ACTFLEX PU300 ACCELERATOR

FORSPEC PROTECTIVE COATINGS

Chemwatch Hazard Alert Code: 3

Chemwatch: **7938-15**
Version No: **2.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Issue Date: **07/03/2025**
Print Date: **10/03/2025**
L.GHS.AUS.EN.E

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

Product Identifier

Product name	ACTFLEX PU300 ACCELERATOR
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S.
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Crack repair for Polyurethane Waterproof.

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**FORSPEC PROTECTIVE COATINGS**
Address	22/872 Canterbury Rd. Roselands NSW 2196 Australia
Telephone	+61 2 8021 3517
Fax	Not Available
Website	www.forspec.com.au
Email	info@forspec.com.au

Emergency telephone number

Association / Organisation	**FORSPEC PROTECTIVE COATINGS**
Emergency telephone number(s)	0424 424178 (Mon-Fri 7.30am to 5pm; Sat 8.30am to 12.30pm)
Other emergency telephone number(s)	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

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

Chemwatch Hazard Ratings

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

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

Poisons Schedule	S6
Classification [1]	Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2A, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Carcinogenicity Category 1B, Reproductive Toxicity Category 1B, Hazardous to the Aquatic Environment Long-Term Hazard Category 2
Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI***

Label elements

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

Hazard statement(s)

Page 1 continued...

H315	Causes skin irritation.
H319	Causes serious eye irritation.
H335	May cause respiratory irritation.
H350	May cause cancer.
H360Df	May damage the unborn child. Suspected of damaging fertility.
H411	Toxic to aquatic life with long lasting effects.
AUH018	In use, may form flammable/explosive vapour/air mixture.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P337+P313	If eye irritation persists: Get medical advice/attention.
P391	Collect spillage.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
117-84-0	50-60	di-n-octyl phthalate
68-12-2	40-50	N,N-dimethylformamide
84-74-2	1-5	dibutyl phthalate
Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available***	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Immediately hold eyelids apart and flush the eye continuously with running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin or hair contact occurs: ▸ Immediately flush body and clothes with large amounts of water, using safety shower if available. ▸ Quickly remove all contaminated clothing, including footwear. ▸ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▸ Transport to hospital, or doctor.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay.
Ingestion	▸ If swallowed do **NOT** induce vomiting.

Continued...

	▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Seek medical advice. ▸ Avoid giving milk or oils. ▸ Avoid giving alcohol. ▸ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

Indication of any immediate medical attention and special treatment needed

As in all cases of suspected poisoning, follow the ABCDEs of emergency medicine (airway, breathing, circulation, disability, exposure), then the ABCDEs of toxicology (antidotes, basics, change absorption, change distribution, change elimination).
For poisons (where specific treatment regime is absent):

BASIC TREATMENT

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

ADVANCED TREATMENT

- Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- Positive-pressure ventilation using a bag-valve mask might be of use.
- Monitor and treat, where necessary, for arrhythmias.
- Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- Drug therapy should be considered for pulmonary oedema.
- Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- Treat seizures with diazepam.
- Proparacaine hydrochloride should be used to assist eye irrigation.

BRONSTEIN, A.C. and CURRANCE, P.L.
EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994
Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.
Treat symptomatically.
Depending on the degree of exposure to dimethylformamide, preplacement and periodic medical examination is desirable, especially evaluating the liver and kidney functions, and possible skin effects.

SECTION 5 Firefighting measures

Extinguishing media

- **DO NOT** use halogenated fire extinguishing agents.
- Foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.
- Water spray or fog - Large fires only.

Special hazards arising from the substrate or mixture

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

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	**WARNING:** In use may form flammable/ explosive vapour-air mixtures. ▸ Combustible. ▸ Slight fire hazard when exposed to heat or flame. ▸ Heating may cause expansion or decomposition leading to violent rupture of containers. ▸ On combustion, may emit toxic fumes of carbon monoxide (CO). ▸ May emit acrid smoke. ▸ Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO_2) nitrogen oxides (NOx) other pyrolysis products typical of burning organic material.
HAZCHEM	•3Z

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Continued...

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	Environmental hazard - contain spillage. ▸ 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. ▸ Clear area of personnel and move upwind. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by all means available, spillage from entering drains or water courses. ▸ Consider evacuation (or protect in place). ▸ No smoking, naked lights or ignition sources. ▸ Increase ventilation. ▸ Stop leak if safe to do so. ▸ Water spray or fog may be used to disperse / absorb vapour. ▸ Contain or absorb spill with sand, earth or vermiculite. ▸ Collect recoverable product into labelled containers for recycling. ▸ Collect solid residues and seal in labelled drums for disposal. ▸ Wash area and prevent runoff into drains. ▸ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▸ 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	▸ Store in original containers. ▸ Keep containers securely sealed. ▸ Store in a cool, dry, well-ventilated area. ▸ Store away from incompatible materials and foodstuff containers. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Metal can or drum ▸ Packaging as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▸ Many aprotic (non-hydroxylic) solvents are not inert towards other reagents and care must be taken when using untried combinations of solvents an reagents for the first time. ▸ Some aprotic solvents have a dramatic effect on reaction rates Phthalates: ▸ react with strong acids, strong oxidisers, permanganates and nitrates ▸ attack some form of plastics Dimethylformamide: ▸ Reacts violently with carbon tetrachloride (above 65 C), 2,5-bis-endo-dichloro-7-bicyclo[2.2.1]heptane, 2,5-dimethylpyrrole and phosphorous trichloride mixture, halogens, halogenated hydrocarbons and metal mixtures, highly halogenated compounds like hexachlorocyclohexane, particularly in the presence of iron or strong oxidisers (may cause fire and explosions), lithium azide, phosphorous trioxide, sodium and compounds. ▸ May polymerise violently with methylene diisocyanate. ▸ Reacts with borohydride and 2,4,6-trichloro-1,3,5-triazine. ▸ Reacts vigorously with strong oxidisers, halogens (e.g. Br2), nitrates (eg. Mg(NO3)2) and alkylaluminiums. ▸ Is incompatible with strong acids, allyl trifluoromethanesulfonate, ammonia, cresols, chromic anhydride, isocyanates, phenols, phosphorous trioxide. ▸ Reaction with inorganic acid chlorides, such as phosphorous oxychloride and thionyl chloride, may form dimethylcarbamoyl chloride, a suspect carcinogen. ▸ Will attack some forms of plastics, rubbers and coatings (polyethylene lined drums are safe to use). ▸ May attack brass, copper and its alloys, plastic, rubber and coatings. ▸ Avoid oxidising agents, acids, acid chlorides, acid anhydrides, chloroformates. ▸ Avoid cross contamination between the two liquid parts of product (kit). ▸ If two part products are mixed or allowed to mix in proportions other than manufacturer's recommendation, polymerisation with gelation and evolution of heat (exotherm) may occur. ▸ This excess heat may generate toxic vapour

Continued...

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	N,N-dimethylformamide	Dimethylformamide	10 ppm / 30 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	dibutyl phthalate	Dibutyl phthalate	5 mg/m3	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
di-n-octyl phthalate	Not Available	Not Available
N,N-dimethylformamide	500 ppm	Not Available
dibutyl phthalate	4,000 mg/m3	Not Available

MATERIAL DATA

Exposure controls

Appropriate engineering controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
Employers may need to use multiple types of controls to prevent employee overexposure.

- Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulated area.
- Work should be undertaken in an isolated system such as a "glove-box" . Employees should wash their hands and arms upon completion of the assigned task and before engaging in other activities not associated with the isolated system.
- Within regulated areas, the carcinogen should be stored in sealed containers, or enclosed in a closed system, including piping systems, with any sample ports or openings closed while the carcinogens are contained within.
- Open-vessel systems are prohibited.
- Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation.
- Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system.
- For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood.
- Except for outdoor systems, regulated areas should be maintained under negative pressure (with respect to non-regulated areas).
- Local exhaust ventilation requires make-up air be supplied in equal volumes to replaced air.
- Laboratory hoods must be designed and maintained so as to draw air inward at an average linear face velocity of 0.76 m/sec with a minimum of 0.64 m/sec. Design and construction of the fume hood requires that insertion of any portion of the employees body, other than hands and arms, be disallowed.

Individual protection measures, such as personal protective equipment



Eye and face protection

- Safety glasses with side shields.
- Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent]
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].

Skin protection

See Hand protection below

Hands/feet protection

- Wear chemical protective gloves, e.g. PVC.
- Wear safety footwear or safety gumboots, e.g. Rubber

NOTE:

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

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:
· frequency and duration of contact,
· chemical resistance of glove material,
· glove thickness and
· dexterity
Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).
· When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
· When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.

Continued...

	· Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. 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. ▸ Aprotic solvents may greatly promote the toxic properties of solutes because of their unique ability to penetrate synthetic rubber protective gloves and the skin (butyl rubber gloves are reported to be more satisfactory than others ▸ Neoprene rubber gloves
Body protection	See Other protection below
Other protection	▸ Employees working with confirmed human carcinogens should be provided with, and be required to wear, clean, full body protective clothing (smocks, coveralls, or long-sleeved shirt and pants), shoe covers and gloves prior to entering the regulated area. [AS/NZS ISO 6529:2006 or national equivalent] ▸ Employees engaged in handling operations involving carcinogens should be provided with, and required to wear and use half-face filter-type respirators with filters for dusts, mists and fumes, or air purifying canisters or cartridges. A respirator affording higher levels of protection may be substituted. [AS/NZS 1715 or national equivalent] ▸ Emergency deluge showers and eyewash fountains, supplied with potable water, should be located near, within sight of, and on the same level with locations where direct exposure is likely. ▸ Prior to each exit from an area containing confirmed human carcinogens, employees should be required to remove and leave protective clothing and equipment at the point of exit and at the last exit of the day, to place used clothing and equipment in impervious containers at the point of exit for purposes of decontamination or disposal. The contents of such impervious containers must be identified with suitable labels. For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. ▸ Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. ▸ Overalls. ▸ P.V.C apron. ▸ Barrier cream. ▸ Skin cleansing cream. ▸ Eye wash unit.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
"Forsberg Clothing Performance Index".
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
ACTFLEX PU300 ACCELERATOR

Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C
CPE	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
SARANEX-23	C
SARANEX-23 2-PLY	C
TEFLON	C
VITON	C
VITON/CHLOROBUTYL	C

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

Ansell Glove Selection

Respiratory protection

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

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

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

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

- Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

Continued...

Glove — *In order of recommendation*
AlphaTec 02-100
MICROFLEX® 93-260
MICROFLEX® NeoPro® NPG-888
AlphaTec® 38-612
AlphaTec® 53-001
AlphaTec® 58-005
AlphaTec® Solvex® 37-175
BioClean™ Emerald BENS
BioClean™ Extra BLAS
BioClean™ Fusion (Sterile) S-BFAP

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Clear (waster colour) liquid; does not mix with water.

Physical state	Liquid	**Relative density (Water = 1)**	0.95-1
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Applicable	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	1.026-10.256 @25C
Initial boiling point and boiling range (°C)	138	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	196-230	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Immiscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available
Heat of Combustion (kJ/g)	Not Available	**Ignition Distance (cm)**	Not Available
Flame Height (cm)	Not Available	**Flame Duration (s)**	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	**Enclosed Space Ignition Deflagration Density (g/m3)**	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

a) Acute Toxicity	Based on available data, the classification criteria are not met.
b) Skin Irritation/Corrosion	There is sufficient evidence to classify this material as skin corrosive or irritating.
c) Serious Eye Damage/Irritation	There is sufficient evidence to classify this material as eye damaging or irritating
d) Respiratory or Skin sensitisation	Based on available data, the classification criteria are not met.
e) Mutagenicity	Based on available data, the classification criteria are not met.
f) Carcinogenicity	There is sufficient evidence to classify this material as carcinogenic
g) Reproductivity	There is sufficient evidence to classify this material as toxic to reproductivity

Continued...

h) STOT - Single Exposure	There is sufficient evidence to classify this material as toxic to specific organs through single exposure
i) STOT - Repeated Exposure	Based on available data, the classification criteria are not met.
j) Aspiration Hazard	Based on available data, the classification criteria are not met.

Inhaled	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. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation. 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 and/or ingestion, of dimethylformamide (DMF), can lead to headache, dizziness, nausea, vomiting, anorexia and abdominal spasm. The use of alcoholic beverages accentuates the toxic effects and intolerance to alcohol can occur up to 4 days after exposure. Case reports in workers acutely exposed to DMF confirm that the liver is the target organ, with hepatic effects and associated disorders of the digestive system being reported. Symptoms include abdominal pain, anorexia, incoordination, and jaundice, as well as nausea, vomiting, and diarrhoea; nasal and skin irritation have also been reported Single exposures of 6 hours duration to air saturated with DMF vapour (approximately 5000 ppm) at room temperature killed rats and produced injury to lungs, liver and kidneys. At 2500 ppm rats were killed after five 6-hour exposures. At 20 ppm dogs survived more than 100 exposure periods of 6 hours and showed no gross pathology but did show a functional drop in systolic blood pressure. Inhalation hazard is increased at higher temperatures.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. At sufficiently high doses the material may be hepatotoxic (i.e. poisonous to the liver). Signs may include nausea, stomach pains, low fever, loss of appetite, dark urine, clay-coloured stools, jaundice (yellowing of the skin or eyes) Phthalates (aromatic dicarboxylic acid esters), in general, exhibit low toxicity, partly because of poor absorption but mainly as a result of rapid metabolism in which the esters are saponified to phthalic acid (which is rapidly excreted) and the parent alcohol (which is subsequently metabolised). The pathology of these compounds seems to be related to the released alcohol and its biological effects. The rate of absorption of ingested phthalate esters is influenced by the content of dietary fat. Ingested phthalate esters may to a lesser degree be absorbed as the monoester derivatives or in the case of di(2-ethylhexyl)phthalate, as the diester. Cumulative toxicity of the phthalates has been observed on repeated administration. Both di-n-octyl phthalate and di(2-ethylhexyl)phthalate were found to have 22-28 times greater toxicity (based on LD50s) following repeated administration to animals. The liver has been shown to be the target organ affected by the phthalates. In general phthalates have induced liver enlargement; this increase in liver weight has been attributed to rapid cell division (hyperplasia) along with the detachment of cells (hypertrophy). The increase in liver weight caused by phthalates has been found to reverse to normal or even below normal levels on prolonged exposure. Exposure to phthalates, in general, has been found to be associated with a reduction in circulating cholesterol and serum triglyceride levels which accounted for a reduction in liver steroidogenesis. The phthalates also effect carbohydrate metabolism in the liver producing depleted glycogen electron transport inhibitors following interaction with mitochondria. Testicular atrophy produced in rats during feeding studies depends on the length and structure of the alcohol; in general the lower molecular weight esters produce the more severe effects. The toxicity of phthalic acid isomers decreases in the order o-phthalic acid, isophthalic acid and terephthalic acid. Phthalic acid is not metabolised but is excreted, unchanged, in the urine and faeces. Terephthalic acid appears to potentiate the biological effects of substances such as antibiotics, thiamine and sulfonamides.
Skin Contact	The material produces mild skin irritation; evidence exists, or practical experience predicts, that the material either ▸ produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or ▸ produces significant, but mild, 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. DMF is extensively absorbed through the skin, its metabolism and kinetics are well known, and urinary metabolites exist that can be accurately measured. As a result, biological monitoring has been extensively used in the assessment of the absorbed amounts in occupationally exposed populations. The metabolite most often analysed is ***N***-methylformamide (NMF) In one case of accidental exposure, to dimethylformamide. the patient showed itching, hyperaemia, abdominal pain, vomiting and increased blood pressure. There are inconclusive reports of human sensitisation Irritation and skin reactions are possible with sensitive skin 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. Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.
Eye	Evidence exists, or practical experience predicts, that the material may cause severe 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. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur. The liquid may produce eye discomfort and is capable of causing temporary impairment of vision and/or transient eye inflammation, ulceration
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals. On the basis, primarily, of animal experiments, the material may be regarded as carcinogenic to humans. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in cancer on the basis of: - appropriate long-term animal studies - other relevant information There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of: - clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects. 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. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. 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,

Continued...

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'.
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)
Liver injury has been produced in cats after prolonged inhalation of 100 ppm dimethylformamide (DMF) (but not rats).
Leather tannery and airframe repair shop workers exposed to DMF and a number of other chemicals showed an increased number of testicular cancers above the number of expected cases. The available data however does not conclusively establish DMF as a carcinogen.
The literature contains citations indicating DMF increases the rate of embryonic mortalities in animals at high maternal doses.
Oral or intraperitoneal administration of dibutyl phthalate, at high doses relative to the TLV, produced a number of resorptions, neural tube defects, skeletal abnormalities and increased foetal deaths.

ACTFLEX PU300 ACCELERATOR

TOXICITY	IRRITATION
Not Available	Not Available

di-n-octyl phthalate

TOXICITY	IRRITATION
Dermal (Guinea Pig) LD50: >5000 mg/kg[2]	Eye (Rodent - rabbit): 20mg - Severe
Oral (Mouse) LD50; 6513 mg/kg[2]	Eye (Rodent - rabbit): 500mg/24H - Mild
	Skin (Rodent - rabbit): 500mg/24H - Mild

N,N-dimethylformamide

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >500 mg/kg[2]	Eye (Rodent - rabbit): 0.1mL
Inhalation (Rat) LC50: >5.85 mg/l4h[1]	Eye (Rodent - rabbit): 0.1mL - Severe
Oral (Rat) LD50: 2800 mg/kg[2]	Eye (Rodent - rabbit): 100% - Severe
	Eye (Rodent - rabbit): 100mg - Severe
	Eye: adverse effect observed (irritating)[1]
	Skin (Human - woman): 0.1%

Continued...

		Skin (Human): 100%/24H - Mild
		Skin: no adverse effect observed (not irritating)[1]
dibutyl phthalate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation (Rat) LC50: >=15.68 mg/l4h[1]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: 8000 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***

DI-N-OCTYL PHTHALATE

Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.
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 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)

Continued...

	*711P - C7,C11, branched and linear esters (CAS Rn: 111381-90-9) * DTDP - di-C11-14, C13 rich ester (CAS 68515-47-9)
N,N-DIMETHYLFORMAMIDE	For dimethylformamide (DMF): In humans, DMF is absorbed by inhalation and through the skin. After high exposures (up to 60 ppm) headaches, abdominal pain, nausea, vomiting, dizziness, elevated liver enzymes, and alcohol intolerance (facial flashing and palpitations) were seen. Case reports of testicular cancer in aircraft repair and leather tannery facilities failed to be confirmed in further studies. Reports of DNA and chromosomal damage in peripheral lymphocytes of subjects exposed to DMF either failed to take into account smoking as a confounder or coexposure to other chemicals. Acute toxicity: N,N-dimethylformamide (DMF) is of low acute toxicity in mammals: LD50 rat (oral) 3040 mg/kg bw, LC50 rat (inhalative, 4 h) > 5900 mg/m3, LD50 rat (dermal) > 3160 mg/kg bw. Main symptoms following exposure were apathy and staggering (oral) and irregular or intermittent respiration (inhalation). It was irritating to the eyes of rabbits but not irritating to the skin of rabbits and rats. DMF did not show a sensitizing potential when used as a vehicle in a local lymph node assay. Repeat dose toxicity: In repeated-dose toxicity studies in rats and mice with chronic exposure over 2 years (rats) or 18 months (mice) and subchronic exposure over 13 weeks by inhalation, or in rats treated by oral administration of DMF (90 day feeding study or administration by gavage for 28 days), the predominant target organ was the liver (NOAEC: chronic inhalation rat: 25 ppm (about 80 mg/m3), LOAEC: chronic inhalation mouse: 25 ppm (about 80 mg/m3); NOAEC: subchronic inhalation rat: 100 ppm, mouse: 400 ppm (about 300 mg/m3 and 1210 mg/m3, respectively); NOAEL: rat, 90 days 200 ppm (about 12 mg/kg bw/day), 28 days about 238 mg/kg bw/day). In a 13-week inhalation study with a limited number of Cynomolgus monkeys no treatment-related effects occurred (NOAEC: 500 ppm (about 1500 mg/m3)). Genetic toxicity: DMF does not induce chromosome aberrations or gene mutations in various test systems in vivo and in vitro. In addition, no increased tumor incidence was found in carcinogenicity studies in rats and mice that were exposed to 25, 100 and 400 ppm DMF (about 80, 300, and 1210 mg/m3) by inhalation for 2 years or 18 months, respectively. Reproductive and developmental toxicity: Reproductive toxicity was observed at the presence of some general toxicity in a continuous breeding study in mice, when DMF was administered orally in the drinking water at doses of 1000, 4000 and 7000 ppm (about 219, 820 and 1455 mg/kg bw/day). The maximal tolerated dose for generalised toxicity was 1000 ppm (about 219 mg/kg bw/day) for the F0 and the F1 generation, thus a systemic NOAEL could not be determined. Significant reproductive toxicity (e.g. reduced fertility and fecundity characterized by reduced pregnancy and mating index (the latter one only in the high dose group), reduced number of litters, reduced average litter size and for the F1 parental males by effects on prostate weight and epididymal spermatozoa concentration, the latter finding only in the high dose group) and developmental toxicity (e.g. reduced survival and growth of pups, increase in craniofacial and sternebral malformations) occurred at 4000 ppm and above. At 1000 ppm, reduced pup weights were found in F2 pups. Thus 1000 ppm (about 219 mg/kg bw/day) was the NOAEL for reproductive and developmental toxicity in F0 and F1, and the LOAEL for developmental toxicity in F2. Developmental toxicity and teratogenicity occurred in rats and rabbits in various studies (inhalation, oral- or dermal administration) and in mice (oral administration). In rats embryo/ foetotoxicity and teratogenicity were mostly seen at maternally toxic doses, whereas in mice and in rabbits embryo-/foetotoxicity and teratogenicity occurred also at dose levels without maternal toxicity. However, the rabbit appeared to be the most sensitive species to the developmental toxic effects of DMF. **WARNING:** This substance has been classified by the IARC as Group 2A: Probably Carcinogenic to Humans.
DIBUTYL PHTHALATE	For dibutyl phthalate (DBP): In studies on rats, DBP is absorbed through the skin, although in ***in vitro*** studies human skin has been found to be less permeable than rat skin to this compound. Studies in laboratory animals indicate that DBP is rapidly absorbed from the gastrointestinal tract, distributed primarily to the liver and kidneys of rats and excreted in urine as metabolites following oral or intravenous administration. Following inhalation, it was consistently detected at low concentrations in the brain. Available data indicate that in rats, following ingestion, DBP is metabolised by nonspecific esterases mainly in the small intestine to yield mono- ***n***-butyl phthalate (MBP) with limited subsequent biochemical oxidation of the alkyl side chain of MBP. MBP is stable and resistant to hydrolysis of the second ester group. Accumulation has not been observed in any organ. The profile of effects following exposure to DBP is similar to that of other phthalate esters, which, in susceptible species, can induce hepatomegaly, increased numbers of hepatic peroxisomes, foetotoxicity, teratogenicity and testicular damage. **Acute toxicity:** The acute toxicity of DBP in rats and mice is low. Signs of acute toxicity in laboratory animals include depression of activity, laboured breathing and lack of coordination. In a case of accidental poisoning of a worker who ingested approximately 10 grams of DBP, recovery was gradual within two weeks and complete after 1 month. On the basis of limited available data in animal species, DBP appears to have little potential to irritate skin or eyes or to induce sensitization. In humans, a few cases of sensitization after exposure to DBP have been reported, although this was not confirmed in controlled studies of larger numbers of individuals reported only in secondary accounts **Repeat dose toxicity:** In short-term repeated-dose toxicity studies, effects at lowest levels in rats after oral administration for 5 to 21 days included peroxisome proliferation and hepatomegaly at doses of 420 mg/kg body weight per day or more. In longer-term studies, the effects in rats observed following ingestion of DBP for periods up to 7 months included reduced rate of weight gain at doses of 250 mg/kg body weight per day or more. Increase in relative liver weight has been observed at doses of 120 mg/kg body weight or more. Peroxisomal proliferation with increased peroxisomal enzyme activity has been observed at doses of 279 mg/kg body weight per day or more. Necrotic hepatic changes in Wistar rats have been reported at doses of 250 mg/kg body weight per day or more but not in F-344 or Sprague-Dawley rats exposed to up to 2500 mg/kg body weight per day. Alteration in testicular enzymes and degeneration of testicular germinal cells of rats have been observed at doses of 250 and 571 mg/kg body weight per day. There are considerable species differences in effects on the testes following exposure to DBP, minimal effects being observed in mice and hamsters at doses as high as 2000 mg/kg body weight per day. In mice, effects on body and organ weights and histological alterations in the liver indicative of metabolic stress have been reported in a recent subchronic bioassay, for which the no-observed-effect-level (NOEL) was 353 mg/kg body weight per day. **Developmental toxicity:** . In a continuous breeding protocol, which included cross-over mating and offspring assessment phases, rats were exposed to 0, 1000, 5000 or 10 000 mg DBP/kg in the diet (equivalent to 0, 66, 320 and 651 mg/kg body weight per day). In the first generation the reduction in pup weight in the mid-dose group, in the absence of any adverse effect on maternal weight, could be regarded as a developmental toxicity effect. There was also a significant reduction of live litter numbers at all three dose levels. The effects in the second generation were more severe, with reduced pup weight in all groups including the low-dose group, structural defects (such as prepucial/ penile malformations, seminiferous tubule degeneration, and absence or underdevelopment of the epididymides) in the mid- and high-dose groups, and severe effects on spermatogenesis in the high-dose group that were not seen in the parent animals. These results suggest that the adverse effects of DBP are more marked in animals exposed during development and maturation than in animals exposed as adults only. No clear NOEL was established in this study. The lowest-observed- adverse-effect-level (LOAEL) was considered to be 66 mg/kg body weight per day. The available studies show that DBP generally induces foetotoxic effects in the absence of maternal toxicity. Available data also indicate that DBP is teratogenic at high doses and that susceptibility to teratogenesis varies with developmental stage and period of administration. In mice, DBP caused dose-dependent increases in the number of resorptions and dead fetuses at oral doses of 400 mg/kg body weight per day or more. Dose-dependent decreases in fetal weights and number of viable litters were also observed in mice at these doses. Adequate carcinogenesis bioassays for DBP have not been conducted. The weight of the available evidence indicates that DBP is not genotoxic. **Transitional Phthalate Esters:** produced from alcohols with straight-chain carbon backbones of C4 to C6. This subcategory also includes a phthalate produced from benzyl alcohol as one ester group with the second ester composed of an alkyl group with a C5 carbon backbone and butyrate group. Phthalate esters containing >10% C4 to C6 molecules were conservatively included in this subcategory. Branched C7 and C8 isomers (di-iso-heptyl, di-iso-octyl and diethylhexyl phthalates) in contrast to linear dihexyl and dioctyl phthalate are members of this family. Transitional phthalates have varied uses, but are largely used as plasticisers for PVC. Physicochemical properties also vary in that the lower molecular weight transitional phthalates are more water-soluble than higher molecular weight transitional phthalates, but none would be characterised as highly water soluble. Transitional phthalates have lower water solubility than the low molecular weight phthalates and except for butylbenzyl phthalate (BBP), existing data suggest they do not exhibit acute or chronic aquatic toxicity. What distinguishes some of the transitional phthalates from others is their greater mammalian toxicity potential, particularly with regard to reproductive and developmental effects, compared to either the low or high molecular weight phthalate subcategories **Acute Toxicity.** The available data on phthalates spanning the carbon range from C4 to C6 indicate that phthalate esters in the transitional subcategory are minimally toxic by acute oral and dermal administration. The oral LD50 value for BBP exceeds 2 g/ kg, and for materials with higher molecular weights, the LD50 values exceed the maximum amounts which can be administered to the animals in a manner consistent with the principles of responsible animal use. One member of this subcategory, diethylhexyl phthalate (DEHP), has been tested for acute inhalation toxicity. It did not cause an effect at the highest concentration tested. Further, considering the low volatility of these substances, inhalation exposure at toxicologically significant levels is not anticipated.

Continued...

	Repeated Dose Toxicity. Several substances in the C4 to C6 range, including BBP, have been tested for repeated dose toxicity in studies ranging from 3 weeks to 2 years . The principal effects found in these studies were those associated with peroxisome proliferation including liver enlargement and induction of peroxisomal enzymes. As shown in a comparative study of liver effects, the strongest inducers of peroxisome proliferation are diisononyl phthalate (DINP) and di-iso-decyl phthalate (DIDP) with substances of shorter chain length (e.g., BBP) showing much less pronounced effects. Thus it is reasonable to conclude that other members of this subcategory would show effects similar to BBP and less pronounced than DINP or DIDP. It should also be noted that the relevance of these findings to human health is, at best, questionable. It has been shown that these effects are mediated through the peroxisome proliferation-activated receptor alpha (PPARa) and that levels of PPARa are much higher in rodents than they are in humans. Thus one would expect humans to be substantially less responsive than rodents to peroxisome proliferating agents. Empirical evidence that this is true is provided by studies in primates in which repeated administration of DINP had no effects on liver, kidney or testicular parameters. Several of the substances in the transitional phthalate esters subcategory, however, have been shown to produce testicular atrophy when given to juvenile rats at high levels. Testicular atrophy has been associated with BBP and other substances with C4 to C6 linear carbon chains. However, molecules with fewer than 4 or more than 6 carbons did not produce testicular atrophy in these studies. Although the relevance of these data are uncertain, as the testes is not a target organ for diethylhexyl phthalate (DEHP) in primates, these data do provide one of the distinguishing toxicological characteristics of this subcategory and are one of the underlying reasons supporting the differentiation of phthalate esters on the basis of length of the linear region of the carbon chain. **Genetic Toxicity (Salmonella)**. A number of the substances in this subcategory including the reference substance BBP has been assessed in the Salmonella and mouse lymphoma assays. All of these substances were inactive in these assays. **Chromosomal Aberrations**. BBP and dihexyl phthalate (DHP) were inactive in micronucleus assays in mice. DEHP was inactive in a cytogenetics assay in rat bone marrow. Diisoheptyl phthalate was inactive in CHO cells, in vitro.. **Reproductive toxicity:** A series of studies assessed the structure-activity relationship of the effects of phthalate esters on fertility using a continuous breeding protocol . The test substances included in these studies were diethyl-, dipropyl-, dibutyl-, dipentyl-, d-n-hexyl-, di-2(ethylhexyl)-, and di-n-octyl phthalates. The most profound effects were on fertility (i.e., number of females delivering/ number mated) and number of live births. The substance showing the greatest activity was DEHP which produced effects at dietary levels of 0.1 % with a no effect level of 0.01 %. The next most active compounds were di-n-hexyl- and di-n-pentyl phthalate which showed effects in the range of 0.3 to 0.5 %; no effect levels were not experimentally defined. Dipropyl phthalate had an effect on live birth index at 2.5 % but produced no effects at 1.25 %. Diethyl phthalate and di-n-octyl phthalate were inactive at the highest levels tested, 2.5 % and 5.0 %, respectively. These data demonstrated that molecules with linear alkyl chains of 4 to **6** carbons profoundly affect fertility in rodents, with DEHP being the most active. Molecules with longer or shorter side chains are essentially inactive in these assays. These data were also a basis for the separation of phthalates into three categories based on length of side chain. In addition to these data there are reproductive toxicity studies on BBP and DEHP . A 2-generation reproductive study was conducted in rats in which BBP was administered via the diet. Parental effects were limited to changes in body weight, weight gain, and increased absolute and relative liver weights. In the F1 parents, treatment with BBP affected mating and fertility indices and sperm number and motility. The F1 male offspring exhibited shortened anogenital distance, delayed acquisition of puberty and retention of nipples and areolae as well as reproductive effects. The NOAEL of the study was reported to be 3750 mg/ kg for reproductive effects. However, for male F1 and F2 offspring, the NOEL for reproductive effects was reported to be 50 mg/ kg based on reductions in anogenital distance. These studies along with previous data provide a good basis to assess the reproductive effects of C4 to C6 phthalate esters. Although several substances (diheptyl, heptyl nonyl, heptyl undecyl) have ester side chain constituents that predominately fall in the high molecular weight subcategory, these substances are conservatively assumed to exhibit reproductive effects similar to other transitional phthalates . **Developmental toxicity:** There have been extensive studies of the developmental toxicity of BBP and DEHP. These substances produce structural malformations and also affect male reproductive development. No effect levels are in the range of 50 to 300 mg/ kg bw/ day. There is also an unpublished developmental toxicity study of di-isoheptyl phthalate (DIHP). The results of these studies are broadly consistent with the structure-activity relationships previously described, i.e., that phthalate esters with linear carbon chains of C4 to C6 carbons produce much more profound effects that either shorter or longer molecules. Phthalate esters with >10% C4 to C6 isomers were conservatively placed in the transitional subcategory. This conclusion is supported by developmental test data on "711P*" (which showed structural malformations in rats at 1000 mg/ kg/ day with a NOAEL of 200 mg/ kg/ day ."711P" is an equal composition mixture of six phthalate esters consisting of linear and methyl-branched C7, C9, and C11 ester side chains. This test substance is considered by EPA under the following CAS Numbers.: 68515-44-6 (di C7), 68515-45-7 (di C9), 3648-20-2 (di C1 I), 111381-89-6 (C7, C9), 111381-90-9 (C7, C11), and 111381-91-0 (C9, C11). The overall content of C4 to C6 isomers in "71 1P" is approximately 10%, based on the contribution from methyl-branched C7 isomers e.g., di C7 (30% C4-C6); C7, C9 (15% C4-C6); and C7, C11 (15 % C4-C6). Test data on 711P were used selectively as read-across data to the C7-containing substances in the mixture, based on the C4 to C6 content of each substance in the mixture.
DI-N-OCTYL PHTHALATE & DIBUTYL PHTHALATE	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.
DI-N-OCTYL PHTHALATE & N,N-DIMETHYLFORMAMIDE	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

Acute Toxicity	✗	**Carcinogenicity**	✓
Skin Irritation/Corrosion	✓	**Reproductivity**	✓
Serious Eye Damage/Irritation	✓	**STOT - Single Exposure**	✓
Respiratory or Skin sensitisation	✗	**STOT - Repeated Exposure**	✗
Mutagenicity	✗	**Aspiration Hazard**	✗

Legend: ✗ *– Data either not available or does not fill the criteria for classification*
✓ *– Data available to make classification*

SECTION 12 Ecological information

Toxicity

ACTFLEX PU300 ACCELERATOR	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available
di-n-octyl phthalate	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**

Continued...

	NOEC(ECx)	0.08h	Fish	0.03-0.04mg/L	4
	LC50	96h	Fish	>0.045mg/l	4

N,N-dimethylformamide					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	96h	Algae or other aquatic plants	>500mg/l	1
	BCF	1344h	Fish	0.3-0.8	7
	EC50	72h	Algae or other aquatic plants	>1000mg/l	2
	NOEC(ECx)	24h	Crustacea	0.03mg/L	4
	LC50	96h	Fish	5714-18967mg/L	4
	EC50	48h	Crustacea	>100mg/l	1

dibutyl phthalate					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	BCF	1344h	Fish	3.1-21.2	7
	EC50	72h	Algae or other aquatic plants	1.2mg/l	1
	EC50	96h	Algae or other aquatic plants	0.003mg/L	4
	EC50	48h	Crustacea	3.4mg/l	1
	NOEC(ECx)	72h	Algae or other aquatic plants	0.5mg/l	1
	ErC50	72h	Algae or other aquatic plants	1.2mg/l	1
	LC50	96h	Fish	0.28-0.44mg/l	4

Legend:	***Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 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***

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

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
di-n-octyl phthalate	HIGH (Half-life = 365 days)	LOW (Half-life = 1.87 days)
N,N-dimethylformamide	LOW	LOW
dibutyl phthalate	LOW (Half-life = 23 days)	LOW (Half-life = 3.08 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
di-n-octyl phthalate	LOW (LogKOW = 8.1)
N,N-dimethylformamide	LOW (BCF = 1.2)
dibutyl phthalate	LOW (BCF = 176)

Mobility in soil

Ingredient	Mobility
di-n-octyl phthalate	LOW (Log KOC = 195500)
N,N-dimethylformamide	MEDIUM (Log KOC = 2.411)
dibutyl phthalate	LOW (Log KOC = 1460)

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. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information

Labels Required

Continued...

	
Marine Pollutant	
HAZCHEM	•3Z

Land transport (ADG)

14.1. **UN number or ID number**	3082
14.2. **UN proper shipping name**	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S.
14.3. **Transport hazard class(es)**	Class: 9 Subsidiary Hazard: Not Applicable
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: 274 331 335 375 AU01 Limited quantity: 5 L

Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082
are not subject to this Code when transported by road or rail in;
(a) packagings;
(b) IBCs; or
(c) any other receptacle not exceeding 500 kg(L).
- Australian Special Provisions (SP AU01) - ADG Code 7th Ed.

Air transport (ICAO-IATA / DGR)

14.1. **UN number**	3082
14.2. **UN proper shipping name**	Environmentally hazardous substance, liquid, n.o.s.
14.3. **Transport hazard class(es)**	ICAO/IATA Class: 9 ICAO / IATA Subsidiary Hazard: Not Applicable ERG Code: 9L
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: A97 A158 A197 A215 Cargo Only Packing Instructions: 964 Cargo Only Maximum Qty / Pack: 450 L Passenger and Cargo Packing Instructions: 964 Passenger and Cargo Maximum Qty / Pack: 450 L Passenger and Cargo Limited Quantity Packing Instructions: Y964 Passenger and Cargo Limited Maximum Qty / Pack: 30 kg G

Sea transport (IMDG-Code / GGVSee)

14.1. **UN number**	3082
14.2. **UN proper shipping name**	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S.
14.3. **Transport hazard class(es)**	IMDG Class: 9 IMDG Subsidiary Hazard: Not Applicable
14.4. **Packing group**	III
14.5 **Environmental hazard**	Marine Pollutant
14.6. **Special precautions for user**	EMS Number: F-A , S-F Special provisions: 274 335 969 Limited Quantities: 5 L

14.7. Maritime transport in bulk according to IMO instruments

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

Not Applicable

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

Continued...

Product name	Group
di-n-octyl phthalate	Not Available
N,N-dimethylformamide	Not Available
dibutyl phthalate	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
di-n-octyl phthalate	Not Available
N,N-dimethylformamide	Not Available
dibutyl phthalate	Not Available

SECTION 15 Regulatory information

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

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

Australian Inventory of Industrial Chemicals (AIIC)

N,N-dimethylformamide is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2A: Probably carcinogenic to humans

dibutyl phthalate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (di-n-octyl phthalate; N,N-dimethylformamide; dibutyl phthalate)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory 'Active'
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	***Yes = All CAS declared ingredients are on the inventory*** ***No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.***

SECTION 16 Other information

Revision Date	07/03/2025
Initial Date	07/03/2025

SDS Version Summary

Version	Date of Update	Sections Updated
2.1	07/03/2025	Toxicological information - Acute Health (inhaled), Toxicological information - Acute Health (skin), Toxicological information - Acute Health (swallowed), Physical and chemical properties - Appearance, Toxicological information - Chronic Health, Hazards identification - Classification, Disposal considerations - Disposal, Ecological Information - Environmental, Exposure controls / personal protection - Exposure Standard, Handling and storage - Storage (storage incompatibility), Toxicological information - Toxicity and Irritation (Other), Identification of the substance / mixture and of the company / undertaking - Use

Other information

Continued...

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

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

Definitions and abbreviations

- PC—TWA: Permissible Concentration-Time Weighted Average
- PC—STEL: Permissible Concentration-Short Term Exposure Limit
- IARC: International Agency for Research on Cancer
- ACGIH: American Conference of Governmental Industrial Hygienists
- STEL: Short Term Exposure Limit
- TEEL: Temporary Emergency Exposure Limit。
- IDLH: Immediately Dangerous to Life or Health Concentrations
- ES: Exposure Standard
- OSF: Odour Safety Factor
- NOAEL: No Observed Adverse Effect Level
- LOAEL: Lowest Observed Adverse Effect Level
- TLV: Threshold Limit Value
- LOD: Limit Of Detection
- OTV: Odour Threshold Value
- BCF: BioConcentration Factors
- BEI: Biological Exposure Index
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration
- MARPOL: International Convention for the Prevention of Pollution from Ships
- IMSBC: International Maritime Solid Bulk Cargoes Code
- IGC: International Gas Carrier Code
- IBC: International Bulk Chemical Code

- AIIC: Australian Inventory of Industrial Chemicals
- DSL: Domestic Substances List
- NDSL: Non-Domestic Substances List
- IECSC: Inventory of Existing Chemical Substance in China
- EINECS: European INventory of Existing Commercial chemical Substances
- ELINCS: European List of Notified Chemical Substances
- NLP: No-Longer Polymers
- ENCS: Existing and New Chemical Substances Inventory
- KECI: Korea Existing Chemicals Inventory
- NZIoC: New Zealand Inventory of Chemicals
- PICCS: Philippine Inventory of Chemicals and Chemical Substances
- TSCA: Toxic Substances Control Act
- TCSI: Taiwan Chemical Substance Inventory
- INSQ: Inventario Nacional de Sustancias Químicas
- NCI: National Chemical Inventory
- FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

This document is copyright.
Apart from any fair dealing for the purposes of private study, research, review or criticism, as permitted under the Copyright Act, no part may be reproduced by any process without written permission from CHEMWATCH.
TEL (+61 3) 9572 4700.