



Diamond Coat

Infinity Innovative Coatings

Part Number: **Not Available**

Version No: **1.2**

Safety Data Sheet (Conforms to Annex II of REACH (1907/2006) - Regulation 2020/878)

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L.REACH.GBR.EN

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

1.1. Product Identifier

Product name	Diamond Coat
Synonyms	Not Available
Other means of identification	Not Available

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

Relevant identified uses	Not Available
Uses advised against	No specific uses advised against are identified.

1.3. Details of the manufacturer or supplier of the safety data sheet

Registered company name	Infinity Innovative Coatings
Address	Not Available Not Available Not Available Not Available United Kingdom
Telephone	Not Available
Fax	Not Available
Website	Not Available
Email	Not Available

1.4. Emergency telephone number

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

SECTION 2 Hazards identification

2.1. Classification of the substance or mixture

Classification according to regulation (EC) No 1272/2008 [CLP] and amendments [1]	H317 - Sensitisation (Skin) Category 1, H341 - Germ Cell Mutagenicity Category 2, H412 - Hazardous to the Aquatic Environment Long-Term Hazard Category 3
Legend:	1. Classification by vendor; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

2.2. Label elements

Hazard pictogram(s)	
Signal word	Warning

Hazard statement(s)

H317	May cause an allergic skin reaction.
H341	Suspected of causing genetic defects.
H412	Harmful to aquatic life with long lasting effects.

Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P280	Wear protective gloves and protective clothing.
P261	Avoid breathing dust/fumes.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

P405	Store locked up.
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Precautionary statement(s) Disposal

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

naphtha petroleum, light aromatic solvent	Determined to have endocrine-disrupting properties according to Europe Regulation (EU) 528/2012, Europe Regulation (EU) 2017/2100, and Europe Regulation (EU) 2018/605
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SECTION 3 Composition / information on ingredients

3.1.Substances

See 'Composition on ingredients' in Section 3.2

3.2.Mixtures

1. CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classification according to regulation (EC) No 1272/2008 [CLP] and amendments	SCL / M-Factor	Nanoform Particle Characteristics
1. 136210-30-5 2.429-270-1 3.607-521-00-8 4.Not Available	80	<u>aspartic acid</u> , <u>N,N'-(methylenedicyclohexanediy)bis-,ester</u>	Sensitisation (Skin) Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 3; H317, H412 [2]	Not Available	Not Available
1. 136210-32-7 2.412-060-9 3.607-350-00-9 4.Not Available	11	<u>aspartic acid</u> , <u>N,N'-(methylenedicyclohexanediy)bis-,ester</u>	Sensitisation (Skin) Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 3; H317, H412 [2]	Not Available	Not Available
1. 64742-95-6. 2.247-093-6 265-199-0 3.649-356-00-4 4.Not Available	1	<u>naphtha petroleum, light aromatic solvent [e]</u>	Flammable Liquids Category 3, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Hazardous to the Aquatic Environment Long-Term Hazard Category 2; H226, H336, H411, EUH066 [1]	Not Available	Not Available
1. 57834-33-0 2.260-976-0 3.Not Available 4.Not Available	1	<u>N-(ethoxycarbonylphenyl)-N'-methyl-N'-phenylformamidine</u>	Acute Toxicity (Oral) Category 4, Sensitisation (Skin) Category 1, Germ Cell Mutagenicity Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 1; H302, H317, H341, H410 [1]	Not Available	Not Available
1. 108-32-7 2.203-572-1 3.607-194-00-1 4.Not Available	5	<u>propylene carbonate</u>	Serious Eye Damage/Eye Irritation Category 2; H319 [2]	Not Available	Not Available
1. 7631-86-9 2.231-545-4 3.Not Available 4.Not Available	2	<u>silica amorphous</u>	EUH210 [1]	Not Available	Not Available

Legend: 1. Classification by vendor; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 3. Classification drawn from C&L; * EU IOELVs available; [e] Substance identified as having endocrine disrupting properties

SECTION 4 First aid measures

4.1. Description of first aid measures

Eye Contact	▸ Generally not applicable.
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. ▸ Generally not applicable.
Inhalation	▸ Generally not applicable.
Ingestion	▸ Generally not applicable.

4.2 Most important symptoms and effects, both acute and delayed

See Section 11

4.3. Indication of any immediate medical attention and special treatment needed

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

- Primary threat to life, from pure petroleum distillate ingestion and/or inhalation, is respiratory failure.
- Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO₂ 50 mm Hg) should be intubated.
- Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial injury has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
- A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
- Epinephrine (adrenalin) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.
- Lavage is indicated in patients who require decontamination; ensure use of cuffed endotracheal tube in adult patients. [Ellenhorn and Barceloux: Medical Toxicology]

Treat symptomatically.

SECTION 5 Firefighting measures

5.1. Extinguishing media

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

5.2. Special hazards arising from the substrate or mixture

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

Fire Fighting	<ul style="list-style-type: none"> ▸ When silica dust is dispersed in air, firefighters should wear inhalation protection as hazardous substances from the fire may be adsorbed on the silica particles. ▸ When heated to extreme temperatures, (>1700 deg.C) amorphous silica can fuse. ▸ 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 courses. ▸ Use water delivered as a fine spray to control fire and cool adjacent area. ▸ 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. ▸ Equipment should be thoroughly decontaminated after use. <p>Slight hazard when exposed to heat, flame and oxidisers.</p>
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▸ When silica dust is dispersed in air, firefighters should wear inhalation protection as hazardous substances from the fire may be adsorbed on the silica particles. ▸ When heated to extreme temperatures, (>1700 deg.C) amorphous silica can fuse. <p>Combustible. Will burn if ignited. Combustion products include: carbon monoxide (CO) carbon dioxide (CO₂) nitrogen oxides (NO_x) silicon dioxide (SiO₂) other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes. Articles and manufactured articles may constitute a fire hazard where polymers form their outer layers or where combustible packaging remains in place. Certain substances, found throughout their construction, may degrade or become volatile when heated to high temperatures. This may create a secondary hazard. WARNING: Long standing in contact with air and light may result in the formation of potentially explosive peroxides.</p>

SECTION 6 Accidental release measures

6.1. Personal precautions, protective equipment and emergency procedures

See section 8

6.2. Environmental precautions

See section 12

6.3. Methods and material for containment and cleaning up

Minor Spills	<ul style="list-style-type: none"> ▶ Clean up all spills immediately. ▶ Secure load if safe to do so. ▶ Bundle/collect recoverable product. ▶ Collect remaining material in containers with covers for disposal.
Major Spills	<ul style="list-style-type: none"> ▶ 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. <p>Minor hazard.</p> <ul style="list-style-type: none"> ▶ Clear area of personnel. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Control personal contact with the substance, by using protective equipment as required. ▶ Prevent spillage from entering drains or water ways. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Absorb remaining product with sand, earth or vermiculite and place in appropriate containers for disposal. ▶ Wash area and prevent runoff into drains or waterways. ▶ If contamination of drains or waterways occurs, advise emergency services. ▶ Clean up all spills immediately. ▶ Wear protective clothing, safety glasses, dust mask, gloves. ▶ Secure load if safe to do so. Bundle/collect recoverable product. ▶ Use dry clean up procedures and avoid generating dust. ▶ Vacuum up (consider explosion-proof machines designed to be grounded during storage and use). ▶ Water may be used to prevent dusting. ▶ Collect remaining material in containers with covers for disposal. ▶ Flush spill area with water.

6.4. Reference to other sections

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

SECTION 7 Handling and storage

7.1. Precautions for safe handling

Safe handling	<p>The substance accumulates peroxides which may become hazardous only if it evaporates or is distilled or otherwise treated to concentrate the peroxides. The substance may concentrate around the container opening for example. Purchases of peroxidisable chemicals should be restricted to ensure that the chemical is used completely before it can become peroxidised.</p> <ul style="list-style-type: none"> ▶ A responsible person should maintain an inventory of peroxidisable chemicals or annotate the general chemical inventory to indicate which chemicals are subject to peroxidation. An expiration date should be determined. The chemical should either be treated to remove peroxides or disposed of before this date. ▶ The person or laboratory receiving the chemical should record a receipt date on the bottle. The individual opening the container should add an opening date. ▶ Unopened containers received from the supplier should be safe to store for 18 months. ▶ Opened containers should not be stored for more than 12 months. ▶ 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.
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	<ul style="list-style-type: none"> ▶ DO NOT allow material to contact humans, exposed food or food utensils. ▶ 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. Launder contaminated clothing before re-use. ▶ 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 are maintained.
Fire and explosion protection	See section 5
Other information	<p>Consider storage under inert gas.</p> <ul style="list-style-type: none"> ▶ Store away from incompatible materials.

7.2. Conditions for safe storage, including any incompatibilities

Suitable container	<p>Generally packaging as originally supplied with the article or manufactured item is sufficient to protect against physical hazards. If repackaging is required ensure the article is intact and does not show signs of wear. As far as is practicably possible, reuse the original packaging or something providing a similar level of protection to both the article and the handler.</p>
Storage incompatibility	<p>For alkyl aromatics: The alkyl side chain of aromatic rings can undergo oxidation by several mechanisms. The most common and dominant one is the attack by oxidation at benzylic carbon as the intermediate formed is stabilised by resonance structure of the ring.</p> <ul style="list-style-type: none"> ▶ Following reaction with oxygen and under the influence of sunlight, a hydroperoxide at the alpha-position to the aromatic ring, is the primary oxidation product formed (provided a hydrogen atom is initially available at this position) - this product is often short-lived but may be stable dependent on the nature of the aromatic substitution; a secondary C-H bond is more easily attacked than a primary C-H bond whilst a tertiary C-H bond is even more susceptible to attack by oxygen ▶ Monoalkylbenzenes may subsequently form monocarboxylic acids; alkyl naphthalenes mainly produce the corresponding naphthalene carboxylic acids. ▶ Oxidation in the presence of transition metal salts not only accelerates but also selectively decomposes the hydroperoxides. ▶ Hock-rearrangement by the influence of strong acids converts the hydroperoxides to hemiacetals. Peresters formed from the hydroperoxides undergo Criegee rearrangement easily. ▶ Alkali metals accelerate the oxidation while CO₂ as co-oxidant enhances the selectivity. ▶ Microwave conditions give improved yields of the oxidation products. ▶ Photo-oxidation products may occur following reaction with hydroxyl radicals and NO_x - these may be components of photochemical smogs. <p>Oxidation of Alkylaromatics: T.S.S Rao and Shubhra Awasthi: E-Journal of Chemistry Vol 4, No. 1, pp 1-13 January 2007</p> <ul style="list-style-type: none"> ▶ Vigorous reactions, sometimes amounting to explosions, can result from the contact between aromatic rings and strong oxidising agents. ▶ Aromatics can react exothermically with bases and with diazo compounds. <p>The substance may be or contains a "metalloid"</p> <p>The following elements are considered to be metalloids; boron, silicon, germanium, arsenic, antimony, tellurium and (possibly) polonium</p> <p>The electronegativities and ionisation energies of the metalloids are between those of the metals and nonmetals, so the metalloids exhibit characteristics of both classes. The reactivity of the metalloids depends on the element with which they are reacting. For example, boron acts as a nonmetal when reacting with sodium yet as a metal when reacting with fluorine. Unlike most metals, most metalloids are amphoteric- that is they can act as both an acid and a base. For instance, arsenic forms not only salts such as arsenic halides, by the reaction with certain strong acid, but it also forms arsenites by reactions with strong bases.</p> <p>Most metalloids have a multiplicity of oxidation states or valences. For instance, tellurium has the oxidation states +2, -2, +4, and +6. Metalloids react like non-metals when they react with metals and act like metals when they react with non-metals.</p> <p>Silicas:</p> <ul style="list-style-type: none"> ▶ react with hydrofluoric acid to produce silicon tetrafluoride gas ▶ react with xenon hexafluoride to produce explosive xenon trioxide ▶ reacts exothermically with oxygen difluoride, and explosively with chlorine trifluoride (these halogenated materials are not commonplace industrial materials) and other fluorine-containing compounds ▶ may react with fluorine, chlorates ▶ are incompatible with strong oxidisers, manganese trioxide, chlorine trioxide, strong alkalis, metal oxides, concentrated orthophosphoric acid, vinyl acetate ▶ may react vigorously when heated with alkali carbonates. ▶ Segregate from alcohol, water. ▶ Avoid strong acids, bases. ▶ The unhindered oxygen atom found on cyclic ethers such as the epoxides, oxetanes, furans, dioxanes and pyrans, carries two unshared pairs of electrons - a structure which favors the formation of coordination complexes and the solvation of cations. ▶ Cyclic ethers are used as important solvents, as chemical intermediate and as monomers for ring-opening polymerization. ▶ They are unstable at room temperature due to possibility of peroxide formation; stabiliser is sometimes needed for storage and transportation.

	NOTE: Ethers lacking non-methyl hydrogen atoms adjacent to the ether link are thought to be relatively safe
Hazard categories in accordance with Regulation (EC) No 1272/2008	Not Available
Qualifying quantity (tonnes) of dangerous substances as referred to in Article 3(10) for the application of	Not Available



X — Must not be stored together

O — May be stored together with specific preventions

+ — May be stored together

Note: Depending on other risk factors, compatibility assessment based on the table above may not be relevant to storage situations, particularly where large volumes of dangerous goods are stored and handled. Reference should be made to the Safety Data Sheets for each substance or article and risks assessed accordingly.

7.3. Specific end use(s)

See section 1.2

SECTION 8 Exposure controls / personal protection

8.1. Control parameters

Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester	Dermal 4 mg/kg bw/day (Systemic, Chronic) Inhalation 28 mg/m ³ (Systemic, Chronic) Inhalation 112 mg/m ³ (Systemic, Acute) Dermal 1.4 mg/kg bw/day (Systemic, Chronic) * Inhalation 4.8 mg/m ³ (Systemic, Chronic) * Oral 1.4 mg/kg bw/day (Systemic, Chronic) * Dermal 1.4 mg/kg bw/day (Systemic, Acute) * Inhalation 4.8 mg/m ³ (Systemic, Acute) * Oral 1.4 mg/kg bw/day (Systemic, Acute) *	0 mg/L (Water (Fresh)) 0 mg/L (Water - Intermittent release) 0.21 mg/kg sediment dw (Sediment (Fresh Water)) 0.02 mg/kg sediment dw (Sediment (Marine)) 0.1 mg/kg soil dw (Soil) 31.1 mg/L (STP)
aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester	Dermal 11.9 mg/kg bw/day (Systemic, Chronic) Inhalation 84 mg/m ³ (Systemic, Chronic) Inhalation 672 mg/m ³ (Systemic, Acute) Dermal 4.2 mg/kg bw/day (Systemic, Chronic) * Inhalation 14.5 mg/m ³ (Systemic, Chronic) * Oral 4.2 mg/kg bw/day (Systemic, Chronic) * Dermal 4.2 mg/kg bw/day (Systemic, Acute) * Inhalation 14.5 mg/m ³ (Systemic, Acute) * Oral 4.2 mg/kg bw/day (Systemic, Acute) *	0 mg/L (Water (Fresh)) 0 mg/L (Water - Intermittent release) 0.21 mg/kg sediment dw (Sediment (Fresh Water)) 0.02 mg/kg sediment dw (Sediment (Marine)) 0.1 mg/kg soil dw (Soil) 31.1 mg/L (STP)
naphtha petroleum, light aromatic solvent	Inhalation 1.9 mg/m ³ (Systemic, Chronic) Inhalation 837.5 mg/m ³ (Local, Chronic) Inhalation 1 286.4 mg/m ³ (Systemic, Acute) Inhalation 1 066.67 mg/m ³ (Local, Acute) Inhalation 0.41 mg/m ³ (Systemic, Chronic) * Inhalation 178.57 mg/m ³ (Local, Chronic) * Inhalation 1 152 mg/m ³ (Systemic, Acute) * Inhalation 640 mg/m ³ (Local, Acute) *	Not Available
N-(ethoxycarbonylphenyl)-N'-methyl-N'-phenylformamidine	Dermal 1 mg/kg bw/day (Systemic, Chronic) Inhalation 0.6 mg/m ³ (Systemic, Chronic) Dermal 0.1 mg/kg bw/day (Systemic, Chronic) * Inhalation 0.1 µg/m ³ (Systemic, Chronic) * Oral 0.1 mg/kg bw/day (Systemic, Chronic) *	1.4 µg/L (Water (Fresh)) 0.14 µg/L (Water - Intermittent release) 14 µg/L (Water (Marine)) 5.26 µg/kg sediment dw (Sediment (Fresh Water)) 0.526 µg/kg sediment dw (Sediment (Marine)) 0.231 µg/kg soil dw (Soil) 10 mg/L (STP)
propylene carbonate	Dermal 20 mg/kg bw/day (Systemic, Chronic) Inhalation 70.53 mg/m ³ (Systemic, Chronic) Dermal 10 mg/cm ² (Local, Chronic)	0.9 mg/L (Water (Fresh)) 0.09 mg/L (Water - Intermittent release) 9 mg/L (Water (Marine))

Ingredient	DNELs	PNECs
	Exposure Pattern Worker	Compartment
	Inhalation 20 mg/m ³ (Local, Chronic) Dermal 10 mg/kg bw/day (Systemic, Chronic) * Inhalation 17.4 mg/m ³ (Systemic, Chronic) * Oral 10 mg/kg bw/day (Systemic, Chronic) * Inhalation 10 mg/m ³ (Local, Chronic) *	0.81 mg/kg soil dw (Soil) 7400 mg/L (STP)
silica amorphous	Inhalation 0.3 mg/m ³ (Local, Chronic) Inhalation 15 mg/m ³ (Local, Acute) Oral 3.29 mg/kg bw/day (Systemic, Chronic) *	Not Available

* Values for General Population

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
European Union Directive (EU) 2017/2398 amending Directive 2004/37/EC on the protection of workers from the risks related to exposure to carcinogens or mutagens at work	silica amorphous	Not Available	0,1 mg/m3	Not Available	Not Available	(TWA (8) Respirable fraction.)

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
naphtha petroleum, light aromatic solvent	1,200 mg/m3	6,700 mg/m3	40,000 mg/m3
propylene carbonate	34 mg/m3	370 mg/m3	2,200 mg/m3
silica amorphous	18 mg/m3	200 mg/m3	1,200 mg/m3
silica amorphous	18 mg/m3	100 mg/m3	630 mg/m3
silica amorphous	120 mg/m3	1,300 mg/m3	7,900 mg/m3
silica amorphous	45 mg/m3	500 mg/m3	3,000 mg/m3
silica amorphous	18 mg/m3	740 mg/m3	4,500 mg/m3

Ingredient	Original IDLH	Revised IDLH
aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester	Not Available	Not Available
aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester	Not Available	Not Available
naphtha petroleum, light aromatic solvent	Not Available	Not Available
N-(ethoxycarbonylphenyl)-N'-methyl-N'-phenylformamidine	Not Available	Not Available
propylene carbonate	Not Available	Not Available
silica amorphous	3,000 mg/m3	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester	D	> 0.1 to ≤ 1 ppm
aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester	D	> 0.1 to ≤ 1 ppm
N-(ethoxycarbonylphenyl)-N'-methyl-N'-phenylformamidine	E	≤ 0.1 ppm
propylene carbonate	E	≤ 0.1 ppm

Notes:

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

MATERIAL DATA

Continued...

Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen

[National Toxicology Program: U.S. Dep. of Health & Human Services 2002]

These exposure guidelines have been derived from a screening level of risk assessment and should not be construed as unequivocally safe limits. ORGS represent an 8-hour time-weighted average unless specified otherwise.

CR = Cancer Risk/10000; UF = Uncertainty factor:

TLV believed to be adequate to protect reproductive health:

LOD: Limit of detection

Toxic endpoints have also been identified as:

D = Developmental; R = Reproductive; TC = Transplacental carcinogen

Jankovic J., Drake F.: A Screening Method for Occupational Reproductive

American Industrial Hygiene Association Journal 57: 641-649 (1996)

NOTE E: Substances with specific effects on human health that are classified as carcinogenic, mutagenic and/ or toxic for reproduction in categories 1 or 2 are ascribed Note E if they are classified as very toxic (T+), toxic (T) or harmful (Xn). For these substances the risk phrases R20 ,R21, R22, R23, R24,R25, R26, R27, R28, R39, R68, R48 and R65 and all combinations of these risk phrases shall be preceded by the word "Also".

R45-23: May cause cancer. Also toxic by inhalation

This note applies only to certain complex oil-derived substances in Annex VI.

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

For amorphous crystalline silica (precipitated silicic acid):

Amorphous crystalline silica shows little potential for producing adverse effects on the lung and exposure standards should reflect a particulate of low intrinsic toxicity. Mixtures of amorphous silicas/ diatomaceous earth and crystalline silica should be monitored as if they comprise only the crystalline forms.

The dusts from precipitated silica and silica gel produce little adverse effect on pulmonary functions and are not known to produce significant disease or toxic effect.

IARC has classified silica, amorphous as Group 3: **NOT** classifiable as to its carcinogenicity to humans.

Evidence of carcinogenicity may be inadequate or limited in animal testing.

For trimethyl benzene as mixed isomers (of unstated proportions)

Odour Threshold Value: 2.4 ppm (detection)

Use care in interpreting effects as a single isomer or other isomer mix. Trimethylbenzene is an eye, nose and respiratory irritant. High concentrations cause central nervous system depression. Exposed workers show CNS changes, asthmatic bronchitis and blood dyscrasias at 60 ppm. The TLV-TWA is thought to be protective against the significant risk of CNS excitation, asthmatic bronchitis and blood dyscrasias associated with exposures above the limit.

Odour Safety Factor (OSF)

OSF=10 (1,2,4-TRIMETHYLBENZENE)

Exposed individuals are **NOT** reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odour Safety Factor (OSF) is determined to fall into either Class C, D or E.

The Odour Safety Factor (OSF) is defined as:

OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm

Classification into classes follows:

ClassOSF Description

A 550 Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by working activities

B 26-550As "A" for 50-90% of persons being distracted

C 1-26 As "A" for less than 50% of persons being distracted

D 0.18-1 10-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached

E <0.18 As "D" for less than 10% of persons aware of being tested

NOTE P: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.01% w/w benzene (EINECS No 200-753-7).

Note E shall also apply when the substance is classified as a carcinogen. This note applies only to certain complex oil-derived substances in Annex VI.

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

8.2. Exposure controls

8.2.1. 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.

	<ul style="list-style-type: none"> ▶ 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. <p>Articles or manufactured items, in their original condition, generally don't require engineering controls during handling or in normal use.</p> <p>Exceptions may arise following extensive use and subsequent wear, during recycling or disposal operations where substances, found in the article, may be released to the environment.</p>
8.2.2. Individual protection measures, such as personal protective equipment	
Eye and face protection	<p>No special equipment required due to the physical form of the product.</p> <ul style="list-style-type: none"> ▶ 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	<p>See Hand protection below</p>
Hands/feet protection	<p>Wear general protective gloves, eg. light weight rubber gloves.</p> <p>NOTE:</p> <ul style="list-style-type: none"> ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.
Body protection	<p>See Other protection below</p>
Other protection	<ul style="list-style-type: none"> ▶ 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.

Respiratory protection

Respiratory protection not normally required due to the physical form of the product.

8.2.3. Environmental exposure controls

See section 12

SECTION 9 Physical and chemical properties

9.1. Information on basic physical and chemical properties

Appearance	Moisture sensitive. Family of products which vary in their physical properties as a result of variations in production. Data presented here is for typical family member.		
Physical state	Manufactured	Relative density (Water = 1)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Available
Flash point (°C)	Not Available	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Available	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Applicable
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
Nanoform Solubility	Not Available	Nanoform Particle Characteristics	Not Available
Particle Size	Not Available		

9.2. Other information

Not Available

SECTION 10 Stability and reactivity

10.1.Reactivity	See section 7.2
10.2. Chemical stability	Product is considered stable and hazardous polymerisation will not occur.
10.3. Possibility of hazardous reactions	See section 7.2
10.4. Conditions to avoid	See section 7.2
10.5. Incompatible materials	See section 7.2
10.6. Hazardous decomposition products	See section 5.3

SECTION 11 Toxicological information

11.1. Information on hazard classes as defined in Regulation (EC) No 1272/2008 Information on toxicological effects

Inhaled	<p>The material is not thought to produce adverse health effects or irritation of the respiratory tract (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.</p> <p>A significant number of individuals exposed to mixed trimethylbenzenes complained of nervousness, tension, anxiety and asthmatic bronchitis. Peripheral blood showed a tendency to hypochromic anaemia and a deviation from normal in coagulability of the blood. Hydrocarbon concentrations ranged from 10 to 60 ppm. Contamination of the mixture with benzene may have been responsible for the blood dyscrasias.</p> <p>High concentrations of mesitylene vapour (5000 to 9000 ppm) caused central nervous system depression in mice. Similar exposures of pseudocumene also produced evidence of CNS involvement.</p> <p>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.</p> <p>The acute toxicity of inhaled alkylbenzene is best described by central nervous system depression. These compounds may also act as general anaesthetics. Whole body symptoms of poisoning include light-headedness, nervousness, apprehension, a feeling of well-being, confusion, dizziness, drowsiness, ringing in the ears, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness, depression of breathing, and arrest. Heart stoppage may result from cardiovascular collapse. A slow heart rate and low blood pressure may also occur.</p> <p>Alkylbenzenes are not generally toxic except at high levels of exposure. Their breakdown products have low toxicity and are easily eliminated from the body.</p>
Ingestion	<p>The material has NOT been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.</p> <p>High molecular weight material; on single acute exposure would be expected to pass through gastrointestinal tract with little change / absorption. Occasionally accumulation of the solid material within the alimentary tract may result in formation of a bezoar (concretion), producing discomfort.</p>
Skin Contact	<p>Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions.</p> <p>Limited evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>
Eye	<p>Although the material is not thought to be an irritant (as classified by EC Directives), direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn).</p>
Chronic	<p>Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.</p> <p>Strong evidence exists that the substance may cause irreversible but non-lethal mutagenic effects following a single exposure. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.</p> <p>Substances that can cause occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers</p> <p>Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.</p> <p>Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.</p> <p>On the basis of epidemiological data, the material is regarded as carcinogenic to humans. There is sufficient data to establish a causal association between human exposure to the material and the development of cancer.</p>

Cyclic ethers, including tetrahydrofuran, furan and 1,4-dioxane, produce neoplasms and carcinomas in experimental animals, typically of the liver; other target organs include the adrenal gland, nasal cavity and gall-bladder. 1,4-Dioxane was a promoter in a two-stage skin carcinogenic study in mice. Results of studies with cyclic ethers indicate that carcinogenicity is often species and sex dependent. Furan has been used to induce apoptosis (programmed cell death). Oxetanes are under investigation.

The material contains a substantial proportion of a polymer considered to be of low concern (PLC). The trend towards production of lower molecular weight polymers (thus reducing the required level of solvent use and creating a more "environmentally-friendly" material) has brought with it the need to define PLCs as those having molecular weights of between 1000 and 10000 and containing less than 10% of the molecules with molecular weight below 500 and less than 25% of the molecules with a molecular weight below 1000. These may contain unlimited low concern functional groups or moderate concern reactive functional groups with a combined functional group equivalent weight (FGEW, a concept developed by the US EPA describing whether the reactive functional group is sufficiently diluted by polymeric material) of a 1000 or more (provided no high concern groups are present) or high concern reactive functional groups with a FGEW of 5000 or more (FGEW includes moderate concern groups if present).

having molecular weights exceeding 10000 (without restriction on reactive groups).

inhalation of polymers with molecular weights > 70,000 Da has been linked with irreversible lung damage due to lung overloading and impaired clearance of particles from the lung, particularly following repeated exposure. If the polymer is inhaled at low levels and/or infrequently, it is assumed that it will be cleared from the lungs.

Reactive functional groups are in turn classified as being of low, moderate or high concern Classification of the polymer as a PLC, in accordance with established criteria, does not mean that hazards will not be associated with the polymer (during its import, manufacture, use, storage, handling or disposal). The polymer may, for example, contain a large number of particles in the respirable range, a hazard which may need to be assessed in the health and safety risk assessment. Similarly a polymer with low concern reactive may be released into the environment in large quantities and produce an environmental hazard.

Whilst it is generally accepted that polymers with a molecular weight exceeding 1000 are unlikely to pass through biological membranes, oligomers with lower molecular weight and specifically, those with a molecular weight below 500, may. Estimations based on a "highly" dispersed polymer population (polydispersity = 10) suggests that the molecular weight of the polymer carrying a reactive group of high concern must be 5000 to be considered a PLC; similarly a polymer of approximate molecular weight 1000 could contain no more than one reactive group of moderate concern (for two moderate concern groups, the molecular weight would be about 2500).

The synthetic, amorphous silicas are believed to represent a very greatly reduced silicosis hazard compared to crystalline silicas and are considered to be nuisance dusts.

When heated to high temperature and a long time, amorphous silica can produce crystalline silica on cooling. Inhalation of dusts containing crystalline silicas may lead to silicosis, a disabling pulmonary fibrosis that may take years to develop. Discrepancies between various studies showing that fibrosis associated with chronic exposure to amorphous silica and those that do not may be explained by assuming that diatomaceous earth (a non-synthetic silica commonly used in industry) is either weakly fibrogenic or nonfibrogenic and that fibrosis is due to contamination by crystalline silica content

Repeated exposure to synthetic amorphous silicas may produce skin dryness and cracking.

Available data confirm the absence of significant toxicity by oral and dermal routes of exposure.

Numerous repeated-dose, subchronic and chronic inhalation toxicity studies have been conducted in a number of species, at airborne concentrations ranging from 0.5 mg/m³ to 150 mg/m³. Lowest-observed adverse effect levels (LOAELs) were typically in the range of 1 to 50 mg/m³. When available, the no-observed adverse effect levels (NOAELs) were between 0.5 and 10 mg/m³. Differences in values may be due to particle size, and therefore the number of particles administered per unit dose.

Generally, as particle size diminishes so does the NOAEL/ LOAEL. Exposure produced transient increases in lung inflammation, markers of cell injury and lung collagen content. There was no evidence of interstitial pulmonary fibrosis.

Diamond Coat	TOXICITY	IRRITATION
	Not Available	Not Available
aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye : Mild
	Inhalation(Rat) LC50: >4.224 mg/L4h ^[1]	Skin : Moderate
	Oral (Rat) LD50: >2000 mg/kg ^[1]	
aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye : Mild
	Inhalation(Rat) LC50: >4.224 mg/L4h ^[1]	Skin : Moderate
	Oral (Rat) LD50: >2000 mg/kg ^[1]	
naphtha petroleum, light aromatic solvent	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1900 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation(Rat) LC50: >4.42 mg/L4h ^[1]	Skin: adverse effect observed (irritating) ^[1]
	Oral (Rat) LD50: >4500 mg/kg ^[1]	
N-(ethoxycarbonylphenyl)-N'-methyl-N'-phenylformamidine	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >1000 mg/kg ^[2]	Skin: no adverse effect observed (not irritating) ^[1]

propylene carbonate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >=2000 mg/kg ^[1]	Eye (rabbit): 60 mg - moderate
	Oral (Rat) LD50: >5000 mg/kg ^[1]	Eye: adverse effect observed (irritating) ^[1]
		Skin (human): 100 mg/3d-I moderate
		Skin (rabbit): 500 mg moderate
		Skin: no adverse effect observed (not irritating) ^[1]
silica amorphous	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): non-irritating ** [Grace]
	Inhalation(Rat) LC50: >0.09<0.84 mg/4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >1000 mg/kg ^[1]	Skin (rabbit): non-irritating *
		Skin: no adverse effect observed (not irritating) ^[1]
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	

Diamond Coat	<p>Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.</p> <p>Data demonstrate that during inhalation exposure,aromatic hydrocarbons undergo substantial partitioning into adipose tissues. Following cessation of exposure, the level of aromatic hydrocarbons in body fats rapidly declines. Thus, the aromatic hydrocarbons are unlikely to bioaccumulate in the body. Selective partitioning of the aromatic hydrocarbons into the non-adipose tissues is unlikely. No data is available regarding distribution following dermal absorption. However, distribution following this route of exposure is likely to resemble the pattern occurring with inhalation exposure.</p> <p>Aromatics hydrocarbons may undergo several different Phase I dealkylation, hydroxylation and oxidation reactions which may or may not be followed by Phase II conjugation to glycine, sulfation or glucuronidation. However, the major predominant biotransformation pathway is typical of that of the alkylbenzenes and consists of: (1) oxidation of one of the alkyl groups to an alcohol moiety; (2) oxidation of the hydroxyl group to a carboxylic acid; (3) the carboxylic acid is then conjugated with glycine to form a hippuric acid. The minor metabolites can be expected to consist of a complex mixture of isomeric triphenols, the sulfate and glucuronide conjugates of dimethylbenzyl alcohols, dimethylbenzoic acids and dimethylhippuric acids. Consistent with the low propensity for bioaccumulation of aromatic hydrocarbons, these substances are likely to be significant inducers of their own metabolism.</p> <p>The predominant route of excretion of aromatic hydrocarbons following inhalation exposure involves either exhalation of the unmetabolized parent compound, or urinary excretion of its metabolites. When oral administration occurs, there is little exhalation of unmetabolized these hydrocarbons, presumably due to the first pass effect in the liver. Under these circumstances, urinary excretion of metabolites is the dominant route of excretion.</p>
ASPARTIC ACID, N,N'-(METHYLENEDICYCLOHEXANEDIYL)BIS-,ESTER	<p>for similar substance CAS 136210-10-32-7:</p> <p>Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure.</p>
NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT	<p>Inhalation (rat) TClO: 1320 ppm/6h/90D-I * [Devoe] For Low Boiling Point Naphthas (LBPNS): Acute toxicity: LBPNS generally have low acute toxicity by the oral (median lethal dose [LD50] in rats > 2000 mg/kg-bw), inhalation (LD50 in rats > 5000 mg/m3) and dermal (LD50 in rabbits > 2000 mg/kg-bw) routes of exposure Most LBPNS are mild to moderate eye and skin irritants in rabbits, with the exception of heavy catalytic cracked and heavy catalytic reformed naphthas, which have higher primary skin irritation indices. Sensitisation: LBPNS do not appear to be skin sensitizers, but a poor response in the positive control was also noted in these studies Repeat dose toxicity: The lowest-observed-adverse-effect concentration (LOAEC) and lowest-observed-adverse-effect level (LOAEL) values identified following short-term (2-89 days) and subchronic (greater than 90 days) exposure to the LBPNS substances. These values were determined for a variety of endpoints after considering the toxicity data for all LBPNS in the group. Most of the studies were carried out by the inhalation route of exposure. Renal effects, including increased kidney weight, renal lesions (renal tubule dilation, necrosis) and hyaline droplet formation, observed in male rats exposed orally or by inhalation to most LBPNS, were considered species- and sex-specific These effects were determined to be due to a mechanism of action not relevant to humans -specifically, the interaction between hydrocarbon metabolites and alpha-2-microglobulin, an enzyme not produced in substantial amounts in female rats, mice and other species, including humans. The resulting nephrotoxicity and subsequent carcinogenesis in male rats were therefore not considered in deriving LOAEC/LOAEL values. Only a limited number of studies of short-term and subchronic duration were identified for site-restricted LBPNS. The lowest LOAEC identified in these studies, via the inhalation</p>

route, is 5475 mg/m³, based on a concentration-related increase in liver weight in both male and female rats following a 13-week exposure to light catalytic cracked naphtha. Shorter exposures of rats to this test substance resulted in nasal irritation at 9041 mg/m³. No systemic toxicity was reported following dermal exposure to light catalytic cracked naphtha, but skin irritation and accompanying histopathological changes were increased, in a dose-dependent manner, at doses as low as 30 mg/kg-bw per day when applied 5 days per week for 90 days in rats. No non-cancer chronic toxicity studies (= 1 year) were identified for site-restricted LBPNS and very few non-cancer chronic toxicity studies were identified for other LBPNS. An LOAEC of 200 mg/m³ was noted in a chronic inhalation study that exposed mice and rats to unleaded gasoline (containing 2% benzene). This inhalation LOAEC was based on ocular discharge and ocular irritation in rats. At the higher concentration of 6170 mg/m³, increased kidney weight was observed in male and female rats (increased kidney weight was also observed in males only at 870 mg/m³). Furthermore, decreased body weight in male and female mice was also observed at 6170 mg/m³. A LOAEL of 714 mg/kg-bw was identified for dermal exposure based on local skin effects (inflammatory and degenerative skin changes) in mice following application of naphtha for 105 weeks. No systemic toxicity was reported.

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

Carcinogenicity: Although a number of epidemiological studies have reported increases in the incidence of a variety of cancers, the majority of these studies are considered to contain incomplete or inadequate information. Limited data, however, are available for skin cancer and leukemia incidence, as well as mortality among petroleum refinery workers. It was concluded that there is limited evidence supporting the view that working in petroleum refineries entails a carcinogenic risk (Group 2A carcinogen). IARC (1989a) also classified gasoline as a Group 2B carcinogen; it considered the evidence for carcinogenicity in humans from gasoline to be inadequate and noted that published epidemiological studies had several limitations, including a lack of exposure data and the fact that it was not possible to separate the effects of combustion products from those of gasoline itself. Similar conclusions were drawn from other reviews of epidemiological studies for gasoline (US EPA 1987a, 1987b). Thus, the evidence gathered from these epidemiological studies is considered to be inadequate to conclude on the effects of human exposure to LBPNS substances. No inhalation studies assessing the carcinogenicity of the site-restricted LBPNS were identified. Only unleaded gasoline has been examined for its carcinogenic potential, in several inhalation studies. In one study, rats and mice were exposed to 0, 200, 870 or 6170 mg/m³ of a 2% benzene formulation of the test substance, via inhalation, for approximately 2 years. A statistically significant increase in hepatocellular adenomas and carcinomas, as well as a non-statistical increase in renal tumours, were observed at the highest dose in female mice. A dose-dependent increase in the incidence of primary renal neoplasms was also detected in male rats, but this was not considered to be relevant to humans, as discussed previously. Carcinogenicity was also assessed for unleaded gasoline, via inhalation, as part of initiation/promotion studies. In these studies, unleaded gasoline did not appear to initiate tumour formation, but did show renal cell and hepatic tumour promotion ability, when rats and mice were exposed, via inhalation, for durations ranging from 13 weeks to approximately 1 year using an initiation/promotion protocol. However, further examination of data relevant to the composition of unleaded gasoline demonstrated that this is a highly-regulated substance; it is expected to contain a lower percentage of benzene and has a discrete component profile when compared to other substances in the LBPNS group. Both the European Commission and the International Agency for Research on Cancer (IARC) have classified LBPNS substances as carcinogenic. All of these substances were classified by the European Commission (2008) as Category 2 (R45: may cause cancer) (benzene content = 0.1% by weight). IARC has classified gasoline, an LBPNS, as a Group 2B carcinogen (possibly carcinogenic to humans) and "occupational exposures in petroleum refining" as Group 2A carcinogens (probably carcinogenic to humans). Several studies were conducted on experimental animals to investigate the dermal carcinogenicity

of LBPNs. The majority of these studies were conducted through exposure of mice to doses ranging from 694-1351 mg/kg-bw, for durations ranging from 1 year to the animals lifetime or until a tumour persisted for 2 weeks. Given the route of exposure, the studies specifically examined the formation of skin tumours. Results for carcinogenicity via dermal exposure are mixed. Both malignant and benign skin tumours were induced with heavy catalytic cracked naphtha, light catalytic cracked naphtha, light straight-run naphtha and naphtha. Significant increases in squamous cell carcinomas were also observed when mice were dermally treated with Stoddard solvent, but the latter was administered as a mixture (90% test substance), and the details of the study were not available. In contrast, insignificant increases in tumour formation or no tumours were observed when light alkylate naphtha, heavy catalytic reformed naphtha, sweetened naphtha, light catalytically cracked naphtha or unleaded gasoline was dermally applied to mice. Negative results for skin tumours were also observed in male mice dermally exposed to sweetened naphtha using an initiation/promotion protocol. Reproductive/ Developmental toxicity: No reproductive or developmental toxicity was observed for the majority of LBPN substances evaluated. Most of these studies were carried out by inhalation exposure in rodents. NOAEC values for reproductive toxicity following inhalation exposure ranged from 1701 mg/m³ (CAS RN 8052-41-3) to 27 687 mg/m³ (CAS RN 64741-63-5) for the LBPNs group evaluated, and from 7690 mg/m³ to 27 059 mg/m³ for the site-restricted light catalytic cracked and full-range catalytic reformed naphthas. However, a decreased number of pups per litter and higher frequency of post-implantation loss were observed following inhalation exposure of female rats to hydrotreated heavy naphtha (CAS RN 64742-48-9) at a concentration of 4679 mg/m³, 6 hours per day, from gestational days 7-20. For dermal exposures, NOAEL values of 714 mg/kg-bw (CAS RN 8030-30-6) and 1000 mg/kg-bw per day (CAS RN 68513-02-0) were noted. For oral exposures, no adverse effects on reproductive parameters were reported when rats were given site-restricted light catalytic cracked naphtha at 2000 mg/kg on gestational day 13. For most LBPNs, no treatment-related developmental effects were observed by the different routes of exposure. However, developmental toxicity was observed for a few naphthas. Decreased foetal body weight and an increased incidence of ossification variations were observed when rat dams were exposed to light aromatized solvent naphtha, by gavage, at 1250 mg/kg-bw per day. In addition, pregnant rats exposed by inhalation to hydrotreated heavy naphtha at 4679 mg/m³ delivered pups with higher birth weights. Cognitive and memory impairments were also observed in the offspring.

Low Boiling Point Naphthas [Site-Restricted]

Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length, with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent than iso- or cyclo-paraffins. The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption, is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.

For C9 aromatics (typically trimethylbenzenes - TMBs)

Acute Toxicity

Acute toxicity studies (oral, dermal and inhalation routes of exposure) have been conducted in rats using various solvent products containing predominantly mixed C9 aromatic hydrocarbons (CAS RN 64742-95-6). Inhalation LC50s range from 6,000 to 10,000 mg/m³ for C9 aromatic naphtha and 18,000 to 24,000 mg/m³ for 1,2,4 and 1,3,5-TMB, respectively. A rat oral LD50 reported for 1,2,4-TMB is 5 grams/kg bw and a rat dermal LD50 for the C9 aromatic naphtha is >4 ml/kg bw. These data indicate that C9 aromatic solvents show that LD50/LC50 values are greater than limit doses for acute toxicity studies established under OECD test guidelines.

Irritation and Sensitization

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

Repeated Dose Toxicity

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

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

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

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

Mutagenicity

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

Reproductive and Developmental Toxicity

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

Systemic Effects on Parental Generations:

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

Reproductive Toxicity-Effects on Parental Generations: There were no pathological changes noted in the reproductive organs of any animal of the F0, F1, or F2 generation. No effects were reported on sperm morphology, gestational period, number of implantation sites, or post-implantation loss in any generation. Also, there were no statistically or biologically significant differences in any of the reproductive parameters, including: number of mated females, copulatory index, copulatory interval, number of females delivering a litter, number of females delivering a live litter, or male fertility in the

	<p>F0 or in the F2 generation. Male fertility was statistically significantly reduced at 1480 ppm in the F1 rats. However, male fertility was not affected in the F0 or in the F2 generations; therefore, the biological significance of this change is unknown and may or may not be attributed to the test substance. No reproductive effects were observed in the F0 or F1 dams exposed to 1480 ppm (7265 mg/m³). Due to excessive mortality at the highest concentration (1480 ppm, only six dams available) in the F2 generation, a complete evaluation is precluded. However, no clear signs of reproductive toxicity were observed in the F2 generation. Therefore, the reproductive NOAEC is considered 495 ppm (2430 mg/m³), which excludes analysis of the highest concentration due to excessive mortality.</p> <p>Developmental Toxicity - Effects on Pups: Because of significant maternal toxicity (including mortality) in dams in all generations at the highest concentration (1480 ppm), effects in offspring at 1480 ppm are not reported here. No significant effects were observed in the F1 and F2 generation offspring at 103 or 495 ppm. However, in F3 offspring, body weights and body weight gain were reduced by ~ 10-11% compared with controls at 495 ppm for approximately a week (PND 14 through 21). Maternal body weight was also depressed by ~ 12% throughout the gestational period compared with controls. The overall developmental LOAEC from this study is 495 ppm (2430 mg/m³) based on the body weights reductions observed in the F3 offspring.</p> <p>Conclusion: No effects on reproductive parameters were observed at any exposure concentration, although a confident assessment of the group exposed at the highest concentration was not possible. A potential developmental effect (reduction in mean pup weight and weight gain) was observed at a concentration that was also associated with maternal toxicity.</p> <p>For petroleum: This product contains benzene, which can cause acute myeloid leukaemia, and n-hexane, which can be metabolized to compounds which are toxic to the nervous system. This product contains toluene, and animal studies suggest high concentrations of toluene lead to hearing loss. This product contains ethyl benzene and naphthalene, from which animal testing shows evidence of tumour formation.</p> <p>Cancer-causing potential: Animal testing shows inhaling petroleum causes tumours of the liver and kidney; these are however not considered to be relevant in humans.</p> <p>Mutation-causing potential: Most studies involving gasoline have returned negative results regarding the potential to cause mutations, including all recent studies in living human subjects (such as in petrol service station attendants).</p> <p>Reproductive toxicity: Animal studies show that high concentrations of toluene (>0.1%) can cause developmental effects such as lower birth weight and developmental toxicity to the nervous system of the foetus. Other studies show no adverse effects on the foetus.</p> <p>Human effects: Prolonged or repeated contact may cause defatting of the skin which can lead to skin inflammation and may make the skin more susceptible to irritation and penetration by other materials.</p> <p>Animal testing shows that exposure to gasoline over a lifetime can cause kidney cancer, but the relevance in humans is questionable.</p>
<p>N-(ETHOXYCARBONYLPHENYL)-N'-METHYL-N'-PHENYLFORMAMIDINE</p>	<p>Ames Test negative * * Nymco SDS Nymassorb FA-1</p> <p>Human exposure to formamidines has been largely limited to agricultural and production workers, as well as intentional ingestion in suicide attempts. Limited data from human oral exposures indicates that effects include lethargy, vomiting, muscle weakness, headaches, decreased monoamine oxidase (MAO) activity and blurred vision. General side-effects of formamidines in mammals are possible alterations in the animals ability to maintain homeostasis for at least 24 hours after exposure. A symptom often observed with formamidine treated mammals is a reversible sedative effect.</p> <p>Formamidine pesticides may exert their effects on the central nervous system by interacting directly with adrenergic receptors, particularly the alpha-2 subtype This interaction appears to mediate several of the observed effects of formamidines, such as changes in heart rate, pupil diameter, visual evoked potential and hormonal secretion.</p> <p>Formamidines inhibit the synthesis of prostaglandin E2 from arachidonic acid by bovine seminal vesicle microsomes.</p>
<p>PROPYLENE CARBONATE</p>	<p>WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.</p> <p>The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterized by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <p>for propylene carbonate:</p> <p>Numerous adequate and reliable acute toxicity tests are available on propylene carbonate. Oral and dermal tests meet OECD and EPA test guidelines. Propylene carbonate is practically nontoxic following acute exposures; the oral LD50 is >.5000 mg/kg and the dermal LD50 is >3000 mg/kg. No further testing is recommended.</p> <p>Subchronic studies (13- 14 weeks) of propylene carbonate by inhalation (aerosol) and oral (gavage) routes were conducted in rats according to current guidelines. The oral study indicated low systemic toxicity from propylene carbonate (NOAEL = 5000 mg/kg/day). In the inhalation study, no systemic toxicity was seen at concentrations up to 1000 mg/m³; however, there was periorbital irritation and swelling in a few males at 500 and 1000 mg/m³. A dermal carcinogenicity study in mice did not</p>

	<p>indicate tumorigenic potential or systemic toxicity from 2 years of exposure to propylene carbonate. No further testing is recommended.</p> <p>There is a negative Ames in vitro mutagenicity assay of propylene carbonate. A single intraperitoneal injection of 1666 mg/kg propylene carbonate did not induce an increase in micronuclei when examined after 30,48 and 72 hours. The mutagenicity battery is satisfactorily filled; no further mutagenicity testing is recommended.</p> <p>Gavage administration of propylene carbonate to pregnant rats days 6-15 of gestation resulted in systemic toxicity at doses of 3000 and 5000 mg/kg/day, including mortality (not seen in 13 week study of non-pregnant rats). The NOAEL for maternal toxicity was 1000 mg/kg/day. This indicates that pregnant rats are more susceptible to propylene carbonate than are non-pregnant rats. There were no significant differences in live litter size, average fetal weight, percentage of males, or malformed fetuses.</p> <p>No studies of the effect of propylene carbonate on reproduction are available. However, no adverse effects on testis, ovaries, or accessory sex organs were noted in rats following oral or inhalation of propylene carbonate for 13 weeks. Therefore, reproductive effects from propylene carbonate are unlikely</p>
<p>SILICA AMORPHOUS</p>	<p>Reports indicate high/prolonged exposures to amorphous silicas induced lung fibrosis in experimental animals; in some experiments these effects were reversible. [PATTYS]</p> <p>The substance is classified by IARC as Group 3: NOT classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.</p>
<p>Diamond Coat & ASPARTIC ACID, N,N'-(METHYLENEDICYCLOHEXANEDIYL)BIS-,ESTER & N-(ETHOXYCARBONYLPHENYL)-N'-METHYL-N'-PHENYLFORMAMIDINE</p>	<p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p>
<p>Diamond Coat & SILICA AMORPHOUS</p>	<p>For silica amorphous:</p> <p>Derived No Adverse Effects Level (NOAEL) in the range of 1000 mg/kg/d.</p> <p>In humans, synthetic amorphous silica (SAS) is essentially non-toxic by mouth, skin or eyes, and by inhalation. Epidemiology studies show little evidence of adverse health effects due to SAS. Repeated exposure (without personal protection) may cause mechanical irritation of the eye and drying/cracking of the skin.</p> <p>When experimental animals inhale synthetic amorphous silica (SAS) dust, it dissolves in the lung fluid and is rapidly eliminated. If swallowed, the vast majority of SAS is excreted in the faeces and there is little accumulation in the body. Following absorption across the gut, SAS is eliminated via urine without modification in animals and humans. SAS is not expected to be broken down (metabolised) in mammals.</p> <p>After ingestion, there is limited accumulation of SAS in body tissues and rapid elimination occurs. Intestinal absorption has not been calculated, but appears to be insignificant in animals and humans. SASs injected subcutaneously are subjected to rapid dissolution and removal. There is no indication of metabolism of SAS in animals or humans based on chemical structure and available data. In contrast to crystalline silica, SAS is soluble in physiological media and the soluble chemical species that are formed are eliminated via the urinary tract without modification.</p> <p>Both the mammalian and environmental toxicology of SASs are significantly influenced by the physical and chemical properties, particularly those of solubility and particle size. SAS has no acute intrinsic toxicity by inhalation. Adverse effects, including suffocation, that have been reported were caused by the presence of high numbers of respirable particles generated to meet the required test atmosphere. These results are not representative of exposure to commercial SASs and should not be used for human risk assessment. Though repeated exposure of the skin may cause dryness and cracking, SAS is not a skin or eye irritant, and it is not a sensitiser.</p> <p>Repeated-dose and chronic toxicity studies confirm the absence of toxicity when SAS is swallowed or upon skin contact.</p> <p>Long-term inhalation of SAS caused some adverse effects in animals (increases in lung inflammation, cell injury and lung collagen content), all of which subsided after exposure. Numerous repeated-dose, subchronic and chronic inhalation toxicity studies have been conducted with SAS in a number of species, at airborne concentrations ranging from 0.5 mg/m³ to 150 mg/m³. Lowest-observed adverse effect levels (LOAELs) were typically in the range of 1 to 50 mg/m³. When available, the no-observed adverse effect levels (NOAELs) were between 0.5 and 10 mg/m³. The difference in values may be explained by different particle size, and therefore the number of particles administered per unit dose. In general, as particle size decreases so does the NOAEL/LOAEL.</p> <p>Neither inhalation nor oral administration caused neoplasms (tumours). SAS is not mutagenic in vitro. No genotoxicity was detected in in vivo assays. SAS does not impair development of the fetus. Fertility was not specifically studied, but the reproductive organs in long-term studies were not affected.</p>

For Synthetic Amorphous Silica (SAS)
 Repeated dose toxicity
 Oral (rat), 2 weeks to 6 months, no significant treatment-related adverse effects at doses of up to 8% silica in the diet.
 Inhalation (rat), 13 weeks, Lowest Observed Effect Level (LOEL) =1.3 mg/m3 based on mild reversible effects in the lungs. Inhalation (rat), 90 days, LOEL = 1 mg/m3 based on reversible effects in the lungs and effects in the nasal cavity.
 For silane treated synthetic amorphous silica:
 Repeated dose toxicity: oral (rat), 28-d, diet, no significant treatment-related adverse effects at the doses tested.
 There is no evidence of cancer or other long-term respiratory health effects (for example, silicosis) in workers employed in the manufacture of SAS. Respiratory symptoms in SAS workers have been shown to correlate with smoking but not with SAS exposure, while serial pulmonary function values and chest radiographs are not adversely affected by long-term exposure to SAS.

Diamond Coat & NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT

For trimethylbenzenes:
 Absorption of 1,2,4-trimethylbenzene occurs after oral, inhalation, or dermal exposure.
 Occupationally, inhalation and dermal exposures are the most important routes of absorption although systemic intoxication from dermal absorption is not likely to occur due to the dermal irritation caused by the chemical prompting quick removal. Following oral administration of the chemical to rats, 62.6% of the dose was recovered as urinary metabolites indicating substantial absorption . 1,2,4-Trimethylbenzene is lipophilic and may accumulate in fat and fatty tissues. In the blood stream, approximately 85% of the chemical is bound to red blood cells Metabolism occurs by side-chain oxidation to form alcohols and carboxylic acids which are then conjugated with glucuronic acid, glycine, or sulfates for urinary excretion . After a single oral dose to rats of 1200 mg/kg, urinary metabolites consisted of approximately 43.2% glycine, 6.6% glucuronic, and 12.9% sulfuric acid conjugates . The two principle metabolites excreted by rabbits after oral administration of 438 mg/kg/day for 5 days were 2,4-dimethylbenzoic acid and 3,4-dimethylhippuric acid . The major routes of excretion of 1,2,4-trimethyl- benzene are exhalation of parent compound and elimination of urinary metabolites. Half-times for urinary metabolites were reported as 9.5 hours for glycine, 22.9 hours for glucuronide, and 37.6 hours for sulfuric acid conjugates.
Acute Toxicity Direct contact with liquid 1,2,4-trimethylbenzene is irritating to the skin and breathing the vapor is irritating to the respiratory tract causing pneumonitis. Breathing high concentrations of the chemical vapor causes headache, fatigue, and drowsiness. In humans liquid 1,2,4-trimethylbenzene is irritating to the skin and inhalation of vapor causes chemical pneumonitis . High concentrations of vapor (5000-9000 ppm) cause headache, fatigue, and drowsiness . The concentration of 5000 ppm is roughly equivalent to a total of 221 mg/kg assuming a 30 minute exposure period (see end note 1). 2. Animals - Mice exposed to 8130-9140 ppm 1,2,4-trimethylbenzene (no duration given) had loss of righting response and loss of reflexes Direct dermal contact with the chemical (no species given) causes vasodilation, erythema, and irritation (U.S. EPA). Seven of 10 rats died after an oral dose of 2.5 mL of a mixture of trimethylbenzenes in olive oil (average dose approximately 4.4 g/kg) . Rats and mice were exposed by inhalation to a coal tar distillate containing about 70% 1,3,5- and 1,2,4-trimethylbenzene; no pathological changes were noted in either species after exposure to 1800-2000 ppm for up to 48 continuous hours, or in rats after 14 exposures of 8 hours each at the same exposure levels . No effects were reported for rats exposed to a mixture of trimethyl- benzenes at 1700 ppm for 10 to 21 days
Neurotoxicity 1,2,4-Trimethylbenzene depresses the central nervous system. Exposure to solvent mixtures containing the chemical causes headache, fatigue, nervousness, and drowsiness. Occupationally, workers exposed to a solvent containing 50% 1,2,4-trimethylbenzene had nervousness, headaches, drowsiness, and vertigo (U.S. EPA). Headache, fatigue, and drowsiness were reported for workers exposed (no dose given) to paint thinner containing 80% 1,2,4- and 1,3,5-trimethylbenzenes
 Results of the developmental toxicity study indicate that the C9 fraction caused adverse neurological effects at the highest dose (1500 ppm) tested.
Subchronic/Chronic Toxicity Long-term exposure to solvents containing 1,2,4-trimethylbenzene may cause nervousness, tension, and bronchitis. Painters that worked for several years with a solvent containing 50% 1,2,4- and 30% 1,3,5-trimethylbenzene showed nervousness, tension and anxiety, asthmatic bronchitis, anemia, and alterations in blood clotting; haematological effects may have been due to trace amounts of benzene
 Rats given 1,2,4-trimethylbenzene orally at doses of 0.5 or 2.0 g/kg/day, 5 days/week for 4 weeks. All rats exposed to the high dose died and 1 rat in the low dose died (no times given); no other effects were reported. Rats exposed by inhalation to 1700 ppm of a trimethylbenzene isomeric mixture for 4 months had decreased weight gain, lymphopenia and neutrophilia .
Genotoxicity: Results of mutagenicity testing, indicate that the C9 fraction does not induce gene mutations in prokaryotes (Salmonella typhimurium/mammalian microsome assay); or in mammalian cells in culture (in Chinese hamster ovary cells with and without activation). The C9 fraction does not does not induce chromosome mutations in Chinese hamster ovary cells with and without activation; does not induce chromosome aberrations in the bone marrow of Sprague-Dawley rats exposed by inhalation (6 hours/day for 5 days); and does not induce sister chromatid exchange in Chinese hamster ovary cells with and without activation.
Developmental/Reproductive Toxicity: A three-generation reproductive study on the C9 fraction was conducted CD rats (30/sex/group) were exposed by inhalation to the C9 fraction at concentrations of 0, 100, 500, or 1500 ppm (0, 100, 500, or 1500 mg/kg/day) for 6 hours/day, 5

	<p>days/week. There was evidence of parental and reproductive toxicity at all dose levels. Indicators of parental toxicity included reduced body weights, increased salivation, hunched posture, aggressive behavior, and death. Indicators of adverse reproductive system effects included reduced litter size and reduced pup body weight. The LOEL was 100 ppm; a no-observed-effect level was not established. Developmental toxicity, including possible developmental neurotoxicity, was evident in rats in a 3-generation reproductive study.</p> <p>No effects on fecundity or fertility occurred in rats treated dermally with up to 0.3 mL/rat/day of a mixture of trimethyl-benzenes, 4-6 hours/day, 5 days/week over one generation.</p>
<p style="text-align: center;">ASPARTIC ACID, N,N'-(METHYLENEDICYCLOHEXANEDIYL)BIS-,ESTER</p>	<p>Evidence of sensitisation (adjuvant test) * After the first challenge very mild to clearly visible skin reddening was observed in 85% of the test substance animals. After the second challenge, very mild to clearly visible skin reddening was observed in 50% and 35% of the test substance animals challenged with 25% and 12% test substance respectively. A scaly administration site was observed in some animals. Rat repeat dose oral toxicity - 29 days NOAEL 1000 mg/kg/day * Genotoxicity ? bacterial reverse mutation non mutagenic * Genotoxicity ? in vitro not determined * Genotoxicity ? in vivo erythrocyte micronucleus test non clastogenic * The notified chemical is considered to be of low acute toxicity via the oral, dermal and inhalation routes. Irritation and Sensitisation. The material is considered to be a slight skin and eye irritant and mild respiratory irritant and a skin sensitiser. As skin reactions were observed in 85% of animals at a concentration of 50%, the substance is considered to be a strong sensitiser. The potential for respiratory sensitisation cannot be ruled out. Repeated Dose Toxicity. In a 28 day study in rats, the No Observed Adverse Effect Level (NOAEL) was established as 1000 mg/kg bw/day based on the absence of adverse treatment related effects. Mutagenicity. The material was negative in an Ames test and an in vivo erythrocyte micronucleus test. The substance is not considered to be mutagenic. Neurotoxicity: In the in vivo mouse erythrocyte micronucleus test, following intraperitoneal administration of a fairly high dose (5345 mg/kg bw) some evidence of non-specific neurological impairment was seen. However, this was not observed in any of the tests conducted on any other species and could either be species-specific or an expression of generalised toxicity induced at high doses, as opposed to specific neurotoxicity. * NICNAS Report</p> <p>Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens).</p> <p>Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis.</p>

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

Legend: ✘ – Data either not available or does not fill the criteria for classification
 ✔ – Data available to make classification

11.2 Information on other hazards

11.2.1. Endocrine disrupting properties

Many chemicals may mimic or interfere with the body's hormones, known as the endocrine system. Endocrine disruptors are chemicals that can interfere with endocrine (or hormonal) systems. Endocrine disruptors interfere with the synthesis, secretion, transport, binding, action, or elimination of natural hormones in the body. Any system in the body controlled by hormones can be derailed by hormone disruptors. Specifically, endocrine disruptors may be associated with the development of learning disabilities, deformations of the body various cancers and sexual development problems. Endocrine disrupting chemicals cause adverse effects in animals. But limited scientific information exists on potential health problems in humans. Because people are typically exposed to multiple endocrine disruptors at the same time, assessing public health effects is difficult.

11.2.2. Other information

See Section 11.1

SECTION 12 Ecological information

12.1. Toxicity

Diamond Coat	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	34mg/l	2
	EC50	48h	Crustacea	88.6mg/l	Not Available
	LC50	96h	Fish	66mg/l	2
aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	34mg/l	2
	EC50	48h	Crustacea	88.6mg/l	Not Available
	LC50	96h	Fish	66mg/l	2
naphtha petroleum, light aromatic solvent	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	19mg/l	1
	EC50	48h	Crustacea	6.14mg/l	1
	EC50	96h	Algae or other aquatic plants	64mg/l	2
N-(ethoxycarbonylphenyl)-N'-methyl-N'-phenylformamide	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	72h	Algae or other aquatic plants	1mg/l	1
	EC50	72h	Algae or other aquatic plants	2.53mg/l	2
	EC50	48h	Crustacea	2.7mg/l	2
	LC50	96h	Fish	1.4mg/l	2
propylene carbonate	Endpoint	Test Duration (hr)	Species	Value	Source
	ErC50	72h	Algae or other aquatic plants	29.09mg/l	2
	EC50(ECx)	72h	Algae or other aquatic plants	2.53mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	900mg/l	1
silica amorphous	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>900mg/l	1
	EC50	48h	Crustacea	>1000mg/l	1
	LC50	96h	Fish	1000mg/l	1
silica amorphous	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	14.1mg/l	2
	EC50	48h	Crustacea	>86mg/l	2
	EC50	96h	Algae or other aquatic plants	217.576mg/l	2
	LC50	96h	Fish	1033.016mg/l	2
Legend:	EC0(ECx) 24h Crustacea >=10000mg/l 1				
	<p>Extracted from 1. IUCALD 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</p>				

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

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

For high molecular weight synthetic polymers: (according to the Sustainable Futures (SF) program (U.S. EPA 2005b; U.S. EPA 2012c) polymer assessment guidance.)

High MW polymers are expected:

- to have low vapour pressure and are not expected to undergo volatilization .

- to adsorb strongly to soil and sediment
- to be non-biodegradable (not anticipated to be assimilated by microorganisms.- therefore, biodegradation is not expected to be an important removal process.

However many exceptions exist

High MW polymers are not expected to undergo removal by other degradative processes under environmental conditions

For 1,2,4 - Trimethylbenzene:

Half-life (hr) air: 0.48-16;

Half-life (hr) H₂O surface water: 0.24 -672;

Half-life (hr) H₂O ground: 336-1344;

Half-life (hr) soil: 168-672;

Henry's Pa m³ /mol: 385 -627;

Bioaccumulation: not significant. 1,2,4-Trimethylbenzene is a volatile organic compound (VOC) substance.

Atmospheric Fate: 1,2,4-trimethylbenzene can contribute to the formation of photochemical smog in the presence of other VOCs. Degradation of 1,2,4-trimethylbenzene in the atmosphere occurs by reaction with hydroxyl radicals. Reaction also occurs with ozone but very slowly (half life 8820 days).

Aquatic Fate: 1,2,4-Trimethylbenzene volatilizes rapidly from surface waters with volatilization half-life from a model river calculated to be 3.4 hours.

Biodegradation of 1,2,4-trimethylbenzene has been noted in both seawater and ground water. Various strains of Pseudomonas can biodegrade 1,2,4-trimethylbenzene.

Terrestrial Fate: 1,2,4-Trimethylbenzene also volatilizes from soils however; moderate adsorption to soils and sediments may occur. Volatilization is the major route of removal of 1,2,4- trimethylbenzene from soils; although, biodegradation may also occur. Due to the high volatility of the chemical it is unlikely to accumulate in soil or surface water to toxic concentrations.

Ecotoxicity: No significant bioaccumulation has been noted. 1,2,4-Trimethylbenzene is moderately toxic to fathead minnow and slightly toxic to dungeness crab.

1,2,4-Trimethylbenzene has moderate acute toxicity to aquatic organisms. No stress was observed in rainbow trout, sea lamprey and Daphnia magna water fleas.

The high concentrations required to induce toxicity in laboratory animals are not likely to be reached in the environment.

For Aromatic Substances Series:

Environmental Fate: Large, molecularly complex polycyclic aromatic hydrocarbons, or PAHs, are persistent in the environment longer than smaller PAHs.

Atmospheric Fate: PAHs are 'semi-volatile substances' which can move between the atmosphere and the Earth's surface in repeated, temperature-driven cycles of deposition and volatilization. Terrestrial Fate: BTEX compounds have the potential to move through soil and contaminate ground water, and their vapors are highly flammable and explosive.

Ecotoxicity - Within an aromatic series, acute toxicity increases with increasing alkyl substitution on the aromatic nucleus. The order of most toxic to least in a study using grass shrimp and brown shrimp was dimethylnaphthalenes > methylnaphthalenes >naphthalenes. Anthracene is a phototoxic PAH. UV light greatly increases the toxicity of anthracene to bluegill sunfish. Biological resources in strong sunlight are at more risk than those that are not. PAHs in general are more frequently associated with chronic risks.

Microbial methylation plays important roles in the biogeochemical cycling of the metalloids and possibly in their detoxification. Many microorganisms (bacteria, fungi, and yeasts) and animals are now known to biomethylate arsenic, forming both volatile (e.g., methylarsines) and nonvolatile (e.g., methylarsonic acid and dimethylarsinic acid) compounds. Antimony and bismuth, also undergo biomethylation to some extent. Trimethylstibine formation by microorganisms is now well established, but this process apparently does not occur in animals. Formation of trimethylbismuth by microorganisms has been reported in a few cases.

For Amorphous Silica: Amorphous silica is chemically and biologically inert. It is not biodegradable.

Aquatic Fate: Due to its insolubility in water there is a separation at every filtration and sedimentation process. On a global scale, the level of man-made synthetic amorphous silicas (SAS) represents up to 2.4% of the dissolved silica naturally present in the aquatic environment and untreated SAS have a relatively low water solubility and an extremely low vapour pressure. Biodegradability in sewage treatment plants or in surface water is not applicable to inorganic substances like SAS.

Terrestrial Fate: Crystalline and/or amorphous silicas are common on the earth in soils and sediments, and in living organisms (e.g. diatoms), but only the dissolved form is bioavailable. On the basis of these properties it is expected that SAS released into the environment will be distributed mainly into soil/sediment. Surface treated silica will be wetted then adsorbed onto soils and sediments.

Atmospheric Fate: SAS is not expected to be distributed into the air if released.

Ecotoxicity: SAS is not toxic to environmental organisms (apart from physical desiccation in insects). SAS presents a low risk for adverse effects to the environment.

For Silica:

Environmental Fate: Most documentation on the fate of silica in the environment concerns dissolved silica, in the aquatic environment, regardless of origin, (man-made or natural), or structure, (crystalline or amorphous).

Terrestrial Fate: Silicon makes up 25.7% of the Earth's crust, by weight, and is the second most abundant element, being exceeded only by oxygen. Silicon is not found free in nature, but occurs chiefly as the oxide and as silicates. Once released into the environment, no distinction can be made between the initial forms of silica.

Aquatic Fate: At normal environmental pH, dissolved silica exists exclusively as monosilicic acid. At pH 9.4, amorphous silica is highly soluble in water. Crystalline silica, in the form of quartz, has low solubility in water. Silicic acid plays an important role in the biological/geological/chemical cycle of silicon, especially in the ocean. Marine organisms such as diatoms, silicoflagellates and radiolarians use silicic acid in their skeletal structures and their skeletal remains leave silica in sea sediment

Ecotoxicity: Silicon is important to plant and animal life and is practically non-toxic to fish including zebrafish, and Daphnia magna water fleas.

DO NOT discharge into sewer or waterways.

12.2. Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
propylene carbonate	HIGH	HIGH
silica amorphous	LOW	LOW

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
propylene carbonate	LOW (LogKOW = -0.41)
silica amorphous	LOW (LogKOW = 0.5294)

12.4. Mobility in soil

Ingredient	Mobility
propylene carbonate	LOW (KOC = 14.85)
silica amorphous	LOW (KOC = 23.74)

12.5. Results of PBT and vPvB assessment

	P	B	T
Relevant available data	Not Available	Not Available	Not Available
PBT	✗	✗	✗
vPvB	✗	✗	✗
PBT Criteria fulfilled?	No		
vPvB	No		

12.6. Endocrine disrupting properties

The evidence linking adverse effects to endocrine disruptors is more compelling in the environment than it is in humans. Endocrine disruptors profoundly alter reproductive physiology of ecosystems and ultimately impact entire populations. Some endocrine-disrupting chemicals are slow to break down in the environment. That characteristic makes them potentially hazardous over long periods of time. Some well established adverse effects of endocrine disruptors in various wildlife species include eggshell-thinning, displayed of characteristics of the opposite sex and impaired reproductive development. Other adverse changes in wildlife species that have been suggested, but not proven include reproductive abnormalities, immune dysfunction and skeletal deformities.

12.7. Other adverse effects

No evidence of ozone depleting properties were found in the current literature.

SECTION 13 Disposal considerations

13.1. Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ Recycle wherever possible or consult manufacturer for recycling options. ▶ Consult State Land Waste Management Authority for disposal. ▶ 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.
Waste treatment options	Not Available
Sewage disposal options	Not Available

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

14.1. UN number or ID number	Not Applicable
14.2. UN proper shipping name	Not Applicable
14.3. Transport hazard class(es)	Class Not Applicable
	Subsidiary risk Not Applicable
14.4. Packing group	Not Applicable

14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Hazard identification (Kemler)	Not Applicable
	Classification code	Not Applicable
	Hazard Label	Not Applicable
	Special provisions	Not Applicable
	Limited quantity	Not Applicable
	Tunnel Restriction Code	Not Applicable

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

14.1. UN number	Not Applicable	
14.2. UN proper shipping name	Not Applicable	
14.3. Transport hazard class(es)	ICAO/IATA Class	Not Applicable
	ICAO / IATA Subsidiary Hazard	Not Applicable
	ERG Code	Not Applicable
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Special provisions	Not Applicable
	Cargo Only Packing Instructions	Not Applicable
	Cargo Only Maximum Qty / Pack	Not Applicable
	Passenger and Cargo Packing Instructions	Not Applicable
	Passenger and Cargo Maximum Qty / Pack	Not Applicable
	Passenger and Cargo Limited Quantity Packing Instructions	Not Applicable
	Passenger and Cargo Limited Maximum Qty / Pack	Not Applicable

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

14.1. UN number	Not Applicable	
14.2. UN proper shipping name	Not Applicable	
14.3. Transport hazard class(es)	IMDG Class	Not Applicable
	IMDG Subrisk	Not Applicable
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	EMS Number	Not Applicable
	Special provisions	Not Applicable
	Limited Quantities	Not Applicable

Inland waterways transport (ADN): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

14.1. UN number	Not Applicable	
14.2. UN proper shipping name	Not Applicable	
14.3. Transport hazard class(es)	Not Applicable	Not Applicable
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Not Applicable	

14.6. Special precautions for user	Classification code	Not Applicable
	Special provisions	Not Applicable
	Limited quantity	Not Applicable
	Equipment required	Not Applicable
	Fire cones number	Not Applicable

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

Product name	Group
aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester	Not Available
aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester	Not Available
naphtha petroleum, light aromatic solvent	Not Available
N-(ethoxycarbonylphenyl)-N'-methyl-N'-phenylformamidine	Not Available
propylene carbonate	Not Available
silica amorphous	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester	Not Available
aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester	Not Available
naphtha petroleum, light aromatic solvent	Not Available
N-(ethoxycarbonylphenyl)-N'-methyl-N'-phenylformamidine	Not Available
propylene carbonate	Not Available
silica amorphous	Not Available

SECTION 15 Regulatory information

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

aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester is found on the following regulatory lists

Europe EC Inventory

European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester is found on the following regulatory lists

Europe EC Inventory

European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

naphtha petroleum, light aromatic solvent is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

EU REACH Regulation (EC) No 1907/2006 - Annex XVII (Appendix 2)

Carcinogens: Category 1 B

EU REACH Regulation (EC) No 1907/2006 - Annex XVII (Appendix 4) Germ cell mutagens: Category 1 B

Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

N-(ethoxycarbonylphenyl)-N'-methyl-N'-phenylformamidine is found on the following regulatory lists

Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

propylene carbonate is found on the following regulatory lists

Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

silica amorphous is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances

European Union Directive (EU) 2017/2398 amending Directive 2004/37/EC on the protection of workers from the risks related to exposure to carcinogens or mutagens at work

Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

This safety data sheet is in compliance with the following EU legislation and its adaptations - as far as applicable - : Directives 98/24/EC, - 92/85/EEC, - 94/33/EC, - 2008/98/EC, - 2010/75/EU; Commission Regulation (EU) 2020/878; Regulation (EC) No 1272/2008 as updated through ATPs.

Information according to 2012/18/EU (Seveso III):**Seveso Category**

Not Available

15.2. Chemical safety assessment

No Chemical Safety Assessment has been carried out for this substance/mixture by the supplier.

ECHA SUMMARY

Ingredient	CAS number	Index No	ECHA Dossier
aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester	136210-30-5	607-521-00-8	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Skin Sens. 1; Aquatic Chronic 3	GHS07; Wng	H317; H412
2	Skin Sens. 1B; Aquatic Chronic 1	GHS07; Wng; GHS09	H317; H410

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester	136210-32-7	607-350-00-9	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Skin Sens. 1; Aquatic Chronic 3	GHS07; Wng	H317; H412
2	Skin Sens. 1B; Aquatic Chronic 1	GHS07; Wng; GHS09	H317; H410

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
naphtha petroleum, light aromatic solvent	64742-95-6.	649-356-00-4	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Flam. Liq. 3; Asp. Tox. 1; Skin Irrit. 2; Eye Irrit. 2; STOT SE 3; STOT SE 3; Aquatic Chronic 2	GHS08; GHS02; GHS09; Dgr	H226; H304; H315; H319; H335; H336
2	Flam. Liq. 3; Asp. Tox. 1; Skin Irrit. 2; Eye Irrit. 2; STOT SE 3; STOT SE 3; Aquatic Chronic 2	GHS08; GHS02; GHS09; Dgr	H226; H304; H315; H319; H335; H336; H411
1	Asp. Tox. 1; Muta. 1B; Carc. 1B	GHS08; Dgr	H304; H340; H350

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
2	Asp. Tox. 1; Muta. 1A; Carc. 1A; Skin Irrit. 2; STOT SE 3; Aquatic Chronic 2; Repr. 2; Flam. Liq. 1; Eye Irrit. 2; STOT SE 3; Acute Tox. 4; Aquatic Acute 1; Acute Tox. 4; STOT RE 1	GHS08; Dgr; GHS02; GHS09	H304; H340; H350; H315; H336; H411; H361; H224; H335; H318; H332; H302; H372

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
N-(ethoxycarbonylphenyl)-N'-methyl-N'-phenylformamidine	57834-33-0	Not Available	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Acute Tox. 4	GHS07; Wng	H302
2	STOT RE 2; Aquatic Chronic 2; Acute Tox. 4; Skin Sens. 1	GHS09; Wng; GHS08	H373; H411; H302; H317; H315; H319; H335

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
propylene carbonate	108-32-7	607-194-00-1	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Eye Irrit. 2	GHS07; Wng	H319
2	Eye Irrit. 2A; Skin Irrit. 2; Acute Tox. 4	GHS07; Wng	H319; H315; H312
1	Skin Irrit. 2; Eye Irrit. 2; STOT SE 3	GHS07; Wng	H315; H319; H335
2	Skin Irrit. 2; Eye Irrit. 2; STOT SE 3	GHS07; Wng	H315; H319; H335
1	Skin Irrit. 2; Eye Irrit. 2; STOT SE 3	GHS07; Wng	H315; H319; H335
2	Skin Irrit. 2; Eye Irrit. 2; STOT SE 3	GHS07; Wng	H315; H319; H335

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
silica amorphous	7631-86-9	Not Available	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Not Classified	Not Available	Not Available
2	Eye Irrit. 2; STOT SE 3; STOT RE 1; Acute Tox. 4; STOT SE 1; Acute Tox. 4; Skin Corr. 1C; Aquatic Chronic 3; Carc. 1A; Flam. Liq. 2; Asp. Tox. 1; Muta. 1B; Water-react. 1; Skin Sens. 1; Acute Tox. 2	GHS08; Dgr; GHS05; GHS09; GHS01; GHS06	H335; H319; H372; H302; H370; H312; H314; H251; H260; H340; H350; H225; H304; H317; H334; H411; H330
1	Skin Irrit. 2; Eye Irrit. 2; STOT SE 3	GHS07; Wng	H315; H319; H335
2	Skin Irrit. 2; STOT SE 3; Carc. 1A; Acute Tox. 4; Acute Tox. 4; Eye Dam. 1; STOT RE 1	GHS08; Dgr; GHS05	H315; H335; H350; H302; H332; H318; H372
1	Not Classified	Not Available	Not Available
2	Eye Irrit. 2; STOT SE 3; Skin Irrit. 2; Carc. 1A; STOT SE 1; STOT RE 1	GHS08; Dgr	H319; H335; H315; H350; H370; H372
1	Not Classified	Not Available	Not Available
2	Acute Tox. 4; Eye Irrit. 2; STOT SE 3; Skin Irrit. 2; STOT RE 1	GHS08; Dgr	H319; H335; H351; H315; H372
2	Acute Tox. 4; Acute Tox. 5	GHS07; Wng	H332; H303
1	Not Classified	Not Available	Not Available
1	Not Classified	Not Available	Not Available
2	Acute Tox. 1; STOT RE 2; Eye Irrit. 2; Skin Irrit. 2; STOT SE 3	GHS06; Dgr; GHS08	H330; H373; H319; H315; H335
1	Not Classified	Not Available	Not Available

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
2	Skin Irrit. 2; Eye Irrit. 2; STOT RE 2; Acute Tox. 4	Wng; GHS08	H315; H319; H373; H332
1	Not Classified	Not Available	Not Available
2	STOT RE 2	GHS08; Wng	H373
1	STOT RE 1	GHS08; Dgr	H372
2	STOT RE 1	GHS08; Dgr	H372
1			
2			
1	Not Classified	Not Available	Not Available
2	STOT SE 2; STOT RE 1	GHS08; Dgr	H371; H372

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

National Inventory Status

National Inventory	Status
Australia - AIIIC / Australia Non-Industrial Use	Yes
Canada - DSL	No (N-(ethoxycarbonylphenyl)-N'-methyl-N'-phenylformamidine)
Canada - NDSL	No (aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester; aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester; naphtha petroleum, light aromatic solvent; propylene carbonate)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester)
Japan - ENCS	No (aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester; aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester; N-(ethoxycarbonylphenyl)-N'-methyl-N'-phenylformamidine)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester; aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester)
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester; aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester; N-(ethoxycarbonylphenyl)-N'-methyl-N'-phenylformamidine)
Vietnam - NCI	Yes
Russia - FBEPH	No (N-(ethoxycarbonylphenyl)-N'-methyl-N'-phenylformamidine)
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	06/09/2023
Initial Date	06/09/2023

Full text Risk and Hazard codes

H224	Extremely flammable liquid and vapour.
H225	Highly flammable liquid and vapour.
H226	Flammable liquid and vapour.
H251	Self-heating: may catch fire.
H260	In contact with water releases flammable gases which may ignite spontaneously.
H302	Harmful if swallowed.
H303	May be harmful if swallowed.
H304	May be fatal if swallowed and enters airways.
H312	Harmful in contact with skin.
H314	Causes severe skin burns and eye damage.

H315	Causes skin irritation.
H318	Causes serious eye damage.
H319	Causes serious eye irritation.
H330	Fatal if inhaled.
H332	Harmful if inhaled.
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H335	May cause respiratory irritation.
H336	May cause drowsiness or dizziness.
H340	May cause genetic defects.
H350	May cause cancer.
H351	Suspected of causing cancer.
H361	Suspected of damaging fertility or the unborn child.
H370	Causes damage to organs.
H371	May cause damage to organs.
H372	Causes damage to organs through prolonged or repeated exposure.
H373	May cause damage to organs through prolonged or repeated exposure.
H410	Very toxic to aquatic life with long lasting effects.
H411	Toxic to aquatic life with long lasting effects.

Other information

Ingredients with multiple cas numbers

Name	CAS No
aspartic acid, N,N'-(methylenedicyclohexanediyl)bis-,ester	136210-32-7, 188364-76-3
naphtha petroleum, light aromatic solvent	64742-95-6., 25550-14-5.
propylene carbonate	108-32-7, 51260-39-0, 16606-55-6
silica amorphous	7631-86-9, 112945-52-5, 67762-90-7, 68611-44-9, 68909-20-6, 112926-00-8, 61790-53-2, 60676-86-0, 91053-39-3, 69012-64-2, 844491-94-7

Classification of the preparation and its individual components has drawn on official and authoritative sources 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.

For detailed advice on Personal Protective Equipment, refer to the following EU CEN Standards:

- EN 166 Personal eye-protection
- EN 340 Protective clothing
- EN 374 Protective gloves against chemicals and micro-organisms
- EN 13832 Footwear protecting against chemicals
- EN 133 Respiratory protective devices

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
- AII: 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

Classification and procedure used to derive the classification for mixtures according to Regulation (EC) 1272/2008 [CLP]

Classification according to regulation (EC) No 1272/2008 [CLP] and amendments	Classification Procedure
Sensitisation (Skin) Category 1, H317	Calculation method
Germ Cell Mutagenicity Category 2, H341	Calculation method
Hazardous to the Aquatic Environment Long-Term Hazard Category 3, H412	Calculation method