate ventilation. If inhalation risk exists wear organic vapour/particulate respirator meeting the requirements of AS/NZS 1715 and AS/NZS 1716. Available information suggests that gloves made from nitrile rubber, polyvinyl alcohol (PVA), teflon should be suitable for intermittent contact. However, due to variations in glove construction and local conditions, the user should make a final assessment. Always wash hands before smoking, eating, drinking or using the toilet. Wash contaminated clothing and other protective equipment before storing or re-using.

RECOMMENDATIONS FOR CONSUMER USE:
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 belowHands/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 tomanufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: •frequency and duration of contact,• chemical resistance of glove material,• glove thickness and• dexteritySelect gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).• When prolonged or frequently repeated contact may occur, a

glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.• When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.• Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.• Contaminated gloves should be replaced.As defined in ASTM F-739-96 in any application, gloves are rated as:• Excellent when breakthrough time > 480 min• Good when breakthrough time > 20 min• Fair when breakthrough time < 20 min• Poor when glove material degradesFor 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.NOTE: Natural rubber, neoprene, PVC can be affected by isocyanatesDO 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 belowOther 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.

Hygiene measures: Keep away from food, drink and animal feeding stuffs. When using do not eat, drink or smoke. Wash hands prior to eating, drinking or smoking. Avoid contact with clothing. Avoid eye contact and skin contact. Avoid inhalation of vapour, mist or aerosols. Ensure that eyewash stations and safety showers are close to the workstation location.

9. PHYSICAL AND CHEMICAL PROPERTIES

Form:	Liquid
Colour:	Grey liquid
Odour:	Mild petroleum solvent odour

Solubility:	Does not mix with water.
Solubility in water:	Immiscible
Density:	1.37
Flash Point (°C):	>100

(Typical values only - consult specification sheet)
N Av = Not available, N App = Not applicable

10. STABILITY AND REACTIVITY

Chemical stability: • Unstable in the presence of incompatible materials.• Product is considered stable.• Hazardous polymerisation will not occur.

Conditions to avoid: See section 7

Incompatible materials: See section 7

Hazardous decomposition products: See section 5

Hazardous reactions: See section 7

11. TOXICOLOGICAL INFORMATION

No adverse health effects expected if the product is handled in accordance with this Safety Data Sheet and the product label. Symptoms or effects that may arise if the product is mishandled and overexposure occurs are:

Acute Effects

Inhalation: Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may produce toxic effects. Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. High inhaled concentrations of mixed hydrocarbons may produce narcosis characterised by nausea, vomiting and lightheadedness. Inhalation of aerosols may produce severe pulmonary oedema, pneumonitis and pulmonary haemorrhage. Inhalation of petroleum hydrocarbons consisting substantially of low molecular weight species (typically C2-C12) may produce irritation of mucous membranes, incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and anaesthetic stupor. Massive exposures may produce central nervous system depression with sudden collapse and deep coma; fatalities have been recorded. Irritation of the brain and/or apnoeic anoxia may produce convulsions. Although recovery following overexposure is generally complete, cerebral micro-haemorrhage of focal post-inflammatory scarring may produce epileptiform seizures some months after the exposure. Pulmonary episodes may include chemical pneumonitis with oedema and haemorrhage. The lighter hydrocarbons may produce kidney and neurotoxic effects. Pulmonary irritancy increases with carbon chain length for paraffins and olefins. Alkenes produce pulmonary oedema at high concentrations. Liquid paraffins may produce anaesthesia and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce polyneuropathy. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue and vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons are cardiac sensitisers and may cause ventricular fibrillations. 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 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 towork 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. Some aliphatic hydrocarbons produce axonal neuropathies. Isoparaffinic hydrocarbons produce injury to the kidneys of male rats. When albino rats were exposed to isoparaffins at 21.4 mg/l for 4 hours, all animals experienced weakness, tremors, salivation, mild to moderate convulsions, chromodacryorrhoea and ataxia within the first 24 hours. Symptoms disappeared after 24 hours. Several studies have evaluated sensory irritation in laboratory animals or odor or sensory response in humans.

When evaluated by a standardprocedure to assess upper airway irritation, isoparaffins did not produce sensory irritation in mice exposed to up to 400 ppm isoparaffin in air. Human volunteers were exposed for six hours to 100 ppm isoparaffin. The subjects were given a self-administered questionnaire to evaluate symptoms, which included dryness of the mucous membranes, loss of appetite, nausea, vomiting, diarrhea, fatigue, headache, dizziness, feeling of inebriation, visual disturbances, tremor, muscular weakness, impairment of coordination or paresthesia. No symptoms associated with solvent exposure were observed. With a human expert panel, odour from liquid imaging copier emissions became weakly discernible at approximately 50ppm. Numerous long-term exposures have been conducted in animals with only one major finding observed. Renal tubular damage has been found in kidneys of male rats upon repeated exposures to isoparaffins. It does not occur in mice or in female rats. This male rat nephropathy has been observed with a number of hydrocarbons, including wholly vaporized unleaded gasoline. The phenomenon has been attributed to reversible binding of hydrocarbon to alpha2-globulin. Since humans do not synthesize alpha2-globulin or a similar protein, the finding is not considered to be of biological significance to man. No clinically significant renal abnormalities have been found in refinery workers exposed to hydrocarbons. When evaluated for developmental toxicity in rats, isoparaffins were neither embryotoxic nor teratogenic. Isoparaffins were consistently negative on standard bacterial genotoxicity assays. They were also non-genotoxic in in vivo mammalian testing for somatic or germ cell mutations (mouse micronucleus test and rat dominant lethal assay, respectively). Mullin et al: Jnl Applied Toxicology 10, pp 136-142, 2006 A respiratory sensitiser. Can cause possible allergic reactions.

Skin contact: The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material eitherproduces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/orproduces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammationbeing 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). Thedermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling andthickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) andintracellular oedema of the epidermis.Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.Dermally, isoparaffins have produced slight to moderate irritation in animals and humans under occluded patch conditions where evaporationcannot freely occur. However, they are not irritating in non-occluded tests, which are a more realistic simulation of human exposure. They havenot been found to be sensitisers in guinea pig or human patch testing. However, occasional rare idiosyncratic sensitisation reactions in humanshave been reported.Open cuts, abraded or irritated skin should not be exposed to this materialEntry 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. A skin sensitiser. Repeated or prolonged skin contact may lead to allergic contact dermatitis.

Ingestion: Harmful if swallowed. Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or mayproduce serious damage to the health of the individual.Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemicalpneumonitis; serious consequences may result.Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, andbluish coloured skin (cyanosis).Many aliphatic hydrocarbons create a burning sensation because they are irritating to the GI mucosa. Vomiting has been reported in up to onethird of all hydrocarbon exposures. While most aliphatic hydrocarbons have little GI absorption, aspiration frequently occurs, either initially or in asemi-delayed fashion as the patient coughs or vomits, thereby resulting in pulmonary effects. Once aspirated, the hydrocarbons can create asevere pneumonitis.Rats given isoparaffinic hydrocarbons - isoalkanes- (after 18-24 hours fasting) showed lethargy and/or general weakness, ataxia and diarrhoea.Symptoms disappeared within 24-28 hours.Ingestion of petroleum hydrocarbons may produce irritation of the pharynx, oesophagus, stomach and small intestine with oedema and mucosalulceration resulting; symptoms include a burning sensation in the mouth and throat. Large amounts may produce narcosis with nausea andvomiting, weakness or dizziness, slow and shallow respiration, swelling of the abdomen, unconsciousness and convulsions. Myocardial injurymay produce arrhythmias, ventricular fibrillation and electrocardiographic changes. Central nervous system depression may also occur. Lightaromatic hydrocarbons produce a warm, sharp, tingling sensation on contact with taste buds and may anaesthetise the tongue. Aspiration intothe lungs may produce coughing, gagging and a chemical pneumonitis with pulmonary oedema and haemorrhage.Phthalates (aromatic dicarboxylic acid esters), in general, exhibit low toxicity, partly because of poor absorption but mainly as a result of rapidmetabolism in which the esters are saponified to phthalic acid (which is rapidly excreted) and the parent alcohol (which is subsequentlymetabolised). The pathology of these compounds seems to be related to the released alcohol and its biological effects. The rate of absorption ofingested phthalate esters is influenced by the content of dietary fat. Ingested phthalate esters may to a lesser degree be absorbed as themonoester derivatives or in the case of di(2-

ethylhexyl)phthalate, as the diester. Cumulative toxicity of the phthalates has been observed onrepeated 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 phthalateshave induced liver enlargement; this increase in liver weight has been attributed to rapid cell division (hyperplasia) along with the detachment ofcells (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 whichaccounted for a reduction in liver steroidogenesis. The phthalates also effect carbohydrate metabolism in the liver producing depleted glycogenelectron transport inhibitors following interaction with mitochondria. Testicular atrophy produced in rats during feeding studies depends on thelength and structure of the alcohol; in general the lower molecular weight esters produce the more severe effects. The toxicity of phthalic acidisomers decreases in the order o-phthalic acid, isophthalic acid and terephthalic acid. Phthalic acid is not metabolised but is excreted,unchanged, in the urine and faeces. Terephthalic acid appears to potentiate the biological effects of substances such as antibiotics, thiamine andsulfonamides. May cause lung damage if swallowed. Small amounts of liquid aspirated into the respiratory system during ingestion or vomiting may cause bronchopneumonia or pulmonary oedema.

Eye contact: When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.Instillation of isoparaffins into rabbit eyes produces only slight irritation.Petroleum hydrocarbons may produce pain after direct contact with the eyes. Slight, but transient disturbances of the corneal epithelium may alsoresult. The aromatic fraction may produce irritation and lachrymation.

Acute toxicity

Inhalation: This material has been classified as not hazardous for acute inhalation exposure. Acute toxicity estimate (based on ingredients): LC_{50} > 20.0 mg/L for vapours or LC_{50} > 5.0 mg/L for dust and mist.

silane LC50 (Mice): >1000 ppm4h[1]
naphtha petroleum, heavy, hydrotreated LC50 (Rat): >4.42 mg/L4h[1]
diisononyl phthalate LC50 (Rat): >4.4 mg/l4h[1]
titanium dioxide LC50 (Rat): >2.28 mg/l4h[1]
calcium oxide LC50 (Rat): >3 mg/l4h[1]
4,4'-diphenylmethane diisocyanate (MDI) LC50 (Rat): 0.368 mg/L4h[1]

Skin contact: This material has been classified as not hazardous for acute dermal exposure. Acute toxicity estimate (based on ingredients): LD_{50} > 2,000 mg/Kg bw

titanium dioxide LD50 (Guinea pig): >=10000 mg/kg[2] (Method: Dermal)
naphtha petroleum, heavy, hydrotreated LD50 (Rabbit): >1900 mg/kg[1] (Method: Dermal)
diisononyl phthalate LD50 (Rabbit): >3160 mg/kg[2] (Method: Dermal)
4,4'-diphenylmethane diisocyanate (MDI) LD50 (Rabbit): >6200 mg/kg[2] (Method: Dermal)
silane LD50 (Rabbit): 3540 mg/kg[2] (Method: Dermal)
calcium oxide LD50 (Rat): >2000 mg/kg[1] (Method: Dermal)

Ingestion: This material has been classified as a Category 4 Hazard. Acute toxicity estimate (based on ingredients): 300 < LD_{50} ≤ 2,000 mg/Kg bw

limestone LD50 (Rat): 6450 mg/kg[2] (Method: Oral)
naphtha petroleum, heavy, hydrotreated LD50 (Rat): >4500 mg/kg[1] (Method: Oral)
diisononyl phthalate LD50 (Rat): 2550 mg/kg[2] (Method: Oral)
MDI-glycerol, propoxylated. ethoxylated LD50 (Rat): >2000 mg/kg[2] (Method: Oral)
titanium dioxide LD50 (Rat): >=2000 mg/kg[1] (Method: Oral)
calcium oxide LD50 (Rat): >2000 mg/kg[1] (Method: Oral)
4,4'-diphenylmethane diisocyanate (MDI) LD50 (Rat): >2000 mg/kg[1] (Method: Oral)

Corrosion/Irritancy: Eye: this material has been classified as a Category 1 Hazard (irreversible effects to eyes). Skin: this material has been classified as a Category 2 Hazard (reversible effects to skin).

limestone Skin irritant (Rabbit): 500 mg/24h-moderate
4,4'-diphenylmethane diisocyanate (MDI) Skin irritant (Rabbit): 500 mg /24 hours

Sensitisation: Inhalation: this material has been classified as a Category 1 Hazard (respiratory sensitiser). Skin: this material has been classified as a Category 1 Hazard (skin sensitiser).

Aspiration hazard: This material has been classified as Aspiration Hazard - Category 1

Specific target organ toxicity (single exposure): This material has been classified as a Category 3 Hazard. Exposure via inhalation may result in respiratory irritation.

Chronic Toxicity

Mutagenicity: This material has been classified as a Category 2 Hazard.

Carcinogenicity: This material has been classified as a Category 1A Hazard.

Reproductive toxicity (including via lactation): This material has been classified as a Category 2 Hazard.

Specific target organ toxicity (repeat exposure): This material has been classified as a Category 2 Hazard. On the basis of epidemiological data, it has been concluded that prolonged inhalation of the material, in an occupational setting, may producecancer in humans.Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.Strong evidence exists that the substance may cause irreversible but non-lethal mutagenic effects following a single exposure.Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial number of individuals at agreater 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 ofnonspecific 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 ofindividuals, and/or of producing a positive response in experimental animals.Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airwayhyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure tothe substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose toasthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely tobecome hyper-responsive.Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in peoplewith pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisersWherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is notpossible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Healthsurveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and thereshould be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.Harmful: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused byrepeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage maybecome apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicitytests.There is sufficient evidence to establish a causal relationship between human exposure to the material and impaired fertilityThere is sufficient evidence to establish a causal relationship between human exposure to the material and subsequent developmental toxiceffects in the off-spring.The various phthalates have different uses, chemical structures and toxicity profiles. It is therefore difficult to generalise about the safety of allphthalates as a group. The main health concern associated with some phthalates is that animal studies have shown that high regular doses canaffect the reproductive system in developing young, particularly males. While there is no significant risk to the general population, young childrenmay experience higher exposures than the general population if they chew or suck on phthalate-containing toys, or if they ingest phthalates overa 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, undescendedtesticles, and low sperm counts. A 2005 study also linked higher foetal exposure to phthalates through the mother's blood with increased risk ofdevelopmental abnormalities in male

infants. Higher phthalate levels are also associated with lower testosterone production and reduced spermcount 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 childrenshow greater exposure to phthalates than non-obese children. It was reported that the obesity risk increases according to the level of thechemical found in the children's bloodstream. in a national cross-section of U.S. men, concentrations of several prevalent phthalate metabolitesshowed statistically significant correlations with abnormal obesity and insulin resistance. A further study found that people with elevated phthalatelevels had roughly twice the risk of developing diabetes compared with those with lower levels. This study also found that phthalates wereassociated 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 athigher risk for potential adverse health effects of phthalates due to increased cosmetic use. According to in vivo and observational studies thereis an association between phthalate exposure and endocrine disruption leading to development of breast cancer. This finding may be associatedwith 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 andlower extremities were related to duration of exposures. Symptoms usually developed after the sixth or seventh year of work. Neurologicalstudies revealed the development of polyneuritis in about 30% of the workers involved in this study. About 30% of the workforce showeddepression of the vestibular receptors. Because the study described mixed exposures it is difficult to determine what, if any, unique role wasplayed by the phthalates. Increased incidences of anovulatory reproductive cycles and low oestrogen concentrations were reported amongRussian 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 areused as ingredients in paints, inks and adhesives. Their potential for entering the human body is marked. They have been added to a list ofchemicals (including alkyl phenolics, polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs) and dioxins) which areimplicated 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 ofoestrogen on the body.Although the human foetus is "bathed" in naturally occurring oestrogens during pregnancy it is suggested that it has developed a protectivemechanism against natural oestrogens but is not safe from synthetic variants. These tend to accumulate in body fats which sets them apart fromthe natural product. During early pregnancy, fats are broken down and may flood the body with concentrated pollutantsHuman phthalate exposure during pregnancy results in decreased anogenital distance among baby boys.Boys born to mothers with the highestlevels 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 assessedin humans, and its significance is unknownOne study also found that female animals exposed to higher levels of phthalates experienced increased risk of miscarriage, a common symptomof 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 isunclear, studies have linked higher rates of respiratory infections and other symptoms in children living in houses with vinyl floors. One possibleexplanation is inhalation of dust tainted by phthalates, which are used in cosmetics such as nail polishes and hand creams precisely because oftheir 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 ratesin 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 averagephthalate 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 deviceapplications. The USA has introduced regulation of phthalate esters as components of children's toys and childcare articles for children underthe age of 12 that could be 'placed in the mouth'.Endocrine disruptors such as phthalates can be add to the effects of other endocrine disruptors, so even very small amounts can interact withother 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 indicatedhepatocarcinogenicity. Later studies on primates showed that the mechanism is specific to rodents - humans are resistant to the effectStudies 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 aremembers 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 inchildren under 5 years of age and increases the risk of cardiovascular and metabolic disease in adulthood. Another study found that women

whodeliver 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 deficithyperactivity disorder (ADHD)Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/ormemory loss, tremor in the fingers and tongue, vertigo, olfactory disorders, constriction of visual field, paraesthesias of the extremities, weightloss and anaemia and degenerative changes in the liver and kidney. Chronic exposure by petroleum workers, to the lighter hydrocarbons, hasbeen associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness andparaesthesias), psychological and neurophysiological deficits, bone marrow toxicities (including hypoplasia possibly due to benzene) and hepaticand renal involvement. Chronic dermal exposure to petroleum hydrocarbons may result in defatting which produces localised dermatoses.Surface cracking and erosion may also increase susceptibility to infection by microorganisms. One epidemiological study of petroleum refineryworkers has reported elevations in standard mortality ratios for skin cancer along with a dose-response relationship indicating an associationbetween routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have beenunable to confirm this finding.Hydrocarbon solvents are liquid hydrocarbon fractions derived from petroleum processing streams, containing only carbon and hydrogen atoms,with carbon numbers ranging from approximately C5-C20 and boiling between approximately 35-370 deg C. Many of the hydrocarbon solventshave complex and variable compositions with constituents of 4 types, alkanes (normal paraffins, isoparaffins, and cycloparaffins) and aromatics(primarily alkylated one- and two-ring species). Despite the compositional complexity, most hydrocarbon solvent constituents have similartoxicological properties, and the overall toxicological hazards can be characterized in generic terms. Hydrocarbon solvents can cause chemicalpneumonitis if aspirated into the lung, and those that are volatile can cause acute CNS effects and/or ocular and respiratory irritation at exposurelevels exceeding occupational recommendations. Otherwise, there are few toxicologically important effects. The exceptions, n-hexane andnaphthalene, have unique toxicological propertiesAnimal studies:No deaths or treatment related signs of toxicity were observed in rats exposed to light alkylate naphtha (paraffinic hydrocarbons) atconcentrations of 668, 2220 and 6646 ppm for 6 hrs/day, 5 days/wk for 13 weeks. Increased liver weights and kidney toxicity (male rats) wasobserved in high dose animals. Exposure to pregnant rats at concentrations of 137, 3425 and 6850 ppm did not adversely affect reproduction orcause maternal or foetal toxicity. Lifetime skin painting studies in mice with similar naphthas have shown weak or no carcinogenic activityfollowing prolonged and repeated exposure. Similarnaphthas/distillates, when tested at nonirritating dose levels, did not show any significant carcinogenic activity indicating that this tumorigenicresponse is likely related to chronic irritation and not to dose. The mutagenic potential of naphthas has been reported to be largely negative in avariety of mutagenicity tests. The exact relationship between these results and human health is not known. Some components of this producthave been shown to produce a species specific, sex hormonal dependent kidney lesion in male rats from repeated oral or inhalation exposure.Subsequent research has shown that the kidney damage develops via the formation of a alpha-2u-globulin, a mechanism unique to the male rat.Humans do not form alpha-2u-globulin, therefore, the kidney effects resulting from this mechanism are not relevant in human.Persons with a history of asthma or other respiratory problems or are known to be sensitised, should not be engaged in any work involving thehandling of isocyanates.The chemistry of reaction of isocyanates, as evidenced by MDI, in biological milieu is such that in the event of a true exposure of small MDIdoses to the mouth, reactions will commence at once with biological macromolecules in the buccal region and will continue along the digestivetract prior to reaching the stomach. Reaction products will be a variety of polyureas and macromolecular conjugates with for example mucus,proteins and cell components.This is corroborated by the results from an MDI inhalation study. Following an inhalation exposure of rats to radiolabelled MDI, 79% of the dosewas excreted in faeces. The faecal excretion in these animals was considered entirely due to ingestion of radioactivity from grooming andingestion of deposited material from the nasopharangeal region via the mucociliary escalator, i.e. not following systemic absorption. The faecalradioactivity was tentatively identified as mixed molecular weight polyureas derived from MDI. Diamine was not present. Thus, for MDI anddiisocyanates in general the oral gavage dosing route is inappropriate for toxicological studies and risk assessment.It is expected that oral gavage dosing will result in a similar outcome to that produced by TDI or MDI, that is (1) reaction with stomach contentsand (2) polymerization to solid polyureas.Reaction with stomach contents is very plausibly described in case reports of accidental ingestion of polymeric MDI based glue in domesticanimals. Extensive polymerization and CO_2 liberation resulting in an expansion of the gastric content is described in the stomach, withoutapparent acute chemical toxicity Polyurea formation in organic and aqueous phases has been described. In this generally accepted chemistry of hydrolysis of an isocyanatethe initially produced carbamate decarboxylates to an amine which. The amine, as a reactive intermediate, then reacts very readily with thepresent isocyanate to produce a solid and inert polyurea. This urea formation acts as a pH buffer in the stomach, thus promotingtransformation of the diisocyanate into polyurea, even under the acidic conditions.At the resorbtive tissues in the small intestine, these high molecular reaction products are likely to be of very low bioavailability, which issubstantiated by the absence of systemic toxicity in acute oral bioassays with rats at the OECD limit dose (LC50>2 g/kg bw).The respiratory tract may be regarded as

the main entry for systemically available isocyanates as evidenced following MDI.exposures.A detailed summary on urinary, plasma and in vitro metabolite studies is provided below. Taken together, all available studies provide convincingevidence that MDI-protein adduct and MDI-metabolite formation proceeds:via formation of a labile isocyanate glutathione (GSH)-adduct,then transfer to a more stable adduct with larger proteins, andwithout formation of free MDA. MDA reported as a metabolite is actually formed by analytical workup procedures (strong acid or basehydrolysis) and is not an identified metabolite in urine or bloodA 90-day inhalation study in rats with polymeric MDI (6 hours/day, 5 days/week) produced moderate to severe hyperplastic inflammatory lesionsin the nasal cavities and lungs at levels of 8 mg/m3 or greater.Rats exposed for two years to a respirable aerosol of polymeric MDI exhibited chronic pulmonary irritation at high concentrations. Only at thehighest level (6 mg/m3),was there a significant incidence of a benign tumour of the lung (adenoma) and one malignant tumour(adenocarcinoma).There were no lung tumours at 1 mg/m3 and no effects at 0.2 mg/m3. Overall, the tumour incidence, both benign andmalignant and the number of animals with the tumours were not different from controls.The increased incidence of lung tumours is associatedwith prolonged respiratory irritation and the concurrent accumulation of yellow material in the lung, which occurred throughout the study.In theabsence of prolonged exposure to high concentrations leading to chronic irritation and lung damage,it is highly unlikely that tumour formation willoccur. On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.

12. ECOLOGICAL INFORMATION

Avoid contaminating waterways.

Acute aquatic hazard: This material has been classified as a Category Acute 2 Hazard. Acute toxicity estimate (based on ingredients): > 1 ≤ 10 mg/L

diisononyl phthalate 48hr EC50 (crustacea): >0.086mg/l
titanium dioxide 48hr EC50 (crustacea): 1.9mg/l
calcium oxide 48hr EC50 (crustacea): 49.1mg/l
limestone 72hr EC50 (algae): >14mg/l
diisononyl phthalate 72hr EC50 (algae): >88mg/l
titanium dioxide 72hr EC50 (algae): 3.75-7.58mg/l
calcium oxide 72hr EC50 (algae): >14mg/l
4,4'-diphenylmethane diisocyanate (MDI) 72hr EC50 (algae): >1640mg/l
naphtha petroleum, heavy, hydrotreated 96hr EC50 (algae): 64mg/l
diisononyl phthalate 96hr EC50 (algae): >2.8mg/l
titanium dioxide 96hr EC50 (algae): 179.05mg/l
limestone 96hr LC50 (fish): >165200mg/L
diisononyl phthalate 96hr LC50 (fish): >0.1mg/l
titanium dioxide 96hr LC50 (fish): 1.85-3.06mg/l
calcium oxide 96hr LC50 (fish): 50.6mg/l
4,4'-diphenylmethane diisocyanate (MDI) 96hr LC50 (fish): >1000mg/l

Long-term aquatic hazard: On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and or observed environmental fate and behaviour, the material may present adanger, 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 disposingof equipment wash-waters.Wastes resulting from use of the product must be disposed of on site or at approved waste sites.For petroleum distillates:Environmental fate:When petroleum substances are released into the environment, four major fate processes will take place: dissolution in water, volatilization, biodegradation and adsorption. Theseprocesses will cause changes in the composition of these UVCB substances. In the case of spills on land or water surfaces, photodegradation-another fate process-can also besignificant.As noted previously, the solubility and vapour pressure of components within a mixture will differ from those of the component alone. These interactions are complex for complexUVCBs such as petroleum hydrocarbons.Each of the fate processes affects hydrocarbon families differently. Aromatics tend to be more water-soluble than aliphatics of the same carbon number, whereas aliphatics tend to bemore volatile. Thus,when a petroleum mixture is released into the environment, the principal water contaminants are likely to be aromatics, whereas aliphatics will be the principal aircontaminants . The trend in volatility by component class is as follows: alkenes = alkanes > aromatics = cycloalkanes.The most soluble and volatile components have the lowest molecular weight; thus there is a general

shift to higher molecular weight components in residual materials.Biodegradation:Biodegradation is almost always operative when petroleum mixtures are released into the environment. It has been widely demonstrated that nearly all soils and sediments havepopulations of bacteria and other organisms capable of degrading petroleum hydrocarbons Degradation occurs both in the presence and absence of oxygen. Two key factors thatdetermine degradation rates are oxygen supply and molecular structure. In general, degradation is more rapid under aerobic conditions. Decreasing trends in degradation ratesaccording to structure are as follows:(1) n-alkanes, especially in the C10–C25 range, which are degraded readily;(2) isoalkanes;(3) alkenes;(4) benzene, toluene, ethylbenzene, xylenes (BTEX) (when present in concentrations that are not toxic to microorganisms);(5) monoaromatics;(6) polynuclear (polycyclic) aromatic hydrocarbons (PAHs); and(7) higher molecular weight cycloalkanes (which may degrade very slowly.Three weathering processes-dissolution in water, volatilization and biodegradation-typically result in the depletion of the more readily soluble,volatile and degradable compounds andthe accumulation of those most resistant to these processes in residues.When large quantities of a hydrocarbon mixture enter the soil compartment, soil organic matter and other sorption sites in soil are fully saturated and the hydrocarbons will begin toform a separate phase (a non-aqueous phase liquid, or NAPL) in the soil. At concentrations below the retention capacity for the hydrocarbon in the soil, the NAPL will be immobile thisis referred to as residual NAPL . Above the retention capacity, the NAPL becomes mobile and will move within the soil.

Ecotoxicity: No information available.

Persistence and degradability: Ingredient: diisononyl phthalate Persistence: Water/Soil: HIGH Persistence: Air: HIGHIngredient: titanium dioxide Persistence: Water/Soil: HIGH Persistence: Air: HIGHIngredient: 4,4'-diphenylmethane diisocyanate (MDI) Persistence: Water/Soil: LOW (Half-life = 1 days) Persistence: Air:LOW (Half-life = 0.24 days)Ingredient: silane Persistence: Water/Soil: LOW Persistence: Air:LOW

diisononyl phthalate Half-life in air: HIGH
titanium dioxide Half-life in air: HIGH
4,4'-diphenylmethane diisocyanate (MDI) Half-life in air: LOW (Half-life = 0.24 days)
silane Half-life in air: LOW
diisononyl phthalate Half-life in soil: HIGH
titanium dioxide Half-life in soil: HIGH
4,4'-diphenylmethane diisocyanate (MDI) Half-life in soil: LOW (Half-life = 1 days)
silane Half-life in soil: LOW
diisononyl phthalate Half-life in water: HIGH
titanium dioxide Half-life in water: HIGH
4,4'-diphenylmethane diisocyanate (MDI) Half-life in water: LOW (Half-life = 1 days)
silane Half-life in water: LOW

Bioaccumulative potential: Risk of bioaccumulation in an aquatic species is low. Ingredient: diisononyl phthalate Bioaccumulation: LOW (BCF = 183.8)Ingredient: titanium dioxide Bioaccumulation: LOW (BCF = 10)Ingredient: 4,4'-diphenylmethane diisocyanate (MDI) Bioaccumulation: LOW (BCF = 15)Ingredient: silane Bioaccumulation: LOW (LogKOW = 0.5294)Bioaccumulation:Bioaccumulation potential was characterized based on empirical and/or modelled data for a suite of petroleum hydrocarbons expected to occur in petroleum substances.Bioaccumulation factors (BAFs) are the preferred metric for assessing the bioaccumulation potential of substances, as the bioconcentration factor (BCF) may not adequately accountfor the bioaccumulation potential of substances via the diet, which predominates for substances with log Kow > ~4.5In addition to fish BCF and BAF data, bioaccumulation data for aquatic invertebrate species were also considered. Biota-sediment/soil accumulation factors (BSAFs), trophicmagnification factors and biomagnification factors were also considered in characterizing bioaccumulation potential.Overall, there is consistent empirical and predicted evidence to suggest that the following components have the potential for high bioaccumulation, with BAF/BCF values greater than5000: C13–C15 isoalkanes, C12 alkenes, C12–C15 one-ring cycloalkanes, C12 and C15 two-ring cycloalkanes, C14 polycycloalkanes, C15 one-ring aromatics, C15 and C20cycloalkane monoaromatics, C12–C13 diaromatics, C20 cycloalkane diaromatics, and C14 and C20 three-ring PAHsThese components are associated with a slow rate of metabolism and are highly lipophilic. Exposures from water and diet, when combined, suggest that the rate of uptake wouldexceed that of the total elimination rate. Most of these components are not expected to biomagnify in aquatic or terrestrial foodwebs, largely because a combination of metabolism, lowdietary assimilation efficiency and growth dilution allows the elimination rate to exceed the uptake rate from the diet; however,one study suggests that some alkyl-PAHs may biomagnify.While only BSAFs were found for some PAHs, it is possible that BSAFs will be > 1 for invertebrates, given that they do nothave the same metabolic competency as fish.In general, fish can efficiently metabolize aromatic compounds. There is some evidence that alkylation increases bioaccumulation of naphthalene but it is not known if this can begeneralized to larger PAHs or if any potential increase in bioaccumulation due to alkylation will be sufficient to exceed a

BAF/BCF of 5000.Some lower trophic level organisms (i.e., invertebrates) appear to lack the capacity to efficiently metabolize aromatic compounds, resulting in high bioaccumulation potential for somearomatic components as compared to fish.This is the case for the C14 three-ring PAH, which was bioconcentrated to a high level (BCF > 5000) by invertebrates but not by fish. There is potential for such bioaccumulativecomponents to reach toxic levels in organisms if exposure is continuous and of sufficient magnitude, though this is unlikely in the water column following a spill scenario due torelatively rapid dispersalBioaccumulation of aromatic compounds might be lower in natural environments than what is observed in the laboratory. PAHs may sorb to organic material suspended in the watercolumn (dissolved humic material), which decreases their overall bioavailability primarily due to an increase in size. This has been observed with fishEcotoxicity:Diesel fuel studies in salt water are available. The values varied greatly for aquatic species such as rainbow trout and Daphnia magna, demonstrating the inherent variability of dieselfuel compositions and its effects on toxicity. Most experimental acute toxicity values are above 1 mg/L. The lowest 48-hour LC50 for salmonids was 2.4 mg/L. Daphnia magna had a24-hour LC50 of 1.8 mg/.The values varied greatly for aquatic species such as rainbow trout and Daphnia magna, demonstrating the inherent variability of diesel fuel compositionsand its effects on toxicity. Most experimental acute toxicity values are above 1 mg/L. The lowest 48-hour LC50 for salmonids was 2.4 mg/L. Daphnia magna had a 24-hour LC50 of 1.8mg/LThe tropical mysid Metamysidopsis insularis was shown to be very sensitive to diesel fuel, with a 96-hour LC50 value of 0.22 mg/L this species has been shown to be as sensitive astemperate mysids to toxicants. However,However this study used nominal concentrations,and therefore was not considered acceptable. In another study involving diesel fuel, theeffect on brown or common shrimp (Crangon crangon) a 96-hour LC50 of 22 mg/L was determined. A “gas oil”was also tested and a 96-hour LC50 of 12 mg/L.was determinedThe steady state cell density of marine phytoplankton decreased with increasing concentrations of diesel fuel, with different sensitivities between species . The diatom Phaeodactylumtricornutum showed a 20% decrease in cell density in 24 hours following a 3 mg/L exposure with a 24-hour no-observed effect concentration (NOEC) of 2.5 mg/L. The microalgaIsochrysis galbana was more tolerant to diesel fuel, with a 24-hour lowest-observed-effect concentration (LOEC) of 26 mg/L (14% decrease in cell density), and a NOEC of 25 mg/L.Finally, the green algae Chlorella salina was relatively insensitive to diesel fuel contamination, with a 24-hour LOEC of 170 mg/L (27% decrease in cell density), and a NOEC of 160mg/L . All populations of phytoplankton returned to a steady state within 5 days of exposureIn sandy soils, earthworm (Eisenia fetida) mortality only occurred at diesel fuel concentrations greater than 10 000 mg/kg, which was also the concentration at which sub-lethal weightloss was recordedNephrotoxic effects of diesel fuel have been documented in several animal and human studies.Some species of birds (mallard ducks in particular) are generally resistant to the toxiceffects of petrochemical ingestion, and large amounts of petrochemicals are needed in order to cause direct mortality

Mobility: Low mobility in soil. Ingredient: diisononyl phthalate Mobility: LOW (KOC = 467200)Ingredient: titanium dioxide Mobility: LOW (KOC = 23.74)Ingredient: 4,4'-diphenylmethane diisocyanate (MDI) Mobility: LOW (KOC = 376200)Ingredient: silane Mobility: LOW (KOC = 14.3)

13. DISPOSAL CONSIDERATIONS

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.

14. TRANSPORT INFORMATION

ROAD AND RAIL TRANSPORT
Not classified as Dangerous Goods by the criteria of the "Australian Code for the Transport of Dangerous Goods by Road & Rail" and the "New Zealand NZS5433: Transport of Dangerous Goods on Land".

MARINE TRANSPORT
Not classified as Dangerous Goods by the criteria of the International Maritime Dangerous Goods Code (IMDG Code) for transport by sea.

AIR TRANSPORT
Not classified as Dangerous Goods by the criteria of the International Air Transport Association (IATA) Dangerous Goods Regulations for transport by air.

15. REGULATORY INFORMATION

This material is not subject to the following international agreements:
Montreal Protocol (Ozone depleting substances)
The Stockholm Convention (Persistent Organic Pollutants)
The Rotterdam Convention (Prior Informed Consent)
Basel Convention (Hazardous Waste)
International Convention for the Prevention of Pollution from Ships (MARPOL)

This material/constituent(s) is covered by the following requirements:

The Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) established under the Therapeutic Goods Act (Commonwealth): .

16. OTHER INFORMATION

Reason for issue: Product name change

This information was prepared in good faith from the best information available at the time of issue. It is based on the present level of research and to this extent we believe it is accurate. However, no guarantee of accuracy is made or implied and since conditions of use are beyond our control, all information relevant to usage is offered without warranty. The manufacturer will not be held responsible for any unauthorised use of this information or for any modified or altered versions.

If you are an employer it is your duty to tell your employees, and any others that may be affected, of any hazards described in this sheet and of any precautions that should be taken.

Safety Data Sheets are updated frequently. Please ensure you have a current copy.