

# DNA BaZecoats (HeliX/Metallic/Profile/Camo)

PTW

Chemwatch Hazard Alert Code: 3

Chemwatch: 4992-78

Version No: 6.1.1.1

Safety Data Sheet according to WHS and ADG requirements

Issue Date: 18/09/2015

Print Date: 26/11/2015

Initial Date: **Not Available**

L.GHS.AUS.EN

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

### Product Identifier

Product name	DNA BaZecoats (HeliX/Metallic/Profile/Camo)
Proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)
Other means of identification	Part No.: B5__-, BM__- & B1__-

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

Relevant identified uses	Surface coatings.
--------------------------	-------------------

### Details of the supplier of the safety data sheet

Registered company name	PTW
Address	5-7 Keith Campbell Court Scoresby 3179 VIC Australia
Telephone	+61 3 9764 2088
Fax	+61 3 9764 1244
Website	Not Available
Email	info@productsthatwork.com.au

### Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	+61 3 9573 3112
Other emergency telephone numbers	Not Available

## SECTION 2 HAZARDS IDENTIFICATION

### Classification of the substance or mixture

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

#### CHEMWATCH HAZARD RATINGS

	Min	Max
Flammability	3	4
Toxicity	2	3
Body Contact	2	3
Reactivity	1	2
Chronic	3	4

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

Poisons Schedule	Not Applicable
GHS Classification [1]	Flammable Liquid Category 2, Acute Toxicity (Dermal) Category 4, Acute Toxicity (Inhalation) Category 4, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Carcinogen Category 2, Reproductive Toxicity Category 1B, STOT - SE (Narcosis) Category 3, Aspiration Hazard Category 1, Chronic Aquatic Hazard Category 3
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HSIS; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

### Label elements

GHS label elements	
--------------------	---

SIGNAL WORD **DANGER**

### Hazard statement(s)

H225	Highly flammable liquid and vapour
H312	Harmful in contact with skin

Continued...

## DNA BaZecoats (HeliX/Metallic/Profile/Camo)

H332	Harmful if inhaled
H315	Causes skin irritation
H319	Causes serious eye irritation
H351	Suspected of causing cancer
H360	May damage fertility or the unborn child
H336	May cause drowsiness or dizziness
H304	May be fatal if swallowed and enters airways
H412	Harmful to aquatic life with long lasting effects
AUH066	Repeated exposure may cause skin dryness and cracking

## Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat/sparks/open flames/hot surfaces. - No smoking.
P271	Use only outdoors or in a well-ventilated area.
P281	Use personal protective equipment as required.
P240	Ground/bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use only non-sparking tools.
P243	Take precautionary measures against static discharge.
P261	Avoid breathing dust/fume/gas/mist/vapours/spray.
P273	Avoid release to the environment.
P280	Wear protective gloves/protective clothing/eye protection/face protection.

## Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER or doctor/physician.
P308+P313	IF exposed or concerned: Get medical advice/attention.
P331	Do NOT induce vomiting.
P362	Take off contaminated clothing and wash before reuse.
P363	Wash contaminated clothing before reuse.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam for extinction.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER or doctor/physician if you feel unwell.
P337+P313	If eye irritation persists: Get medical advice/attention.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P303+P361+P353	IF ON SKIN (or hair): Remove/Take off immediately all contaminated clothing. Rinse skin with water/shower.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.
P332+P313	If skin irritation occurs: Get medical advice/attention.

## Precautionary statement(s) Storage

P403+P235	Store in a well-ventilated place. Keep cool.
P405	Store locked up.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

## Precautionary statement(s) Disposal

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

## SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

## Substances

See section below for composition of Mixtures

## Mixtures

CAS No	%[weight]	Name
1330-20-7	20-25	<u>xylene</u>
123-86-4	20-25	<u>n-butyl acetate</u>
108-65-6	10-20	<u>propylene glycol monomethyl ether acetate, alpha-isomer</u>
Not Available	5-15	acrylic polyol/polymer/resin
8032-32-4.	5-10	<u>petroleum ether</u>
100-41-4	0-5	<u>ethylbenzene</u>
71-36-3	0-5	<u>n-butanol</u>

## DNA BaZecoats (HeliX/Metallic/Profile/Camo)

1333-86-4	0-5	<u>carbon black</u>
112-07-2	0-5	<u>ethylene glycol monobutyl ether acetate</u>
111-15-9	0-5	<u>2-ethoxyethyl acetate</u>

## SECTION 4 FIRST AID MEASURES

## Description of first aid measures

<b>Eye Contact</b>	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> <li>▶ Wash out immediately with fresh running water.</li> <li>▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.</li> <li>▶ Seek medical attention without delay; if pain persists or recurs seek medical attention.</li> <li>▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.</li> </ul>
<b>Skin Contact</b>	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> <li>▶ Immediately remove all contaminated clothing, including footwear.</li> <li>▶ Flush skin and hair with running water (and soap if available).</li> <li>▶ Seek medical attention in event of irritation.</li> </ul>
<b>Inhalation</b>	<ul style="list-style-type: none"> <li>▶ If fumes or combustion products are inhaled remove from contaminated area.</li> <li>▶ Lay patient down. Keep warm and rested.</li> <li>▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.</li> <li>▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.</li> <li>▶ Transport to hospital, or doctor.</li> </ul>
<b>Ingestion</b>	<ul style="list-style-type: none"> <li>▶ <b>If swallowed do NOT induce vomiting.</b></li> <li>▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.</li> <li>▶ Observe the patient carefully.</li> <li>▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.</li> <li>▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.</li> <li>▶ Seek medical advice.</li> <li>▶ Avoid giving milk or oils.</li> <li>▶ Avoid giving alcohol.</li> </ul>

## Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.

for simple esters:

## BASIC TREATMENT

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

## ADVANCED TREATMENT

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

## EMERGENCY DEPARTMENT

- ▶ Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.
- ▶ Positive end-expiratory pressure (PEEP)-assisted ventilation may be required for acute parenchymal injury or adult respiratory distress syndrome.
- ▶ Consult a toxicologist as necessary.

BRONSTEIN, A.C. and CURRANCE, P.L. EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

For acute or short term repeated exposures to xylene:

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

## BIOLOGICAL EXPOSURE INDEX - BEI

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

Continued...

## DNA BaZecoats (HeliX/Metallic/Profile/Camo)

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

## SECTION 5 FIREFIGHTING MEASURES

## Extinguishing media

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

## Special hazards arising from the substrate or mixture

<b>Fire Incompatibility</b>	▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
-----------------------------	--

## Advice for firefighters

<b>Fire Fighting</b>	<ul style="list-style-type: none"> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ May be violently or explosively reactive.</li> <li>▶ Wear breathing apparatus plus protective gloves in the event of a fire.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▶ Consider evacuation (or protect in place).</li> <li>▶ Fight fire from a safe distance, with adequate cover.</li> <li>▶ If safe, switch off electrical equipment until vapour fire hazard removed.</li> <li>▶ Use water delivered as a fine spray to control the fire and cool adjacent area.</li> <li>▶ Avoid spraying water onto liquid pools.</li> <li>▶ <b>Do not approach containers suspected to be hot.</b></li> <li>▶ Cool fire exposed containers with water spray from a protected location.</li> <li>▶ If safe to do so, remove containers from path of fire.</li> </ul>
<b>Fire/Explosion Hazard</b>	<ul style="list-style-type: none"> <li>▶ Liquid and vapour are highly flammable.</li> <li>▶ Severe fire hazard when exposed to heat, flame and/or oxidisers.</li> <li>▶ Vapour may travel a considerable distance to source of ignition.</li> <li>▶ Heating may cause expansion or decomposition leading to violent rupture of containers.</li> <li>▶ On combustion, may emit toxic fumes of carbon monoxide (CO).</li> </ul> <p>Combustion products include; carbon dioxide (CO<sub>2</sub>) other pyrolysis products typical of burning organic material</p>

## SECTION 6 ACCIDENTAL RELEASE MEASURES

## Personal precautions, protective equipment and emergency procedures

<b>Minor Spills</b>	<ul style="list-style-type: none"> <li>▶ Remove all ignition sources.</li> <li>▶ Clean up all spills immediately.</li> <li>▶ Avoid breathing vapours and contact with skin and eyes.</li> <li>▶ Control personal contact with the substance, by using protective equipment.</li> <li>▶ Contain and absorb small quantities with vermiculite or other absorbent material.</li> <li>▶ Wipe up.</li> <li>▶ Collect residues in a flammable waste container.</li> </ul>
<b>Major Spills</b>	<ul style="list-style-type: none"> <li>▶ Clear area of personnel and move upwind.</li> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ May be violently or explosively reactive.</li> <li>▶ Wear breathing apparatus plus protective gloves.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▶ Consider evacuation (or protect in place).</li> <li>▶ No smoking, naked lights or ignition sources.</li> <li>▶ Increase ventilation.</li> <li>▶ Stop leak if safe to do so.</li> <li>▶ Water spray or fog may be used to disperse /absorb vapour.</li> <li>▶ Contain spill with sand, earth or vermiculite.</li> <li>▶ Use only spark-free shovels and explosion proof equipment.</li> <li>▶ Collect recoverable product into labelled containers for recycling.</li> <li>▶ Absorb remaining product with sand, earth or vermiculite.</li> <li>▶ Collect solid residues and seal in labelled drums for disposal.</li> <li>▶ Wash area and prevent runoff into drains.</li> <li>▶ If contamination of drains or waterways occurs, advise emergency services.</li> </ul>

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

## SECTION 7 HANDLING AND STORAGE

## Precautions for safe handling

<b>Safe handling</b>	<ul style="list-style-type: none"> <li>▶ Avoid all personal contact, including inhalation.</li> <li>▶ Wear protective clothing when risk of exposure occurs.</li> <li>▶ Use in a well-ventilated area.</li> <li>▶ Prevent concentration in hollows and sumps.</li> <li>▶ <b>DO NOT enter confined spaces until atmosphere has been checked.</b></li> <li>▶ Avoid smoking, naked lights, heat or ignition sources.</li> <li>▶ When handling, <b>DO NOT eat, drink or smoke.</b></li> <li>▶ Vapour may ignite on pumping or pouring due to static electricity.</li> <li>▶ <b>DO NOT use plastic buckets.</b></li> <li>▶ Earth and secure metal containers when dispensing or pouring product.</li> </ul>
----------------------	--

## DNA BaZecoats (HeliX/Metallic/Profile/Camo)

	<ul style="list-style-type: none"> <li>▶ Use spark-free tools when handling.</li> <li>▶ Avoid contact with incompatible materials.</li> <li>▶ Keep containers securely sealed.</li> <li>▶ Avoid physical damage to containers.</li> <li>▶ Always wash hands with soap and water after handling.</li> <li>▶ Work clothes should be laundered separately.</li> <li>▶ Use good occupational work practice.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> <li>▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.</li> <li>▶ <b>DO NOT allow clothing wet with material to stay in contact with skin</b></li> </ul>
<b>Other information</b>	<ul style="list-style-type: none"> <li>▶ Store in original containers in approved flame-proof area.</li> <li>▶ No smoking, naked lights, heat or ignition sources.</li> <li>▶ <b>DO NOT store in pits, depressions, basements or areas where vapours may be trapped.</b></li> <li>▶ Keep containers securely sealed.</li> <li>▶ Store away from incompatible materials in a cool, dry well ventilated area.</li> <li>▶ Protect containers against physical damage and check regularly for leaks.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> </ul>

**Conditions for safe storage, including any incompatibilities**

<b>Suitable container</b>	<ul style="list-style-type: none"> <li>▶ Packing as supplied by manufacturer.</li> <li>▶ Plastic containers may only be used if approved for flammable liquid.</li> <li>▶ Check that containers are clearly labelled and free from leaks.</li> <li>▶ For low viscosity materials (i) : Drums and jerry cans must be of the non-removable head type. (ii) : Where a can is to be used as an inner package, the can must have a screwed enclosure.</li> <li>▶ For materials with a viscosity of at least 2680 cSt. (23 deg. C)</li> <li>▶ For manufactured product having a viscosity of at least 250 cSt. (23 deg. C)</li> <li>▶ Manufactured product that requires stirring before use and having a viscosity of at least 20 cSt (25 deg. C): (i) Removable head packaging; (ii) Cans with friction closures and (iii) low pressure tubes and cartridges may be used.</li> <li>▶ Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages</li> <li>▶ In addition, where inner packagings are glass and contain liquids of packing group I there must be sufficient inert absorbent to absorb any spillage, unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.</li> </ul>
<b>Storage incompatibility</b>	<ul style="list-style-type: none"> <li>▶ Avoid reaction with oxidising agents</li> </ul>



X      X      X      X      +      X      +

**X** — Must not be stored together  
**0** — May be stored together with specific preventions  
**+** — May be stored together

**SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION****Control parameters****OCCUPATIONAL EXPOSURE LIMITS (OEL)****INGREDIENT DATA**

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	xylene	Xylene (o-, m-, p-isomers)	350 mg/m <sup>3</sup> / 80 ppm	655 mg/m <sup>3</sup> / 150 ppm	Not Available	Not Available
Australia Exposure Standards	n-butyl acetate	n-Butyl acetate	713 mg/m <sup>3</sup> / 150 ppm	950 mg/m <sup>3</sup> / 200 ppm	Not Available	Not Available
Australia Exposure Standards	propylene glycol monomethyl ether acetate, alpha-isomer	1-Methoxy-2-propanol acetate	274 mg/m <sup>3</sup> / 50 ppm	548 mg/m <sup>3</sup> / 100 ppm	Not Available	Sk
Australia Exposure Standards	ethylbenzene	Ethyl benzene	434 mg/m <sup>3</sup> / 100 ppm	543 mg/m <sup>3</sup> / 125 ppm	Not Available	Not Available
Australia Exposure Standards	n-butanol	n-Butyl alcohol	Not Available	Not Available	152 mg/m <sup>3</sup> / 50 ppm	Sk
Australia Exposure Standards	carbon black	Carbon black	3 mg/m <sup>3</sup>	Not Available	Not Available	Not Available
Australia Exposure Standards	ethylene glycol monobutyl ether acetate	2-Butoxyethyl acetate	133 mg/m <sup>3</sup> / 20 ppm	333 mg/m <sup>3</sup> / 50 ppm	Not Available	Sk
Australia Exposure Standards	2-ethoxyethyl acetate	2-Ethoxyethyl acetate	27 mg/m <sup>3</sup> / 5 ppm	Not Available	Not Available	Sk

**EMERGENCY LIMITS**

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
xylene	Xylenes	Not Available	Not Available	Not Available
n-butyl acetate	Butyl acetate, n-	Not Available	Not Available	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Propylene glycol monomethyl ether acetate, alpha-isomer; (1-Methoxypropyl-2-acetate)	Not Available	Not Available	Not Available
petroleum ether	Petroleum spirits; (VM & P Naphtha, Ligroine, Paint solvent)	75 ppm	400 ppm	400 ppm
ethylbenzene	Ethyl benzene	Not Available	Not Available	Not Available
n-butanol	Butyl alcohol, n-; (n-Butanol)	20 ppm	50 ppm	8000 ppm

## DNA BaZecoats (HeliX/Metallic/Profile/Camo)

carbon black	Carbon black	9 mg/m3	99 mg/m3	590 mg/m3
ethylene glycol monobutyl ether acetate	Butoxyethanol acetate, 2-; (Ethylene glycol monobutyl ether acetate)	20 ppm	20 ppm	73 ppm
2-ethoxyethyl acetate	Ethoxyethylacetate, 2-	15 ppm	420 ppm	2500 ppm

Ingredient	Original IDLH	Revised IDLH
xylene	1,000 ppm	900 ppm
n-butyl acetate	10,000 ppm	1,700 [LEL] ppm
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available	Not Available
acrylic polyol/polymer/resin	Not Available	Not Available
petroleum ether	Not Available	Not Available
ethylbenzene	2,000 ppm	800 [LEL] ppm
n-butanol	8,000 ppm	1,400 [LEL] ppm
carbon black	N.E. mg/m3 / N.E. ppm	1,750 mg/m3
ethylene glycol monobutyl ether acetate	Not Available	Not Available
2-ethoxyethyl acetate	2,500 ppm	500 ppm

**MATERIAL DATA**

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:

Class OSF 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 26-550 As "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

Odour Safety Factor(OSF) OSF=0.15 (petroleum ether)

NOTE M: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.005% w/w benzo[a]pyrene (EINECS No 200-028-5). 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

NOTE P: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.01% w/w benzene (EINECS No 200-753-7). Note E shall also apply when the substance is classified as a carcinogen. This note applies only to certain complex oil-derived substances in Annex VI.

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

**Exposure controls**

<b>Appropriate engineering controls</b>	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>For flammable liquids and flammable gases, local exhaust ventilation or a process enclosure ventilation system may be required. Ventilation equipment should be explosion-resistant.</p> <p>Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.</p>								
	<table border="1"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>solvent, vapours, degreasing etc., evaporating from tank (in still air).</td> <td>0.25-0.5 m/s (50-100 f/min.)</td> </tr> <tr> <td>aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)</td> <td>0.5-1 m/s (100-200 f/min.)</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> </tbody> </table>	Type of Contaminant:	Air Speed:	solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)	aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
	Type of Contaminant:	Air Speed:							
	solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)							
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)								
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)								
<p>Within each range the appropriate value depends on:</p> <table border="1"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> </tbody> </table>	Lower end of the range	Upper end of the range	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity			
Lower end of the range	Upper end of the range								
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents								
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity								

Continued...

## DNA BaZecoats (HeliX/Metallic/Profile/Camo)

	3: Intermittent, low production. 4: Large hood or large air mass in motion	3: High production, heavy use 4: Small hood-local control only
	Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.	
Personal protection		
Eye and face protection	<ul style="list-style-type: none"> <li>▶ Safety glasses with side shields.</li> <li>▶ Chemical goggles.</li> <li>▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]</li> </ul>	
Skin protection	See Hand protection below	
Hands/feet protection	<ul style="list-style-type: none"> <li>▶ Wear chemical protective gloves, e.g. PVC.</li> <li>▶ Wear safety footwear or safety gumboots, e.g. Rubber</li> </ul>	
Body protection	See Other protection below	
Other protection	<ul style="list-style-type: none"> <li>▶ Overalls.</li> <li>▶ PVC Apron.</li> <li>▶ PVC protective suit may be required if exposure severe.</li> <li>▶ Eyewash unit.</li> <li>▶ Ensure there is ready access to a safety shower.</li> </ul>	
Thermal hazards	Not Available	

## Recommended material(s)

## GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

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

DNA BaZecoats (HeliX/Metallic/Profile/Camo)

Material	CPI
##2-ethoxyethyl	acetate
##ethylene glycol monobutyl ether	acetate
##n-butyl	acetate
BUTYL	C
BUTYL/NEOPRENE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE	C
PE/EVAL/PE	C
PVA	C
PVC	C
PVDC/PE/PVDC	C
SARANEX-23	C
TEFLON	C
VITON	C
VITON/BUTYL	C

\* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

\* Where the glove is to be used on a short term, casual or infrequent basis, factors such as

## Respiratory protection

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

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 5 x ES	AX-AUS / Class 1	-	AX-PAPR-AUS / Class 1
up to 25 x ES	Air-line*	AX-2	AX-PAPR-2
up to 50 x ES	-	AX-3	-
50+ x ES	-	Air-line**	-

\* - Continuous-flow; \*\* - Continuous-flow or positive pressure demand

^ - Full-face

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

## DNA BaZecoats (HeliX/Metallic/Profile/Camo)

"feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

## SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

## Information on basic physical and chemical properties

<b>Appearance</b>	Opaque, highly flammable liquid with strong solvent odour; does not mix with water.		
<b>Physical state</b>	Liquid	<b>Relative density (Water = 1)</b>	0.93
<b>Odour</b>	Not Available	<b>Partition coefficient n-octanol / water</b>	Not Available
<b>Odour threshold</b>	Not Available	<b>Auto-ignition temperature (°C)</b>	Not Available
<b>pH (as supplied)</b>	Not Applicable	<b>Decomposition temperature</b>	Not Available
<b>Melting point / freezing point (°C)</b>	Not Available	<b>Viscosity (cSt)</b>	Not Available
<b>Initial boiling point and boiling range (°C)</b>	108-186	<b>Molecular weight (g/mol)</b>	Not Applicable
<b>Flash point (°C)</b>	7	<b>Taste</b>	Not Available
<b>Evaporation rate</b>	< 1 Ether = 1	<b>Explosive properties</b>	Not Available
<b>Flammability</b>	HIGHLY FLAMMABLE.	<b>Oxidising properties</b>	Not Available
<b>Upper Explosive Limit (%)</b>	15.7	<b>Surface Tension (dyn/cm or mN/m)</b>	Not Available
<b>Lower Explosive Limit (%)</b>	0.88	<b>Volatile Component (%vol)</b>	85
<b>Vapour pressure (kPa)</b>	Not Available	<b>Gas group</b>	Not Available
<b>Solubility in water (g/L)</b>	Immiscible	<b>pH as a solution (1%)</b>	Not Applicable
<b>Vapour density (Air = 1)</b>	> 1	<b>VOC g/L</b>	Not Available

## SECTION 10 STABILITY AND REACTIVITY

<b>Reactivity</b>	See section 7
<b>Chemical stability</b>	<ul style="list-style-type: none"> <li>▶ Unstable in the presence of incompatible materials.</li> <li>▶ Product is considered stable.</li> <li>▶ Hazardous polymerisation will not occur.</li> </ul>
<b>Possibility of hazardous reactions</b>	See section 7
<b>Conditions to avoid</b>	See section 7
<b>Incompatible materials</b>	See section 7
<b>Hazardous decomposition products</b>	See section 5

## SECTION 11 TOXICOLOGICAL INFORMATION

## Information on toxicological effects

<b>Inhaled</b>	<p>Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful.</p> <p>Limited evidence exists that exposure to the material may produce irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation.</p> <p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>If exposure to highly concentrated solvent atmosphere is prolonged this may lead to narcosis, unconsciousness, even coma and possible death.</p> <p>Xylene is a central nervous system depressant. 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>Headache, fatigue, lassitude, irritability and gastrointestinal disturbances (e.g., nausea, anorexia and flatulence) are the most common symptoms of xylene overexposure. Injury to the heart, liver, kidneys and nervous system has also been noted amongst workers. Transient memory loss, renal impairment, temporary confusion and some evidence of disturbance of liver function was reported in three workers overcome by gross exposure to xylene (10000 ppm). One worker died and autopsy revealed pulmonary congestion, oedema and focal alveolar haemorrhage. Volunteers inhaling xylene at 100 ppm for 5 to 6 hours showed changes in manual coordination reaction time and slight ataxia. Tolerance developed during the workweek but was lost over the weekend. Physical exercise may antagonise this effect. Xylene body burden in humans exposed to 100 or 200 ppm xylene in air depends on the amount of body fat with 4% to 8% of total absorbed xylene accumulating in adipose tissue.</p>
<b>Ingestion</b>	<p>Accidental ingestion of the material may be damaging to the health of the individual.</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>Ingestion of petroleum hydrocarbons may produce irritation of the pharynx, oesophagus, stomach and small intestine with oedema and mucosal ulceration resulting; symptoms include a burning sensation in the mouth and throat. Large amounts may produce narcosis with nausea and vomiting, weakness or dizziness, slow and shallow respiration, swelling of the abdomen, unconsciousness and convulsions. Myocardial injury may produce arrhythmias, ventricular fibrillation and electrocardiographic changes. Central nervous system depression may also occur. Light aromatic hydrocarbons produce a warm, sharp, tingling sensation on contact with taste buds and may anaesthetise the tongue. Aspiration into the lungs may produce coughing, gagging and a chemical pneumonitis with pulmonary oedema and haemorrhage.</p> <p>Considered an unlikely route of entry in commercial/industrial environments The liquid may produce considerable gastrointestinal discomfort and may be</p>

## DNA BaZecoats (HeliX/Metallic/Profile/Camo)

	harmful or toxic if swallowed. Ingestion may result in nausea, pain and vomiting. Vomit entering the lungs by aspiration may cause potentially lethal chemical pneumonitis	
<b>Skin Contact</b>	<p>Limited evidence exists that exposure to the material may produce irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact.</p> <p>The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either</p> <ul style="list-style-type: none"> <li>▶ produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or</li> <li>▶ produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period.</li> </ul> <p>Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. Toxic effects may result from skin absorption</p> <p>Exposure limits with "skin" notation indicate that vapour and liquid may be absorbed through intact skin. Absorption by skin may readily exceed vapour inhalation exposure. Symptoms for skin absorption are the same as for inhalation. Contact with eyes and mucous membranes may also contribute to overall exposure and may also invalidate the exposure standard.</p> <p>Absorption by skin may readily exceed vapour inhalation exposure. Symptoms for skin absorption are the same as for inhalation.</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>	
<b>Eye</b>	<p>Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p> <p>Symptoms of exposure by the eye to carbon particulates include irritation and a burning sensation. Following an industrial explosion, fine particles become embedded in the cornea and conjunctiva resulting in an inflammation which persisted for 2-3 weeks. Some particles remained permanently producing a punctate purplish-black discolouration.</p>	
<b>Chronic</b>	<p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.</p> <p>Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).</p> <p>Chronic solvent inhalation exposures may result in nervous system impairment and liver and blood changes. [PATTYS]</p> <p>Prolonged or repeated contact with xylenes may cause defatting dermatitis with drying and cracking. Chronic inhalation of xylenes has been associated with central nervous system effects, loss of appetite, nausea, ringing in the ears, irritability, thirst anaemia, mucosal bleeding, enlarged liver and hyperplasia.</p> <p>Exposure may produce kidney and liver damage. In chronic occupational exposure, xylene (usually mixed with other solvents) has produced irreversible damage to the central nervous system and ototoxicity (damages hearing and increases sensitivity to noise), probably due to neurotoxic mechanisms.</p> <p>Industrial workers exposed to xylene with a maximum level of ethyl benzene of 0.06 mg/l (14 ppm) reported headaches and irritability and tired quickly. Functional nervous system disturbances were found in some workers employed for over 7 years whilst other workers had enlarged livers.</p> <p>Xylene has been classed as a developmental toxin in some jurisdictions.</p> <p>Small excess risks of spontaneous abortion and congenital malformations were reported amongst women exposed to xylene in the first trimester of pregnancy. In all cases, however, the women were also exposed to other substances. Evaluation of workers chronically exposed to xylene has demonstrated lack of genotoxicity. Exposure to xylene has been associated with increased risks of haemopoietic malignancies but, again, simultaneous exposure to other substances (including benzene) complicates the picture. A long-term gavage study to mixed xylenes (containing 17% ethyl benzene) found no evidence of carcinogenic activity in rats and mice of either sex.</p>	
<b>DNA BaZecoats (HeliX/Metallic/Profile/Camo)</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
<b>xylene</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >1700 mg/kg <sup>[2]</sup>	Eye (human): 200 ppm irritant
	Inhalation (rat) LC50: 5000 ppm/4h <sup>[2]</sup>	Eye (rabbit): 5 mg/24h SEVERE
	Oral (rat) LD50: 4300 mg/kg <sup>[2]</sup>	Eye (rabbit): 87 mg mild Skin (rabbit):500 mg/24h moderate
<b>n-butyl acetate</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >14080 mg/kg <sup>[1]</sup>	* [PPG]
	Inhalation (rat) LC50: 2000 ppm/4Hg <sup>[2]</sup>	Eye (human): 300 mg
	Inhalation (rat) LC50: 390 ppm/4h <sup>[2]</sup>	Eye (rabbit): 20 mg (open)-SEVERE
	Oral (rat) LD50: 10736 mg/kg <sup>[1]</sup>	Eye (rabbit): 20 mg/24h - moderate Skin (rabbit): 500 mg/24h-moderate
<b>propylene glycol monomethyl ether acetate, alpha-isomer</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup>	* [CCINFO]
	Inhalation (rat) LC50: 4345 ppm/6h <sup>[2]</sup>	Nil reported
	Oral (rat) LD50: >14.1 ml <sup>[1]</sup>	
<b>petroleum ether</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >1900 mg/kg <sup>[1]</sup>	Eye (human): 880 ppm/15m
	Inhalation (rat) LC50: 3400 ppm/4H <sup>[2]</sup>	
	Oral (rat) LD50: >4500 mg/kg <sup>[1]</sup>	

## DNA BaZecoats (HeliX/Metallic/Profile/Camo)

ethylbenzene	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: ca.15432.6 mg/kg <sup>[1]</sup>	Eye (rabbit): 500 mg - SEVERE
	Inhalation (mouse) LC50: 35.5 mg/L/2H <sup>[2]</sup>	Skin (rabbit): 15 mg/24h mild
	Inhalation (rat) LC50: 55 mg/L/2H <sup>[2]</sup>	
	Oral (rat) LD50: 3500 mg/kg <sup>[2]</sup>	
n-butanol	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 3434.4 mg/kg <sup>[1]</sup>	Not Available
	Inhalation (rat) LC50: 24 mg/L/4H <sup>[2]</sup>	
	Inhalation (rat) LC50: 8000 ppm/4hE <sup>[2]</sup>	
	Oral (rat) LD50: 2292.3 mg/kg <sup>[1]</sup>	
carbon black	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >3000 mg/kg <sup>[2]</sup>	Not Available
	Oral (rat) LD50: >8000 mg/kg <sup>[1]</sup>	
ethylene glycol monobutyl ether acetate	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 1500 mg/kg <sup>[2]</sup>	Eye (rabbit): 500 mg/24hr - mild
	Oral (rat) LD50: 7012.4 mg/kg <sup>[1]</sup>	Skin (rabbit): 500 mg - mild
2-ethoxyethyl acetate	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 10500 mg/kg <sup>[2]</sup>	Dermal (rabbit):420 mg(open)-mild
	Inhalation (rat) LC50: >1500 ppm/8H <sup>[2]</sup>	Eye (rabbit): 40 mg - moderate
	Inhalation (rat) LC50: 12.1 mg/L/8 hd <sup>[2]</sup>	
	Oral (rat) LD50: 2700 mg/kg <sup>[2]</sup>	
<b>Legend:</b>	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	

<b>XYLENE</b>	<p>The material may produce severe irritation to the eye causing pronounced 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 characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <p>The substance is classified by IARC as Group 3: <b>NOT</b> classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing. Reproductive effector in rats</p>
<b>N-BUTYL ACETATE</b>	<p>The material may produce severe irritation to the eye causing pronounced 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 characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p>
<b>PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER</b>	<p>for propylene glycol ethers (PGEs): Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM). Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids. Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects). This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product. Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono-, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body. As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces. As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from &gt;3,000 mg/kg (PnB) to &gt;5,000 mg/kg (DPMA). Dermal LD50s are all &gt; 2,000 mg/kg (PnB, &amp; DPnB; where no deaths occurred), and ranging up to &gt;15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m3 for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is &gt;2,040 mg/m3. For PnB, the 4-hour LC50 was &gt;651 ppm (&gt;3,412 mg/m3), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating</p>

## DNA BaZecoats (HeliX/Metallic/Profile/Camo)

None are skin sensitizers.

In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested).

Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m<sup>3</sup> (600 ppm) for PnB and 2,010 mg/m<sup>3</sup> (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m<sup>3</sup> (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m<sup>3</sup> (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members.

One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m<sup>3</sup>) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m<sup>3</sup>). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m<sup>3</sup>), with decreased body weights occurring at 3000 ppm (11058 mg/m<sup>3</sup>). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. In a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health.

In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity.

The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic *in vivo*. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice.

A BASF report (in ECETOC ) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects.

The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.]

A BASF report (in ECETOC ) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects. The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.] \*Shin-Etsu SDS

**for petroleum:**

This product contains benzene which is known to cause acute myeloid leukaemia and n-hexane which has been shown to metabolize to compounds which are neuropathic.

This product contains toluene. There are indications from animal studies that prolonged exposure to high concentrations of toluene may lead to hearing loss. This product contains ethyl benzene and naphthalene from which there is evidence of tumours in rodents

**Carcinogenicity:** Inhalation exposure to mice causes liver tumours, which are not considered relevant to humans. Inhalation exposure to rats causes kidney tumours which are not considered relevant to humans.

**Mutagenicity:** There is a large database of mutagenicity studies on gasoline and gasoline blending streams, which use a wide variety of endpoints and give predominantly negative results. All *in vivo* studies in animals and recent studies in exposed humans (e.g. petrol service station attendants) have shown negative results in mutagenicity assays.

**Reproductive Toxicity:** Repeated exposure of pregnant rats to high concentrations of toluene (around or exceeding 1000 ppm) can cause developmental effects, such as lower birth weight and developmental neurotoxicity, on the foetus. However, in a two-generation reproductive study in rats exposed to gasoline vapour condensate, no adverse effects on the foetus were observed.

**Human Effects:** Prolonged/ repeated contact may cause defatting of the skin which can lead to dermatitis and may make the skin more susceptible to irritation and penetration by other materials.

Lifetime exposure of rodents to gasoline produces carcinogenicity although the relevance to humans has been questioned. Gasoline induces kidney cancer in male rats as a consequence of accumulation of the alpha2-microglobulin protein in hyaline droplets in the male (but not female) rat kidney. Such abnormal accumulation represents lysosomal overload and leads to chronic renal tubular cell degeneration, accumulation of cell debris, mineralisation of renal medullary tubules and necrosis. A sustained regenerative proliferation occurs in epithelial cells with subsequent neoplastic transformation with continued exposure. The alpha2-microglobulin is produced under the influence of hormonal controls in male rats but not in females and, more importantly, not in humans.

**PETROLEUM ETHER**

The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

Ethylbenzene is readily absorbed following inhalation, oral, and dermal exposures, distributed throughout the body, and excreted primarily through urine. There are two different metabolic pathways for ethylbenzene with the primary pathway being the alpha-oxidation of ethylbenzene to 1-phenylethanol, mostly as the R-enantiomer. The pattern of urinary metabolite excretion varies with different mammalian species. In humans, ethylbenzene is excreted in the urine as mandelic acid and phenylglyoxylic acids; whereas rats and rabbits excrete hippuric acid and phenacetic acid as the main metabolites. Ethylbenzene can induce liver enzymes and hence its own metabolism as well as the metabolism of other substances.

Ethylbenzene has a low order of acute toxicity by the oral, dermal or inhalation routes of exposure. Studies in rabbits indicate that ethylbenzene is irritating to the skin and eyes. There are numerous repeat dose studies available in a variety of species, these include: rats, mice, rabbits, guinea pig and rhesus monkeys.

Hearing loss has been reported in rats (but not guinea pigs) exposed to relatively high exposures (400 ppm and greater) of ethylbenzene

In chronic toxicity/carcinogenicity studies, both rats and mice were exposed via inhalation to 0, 75, 250 or 750 ppm for 104 weeks. In rats, the kidney was the target organ of toxicity, with renal tubular hyperplasia noted in both males and females at the 750 ppm level only. In mice, the liver and lung were the principal target organs of toxicity. In male mice at 750 ppm, lung toxicity was described as alveolar epithelial metaplasia, and liver toxicity was described as hepatocellular syncytial alteration, hypertrophy and mild necrosis; this was accompanied by increased follicular cell hyperplasia in the thyroid. As a result the NOAEL in male mice was determined to be 250 ppm. In female mice, the 750 ppm dose group had an increased incidence of eosinophilic foci in the liver (44% vs 10% in the controls) and an increased incidence in follicular cell hyperplasia in the thyroid gland.

In studies conducted by the U.S. National Toxicology Program, inhalation of ethylbenzene at 750 ppm resulted in increased lung tumors in male mice, liver tumors in female mice, and increased kidney tumors in male and female rats. No increase in tumors was reported at 75 or 250 ppm. Ethylbenzene is considered to be an animal carcinogen, however, the relevance of these findings to humans is currently unknown. Although no reproductive toxicity studies have been conducted on ethylbenzene, repeated-dose studies indicate that the reproductive organs are not a target for ethylbenzene toxicity

Ethylbenzene was negative in bacterial gene mutation tests and in a yeast assay on mitotic recombination.

**NOTE:** Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.

**WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.

Liver changes, uterine tract, effects on fertility, foetotoxicity, specific developmental abnormalities (musculoskeletal system) recorded.

**ETHYLBENZENE****N-BUTANOL**

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis

## DNA BaZecoats (HeliX/Metallic/Profile/Camo)

of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

for n-butanol

**Acute toxicity:** n-Butanol (BA) was only slightly toxic to experimental animals following acute oral, dermal, or inhalation exposure. The acute oral LD50 values for female rats ranged from 790 to 4360 mg/kg. Different strains of rat were used in each of four studies, which may account for the variability. Oral LD50 values for mice, rabbits, hamsters, dogs, and male rats all fell within the same range. The rat inhalation LC0 of 8000 ppm (24000 mg/m3) indicates very low inhalation toxicity (no lethality at 8000 ppm). The rabbit dermal LD50 was 3402 mg/kg, indicating that BA can penetrate the skin, but not very readily. Animal experiments and human experience indicate that BA is, at most, moderately irritating to the skin, but it is a severe eye irritant. These effects are most likely due to BA's localised defatting and drying characteristics. Although no animal data are available, human studies and experience show that BA is not likely to be a skin sensitiser.

The median odor threshold for BA (0.17 ppm) is well below the lowest nasal irritation threshold in humans (289 ppm), allowing warning of possible chemical exposure prior to nasal irritation occurring. Human studies are complicated by the odor characteristics of the material, as the odor threshold is well below the levels at which irritation is observed.

**Repeat dose toxicity:** An in vivo toxicokinetics study confirmed the rapid metabolism of n-butyl acetate (BAC) to BA. Hydrolysis of BAC in blood and brain was estimated to be 99 percent complete within

2.7 minutes (elimination t1/2 = 0.41 minute). Thus, organisms exposed to BAC can experience appreciable tissue concentrations of BA. In this way, the results of toxicity studies with BAC can be used as supplemental, surrogate data to provide information on the toxicity of BA.

A thirteen-week, subchronic exposure to BAC, the metabolic precursor of BA, produced transient hypoactivity (during exposure only) at 1500 and 3000 ppm (7185 and 14370 mg/m3) along with decreased body weight and food consumption, but no post exposure neurotoxicity even at 3000 ppm. A concurrent subchronic neurotoxicity study under the same exposure conditions showed no evidence of cumulative neurotoxicity based upon functional observational battery endpoints, quantitative motor activity, neuropathology and scheduled-controlled operant behavior endpoints. A no observable effect level (NOAEL) of 500 ppm (2395 mg/m3) was reported for systemic effects in rats, and a NOAEL of 3000 ppm (14370 mg/m3) was reported for post exposure neurotoxicity in rats.

**Reproductive toxicity:** Several studies indicate that BA is not a reproductive toxicant.

Female rats exposed to 6000 ppm (18000 mg/m3) BA throughout gestation and male rats exposed to 6000 ppm (18000 mg/m3) BA for six weeks prior to mating showed no effects on fertility or pregnancy rate. Male rats given BA at 533 mg/kg/day for 5 days had no testicular toxicity.

**Developmental toxicity:** BA produced only mild foetotoxicity and developmental alterations at or near the maternally toxic (even lethal) dose of 8000 ppm (24000 mg/m3) throughout gestation.

**Genotoxicity:** An entire battery of negative in vitro tests and a negative in vivo micronucleus test indicate that BA is not genotoxic.

**Carcinogenicity:** Based upon the battery of negative mutagenicity and clastogenicity findings, BA presents a very small potential for carcinogenicity.

## CARBON BLACK

No significant acute toxicological data identified in literature search.

**WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.  
Inhalation (rat) TCLo: 50 mg/m3/6h/90D-I Nil reported

ETHYLENE GLYCOL  
MONOBUTYL ETHER  
ACETATE

The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

For ethylene glycol:

Ethylene glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol.

dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO<sub>2</sub>, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO<sub>2</sub>, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested.

**Respiratory Effects.** Respiratory system involvement occurs 12-24 hours after ingestion of sufficient amounts of ethylene glycol and is considered to be part of a second stage in ethylene glycol poisoning. The symptoms include hyperventilation, shallow rapid breathing, and generalized pulmonary edema with calcium oxalate crystals occasionally present in the lung parenchyma. Respiratory system involvement appears to be dose-dependent and occurs concomitantly with cardiovascular changes. Pulmonary infiltrates and other changes compatible with adult respiratory distress syndrome (ARDS) may characterise the second stage of ethylene glycol poisoning. Pulmonary oedema can be secondary to cardiac failure, ARDS, or aspiration of gastric contents. Symptoms related to acidosis such as hyperpnea and tachypnea are frequently observed; however, major respiratory morbidities such as pulmonary edema and bronchopneumonia are relatively rare and usually only observed with extreme poisoning (e.g., in only 5 of 36 severely poisoned cases).

**Cardiovascular Effects.** Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12-24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of ethylene glycol.

Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases. Therefore, it appears that acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown.

**Gastrointestinal Effects.** Nausea, vomiting with or without blood, pyrosis, and abdominal cramping and pain are common early effects of acute ethylene glycol ingestion. Acute effects of ethylene glycol ingestion in one patient included intermittent diarrhea and abdominal pain, which were attributed to mild colonic ischaemia; severe abdominal pain secondary to colonic stricture and perforation developed 3 months after ingestion, and histology of the resected colon showed birefringent crystals highly suggestive of oxalate deposition.

**Musculoskeletal Effects.** Reported musculoskeletal effects in cases of acute ethylene glycol poisoning have included diffuse muscle tenderness and myalgias associated with elevated serum creatinine phosphokinase levels, and myoclonic jerks and tetanic contractions associated with hypocalcaemia.

**Hepatic Effects.** Central hydropic or fatty degeneration, parenchymal necrosis, and calcium oxalate crystals in the liver have been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol.

**Renal Effects.** Adverse renal effects after ethylene glycol ingestion in humans can be observed during the third stage of ethylene glycol toxicity 24-72 hours after acute exposure. The hallmark of renal toxicity is the presence of birefringent calcium oxalate monohydrate crystals deposited in renal tubules and their presence in urine after ingestion of relatively high amounts of ethylene glycol. Other signs of nephrotoxicity can include tubular cell degeneration and necrosis and tubular interstitial inflammation. If untreated, the degree of renal damage caused by high doses of ethylene glycol progresses and leads to haematuria, proteinuria, decreased renal function, oliguria, anuria, and ultimately renal failure. These changes in the kidney are linked to acute tubular necrosis but normal or near normal renal function can return with adequate supportive therapy.

**Metabolic Effects.** One of the major adverse effects following acute oral exposure of humans to ethylene glycol involves metabolic changes. These changes occur as early as 12 hours after ethylene glycol exposure. Ethylene glycol intoxication is accompanied by metabolic acidosis which is manifested by decreased pH and bicarbonate content of serum and other bodily fluids caused by accumulation of excess glycolic acid. Other characteristic metabolic effects of ethylene

## DNA BaZecoats (HeliX/Metallic/Profile/Camo)

glycol poisoning are increased serum anion gap, increased osmolal gap, and hypocalcaemia. Serum anion gap is calculated from concentrations of sodium, chloride, and bicarbonate, is normally 12-16 mM, and is typically elevated after ethylene glycol ingestion due to increases in unmeasured metabolite anions (mainly glycolate).

**Neurological Effects:** Adverse neurological reactions are among the first symptoms to appear in humans after ethylene glycol ingestion. These early neurotoxic effects are also the only symptoms attributed to unmetabolised ethylene glycol. Together with metabolic changes, they occur during the period of 30 minutes to 12 hours after exposure and are considered to be part of the first stage in ethylene glycol intoxication. In cases of acute intoxication, in which a large amount of ethylene glycol is ingested over a very short time period, there is a progression of neurological manifestations which, if not treated, may lead to generalized seizures and coma. Ataxia, slurred speech, confusion, and somnolence are common during the initial phase of ethylene glycol intoxication as are irritation, restlessness, and disorientation. Cerebral edema and crystalline deposits of calcium oxalate in the walls of small blood vessels in the brain were found at autopsy in people who died after acute ethylene glycol ingestion.

Effects on cranial nerves appear late (generally 5-20 days post-ingestion), are relatively rare, and according to some investigators constitute a fourth, late cerebral phase in ethylene glycol intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar nerves and are reversible over many months.

**Reproductive Effects:** Reproductive function after intermediate-duration oral exposure to ethylene glycol has been tested in three multi-generation studies (one in rats and two in mice) and several shorter studies (15-20 days in rats and mice). In these studies, effects on fertility, foetal viability, and male reproductive organs were observed in mice, while the only effect in rats was an increase in gestational duration.

**Developmental Effects:** The developmental toxicity of ethylene glycol has been assessed in several acute-duration studies using mice, rats, and rabbits. Available studies indicate that malformations, especially skeletal malformations occur in both mice and rats exposed during gestation; mice are apparently more sensitive to the developmental effects of ethylene glycol. Other evidence of embryotoxicity in laboratory animals exposed to ethylene glycol exposure includes reduction in foetal body weight.

**Cancer:** No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol.

**Genotoxic Effects:** Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available *in vivo* and *in vitro* laboratory studies provide consistently negative genotoxicity results for ethylene glycol.

For ethylene glycol monoalkyl ethers and their acetates (EGMAEs):

Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates.

EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers.

**Acute Toxicity:** Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m<sup>3</sup>) for EGHE, LC50 > 400ppm (2620 mg/m<sup>3</sup>) for EGBEA to LC50 > 2132 ppm (9061 mg/m<sup>3</sup>) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitizers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE *in vitro* than those of rats.

**Repeat dose toxicity:** The fact that the NOAEL for repeated dose toxicity of EGBE is less than that of EGPE is consistent with red blood cells being more sensitive to EGBE than EGPE. Blood from mice, rats, hamsters, rabbits and baboons were sensitive to the effects of BAA *in vitro* and displayed similar responses, which included erythrocyte swelling (increased haematocrit and mean corpuscular hemoglobin), followed by hemolysis. Blood from humans, pigs, dogs, cats, and guinea pigs was less sensitive to haemolysis by BAA *in vitro*.

**Mutagenicity:** In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in *S. typhimurium* strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. *In vitro* cytogenetic and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and *in vivo* micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic.

**Carcinogenicity:** In a 2-year inhalation chronic toxicity and carcinogenicity study with EGBE in rats and mice a significant increase in the incidence of liver haemangiosarcomas was seen in male mice and forestomach tumours in female mice. It was decided that based on the mode of action data available, there was no significant hazard for human carcinogenicity.

**Reproductive and developmental toxicity.** The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes).

Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m<sup>3</sup> and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m<sup>3</sup>), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m<sup>3</sup>), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m<sup>3</sup>) indicate that the members of the category are not teratogenic.

The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m<sup>3</sup> (rabbit-EGPE), 100 ppm or 425 mg/m<sup>3</sup> (rat-EGPE), 50 ppm or 241 mg/m<sup>3</sup> (rat EGBE) and 100 ppm or 483 mg/m<sup>3</sup> (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m<sup>3</sup> (rat and rabbit-EGHE).

mae

## 2-ETHOXYETHYL ACETATE

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).

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 available but does not fill the criteria for classification  
✓ – Data required to make classification available  
⊖ – Data Not Available to make classification

## SECTION 12 ECOLOGICAL INFORMATION

## Toxicity

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
------------	----------	--------------------	---------	-------	--------

Continued...

## DNA BaZecoats (HeliX/Metallic/Profile/Camo)

xylene	LC50	96	Fish	0.0013404mg/L	4
xylene	EC50	48	Crustacea	>3.4mg/L	2
xylene	EC50	72	Algae or other aquatic plants	4.6mg/L	2
xylene	EC50	24	Crustacea	0.711mg/L	4
xylene	NOEC	73	Algae or other aquatic plants	0.44mg/L	2
n-butyl acetate	LC50	96	Fish	18mg/L	2
n-butyl acetate	EC50	48	Crustacea	=32mg/L	1
n-butyl acetate	EC50	96	Algae or other aquatic plants	1.675mg/L	3
n-butyl acetate	EC50	96	Fish	18mg/L	2
n-butyl acetate	NOEC	504	Crustacea	23mg/L	2
propylene glycol monomethyl ether acetate, alpha-isomer	LC50	96	Fish	100mg/L	1
propylene glycol monomethyl ether acetate, alpha-isomer	EC50	48	Crustacea	373mg/L	2
propylene glycol monomethyl ether acetate, alpha-isomer	EC50	96	Algae or other aquatic plants	9.337mg/L	3
propylene glycol monomethyl ether acetate, alpha-isomer	EC50	504	Crustacea	>100mg/L	2
propylene glycol monomethyl ether acetate, alpha-isomer	NOEC	336	Fish	47.5mg/L	2
petroleum ether	EC50	72	Algae or other aquatic plants	=6.5mg/L	1
petroleum ether	EC50	72	Algae or other aquatic plants	=6.5mg/L	1
petroleum ether	NOEC	72	Algae or other aquatic plants	<0.1mg/L	1
ethylbenzene	LC50	96	Fish	0.0043mg/L	4
ethylbenzene	EC50	48	Crustacea	0.0021234mg/L	4
ethylbenzene	EC50	96	Algae or other aquatic plants	3.6mg/L	4
ethylbenzene	EC50	3	Algae or other aquatic plants	0.0509616mg/L	4
ethylbenzene	NOEC	168	Crustacea	0.96mg/L	2
n-butanol	LC50	96	Fish	88.462mg/L	3
n-butanol	EC50	48	Crustacea	>500mg/L	1
n-butanol	EC50	96	Algae or other aquatic plants	225mg/L	2
n-butanol	BCF	24	Fish	921mg/L	4
n-butanol	EC50	504	Crustacea	18mg/L	2
n-butanol	NOEC	504	Crustacea	4.1mg/L	2
carbon black	LC50	96	Fish	>100mg/L	2
carbon black	EC50	48	Crustacea	>100mg/L	2
carbon black	EC50	96	Algae or other aquatic plants	95mg/L	2
carbon black	EC50	384	Crustacea	4.9mg/L	2
carbon black	NOEC	720	Fish	17mg/L	2
ethylene glycol monobutyl ether acetate	LC50	96	Fish	>20- <40mg/L	2
ethylene glycol monobutyl ether acetate	EC50	48	Crustacea	=37mg/L	1
ethylene glycol monobutyl ether acetate	EC50	96	Algae or other aquatic plants	3.228mg/L	3
ethylene glycol monobutyl ether acetate	EC0	48	Crustacea	=10mg/L	1
2-ethoxyethyl acetate	LC50	96	Fish	40mg/L	4
2-ethoxyethyl acetate	EC50	96	Algae or other aquatic plants	8.588mg/L	3
2-ethoxyethyl acetate	EC50	96	Fish	41mg/L	5
2-ethoxyethyl acetate	NOEC	504	Crustacea	=30mg/L	1

**Legend:**

Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data

Drinking Water Standards: hydrocarbon total: 10 ug/l (UK max.).

**DO NOT discharge into sewer or waterways.**

**Persistence and degradability**

Ingredient	Persistence: Water/Soil	Persistence: Air
------------	-------------------------	------------------

Continued...

## DNA BaZecoats (HeliX/Metallic/Profile/Camo)

xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
n-butyl acetate	LOW	LOW
propylene glycol monomethyl ether acetate, alpha-isomer	LOW	LOW
ethylbenzene	HIGH (Half-life = 228 days)	LOW (Half-life = 3.57 days)
n-butanol	LOW (Half-life = 54 days)	LOW (Half-life = 3.65 days)
ethylene glycol monobutyl ether acetate	LOW	LOW
2-ethoxyethyl acetate	LOW	LOW

## Bioaccumulative potential

Ingredient	Bioaccumulation
xylene	MEDIUM (BCF = 740)
n-butyl acetate	LOW (BCF = 14)
propylene glycol monomethyl ether acetate, alpha-isomer	LOW (LogKOW = 0.56)
ethylbenzene	LOW (BCF = 79.43)
n-butanol	LOW (BCF = 64)
ethylene glycol monobutyl ether acetate	LOW (BCF = 3.2)
2-ethoxyethyl acetate	LOW (LogKOW = 0.5898)

## Mobility in soil

Ingredient	Mobility
n-butyl acetate	LOW (KOC = 20.86)
propylene glycol monomethyl ether acetate, alpha-isomer	HIGH (KOC = 1.838)
ethylbenzene	LOW (KOC = 517.8)
n-butanol	MEDIUM (KOC = 2.443)
ethylene glycol monobutyl ether acetate	LOW (KOC = 10)
2-ethoxyethyl acetate	MEDIUM (KOC = 2.093)

## SECTION 13 DISPOSAL CONSIDERATIONS

## Waste treatment methods

<b>Product / Packaging disposal</b>	<ul style="list-style-type: none"> <li>▶ Containers may still present a chemical hazard/ danger when empty.</li> <li>▶ Return to supplier for reuse/ recycling if possible.</li> </ul> <p>Otherwise:</p> <ul style="list-style-type: none"> <li>▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.</li> <li>▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product.</li> </ul>
-------------------------------------	---

## SECTION 14 TRANSPORT INFORMATION

## Labels Required

<b>Marine Pollutant</b>	NO
<b>HAZCHEM</b>	☣3YE



## Land transport (ADG)

<b>UN number</b>	1263				
<b>Packing group</b>	II				
<b>UN proper shipping name</b>	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)				
<b>Environmental hazard</b>	No relevant data				
<b>Transport hazard class(es)</b>	<table style="border: none;"> <tr> <td style="padding-right: 10px;">Class</td> <td style="border-left: 1px dashed black; padding-left: 5px;">3</td> </tr> <tr> <td style="padding-right: 10px;">Subrisk</td> <td style="border-left: 1px dashed black; padding-left: 5px;">Not Applicable</td> </tr> </table>	Class	3	Subrisk	Not Applicable
Class	3				
Subrisk	Not Applicable				

<b>Special precautions for user</b>	Special provisions	163 *
	Limited quantity	5 L

**Air transport (ICAO-IATA / DGR)**

<b>UN number</b>	1263	
<b>Packing group</b>	II	
<b>UN proper shipping name</b>	Paint (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base); Paint related material (including paint thinning or reducing compounds)	
<b>Environmental hazard</b>	No relevant data	
<b>Transport hazard class(es)</b>	ICAO/IATA Class	3
	ICAO / IATA Subrisk	Not Applicable
	ERG Code	3L
<b>Special precautions for user</b>	Special provisions	A3 A72 A192
	Cargo Only Packing Instructions	364
	Cargo Only Maximum Qty / Pack	60 L
	Passenger and Cargo Packing Instructions	353
	Passenger and Cargo Maximum Qty / Pack	5 L
	Passenger and Cargo Limited Quantity Packing Instructions	Y341
	Passenger and Cargo Limited Maximum Qty / Pack	1 L

**Sea transport (IMDG-Code / GGVSee)**

<b>UN number</b>	1263	
<b>Packing group</b>	II	
<b>UN proper shipping name</b>	PAINT (including paint, lacquer, enamel, stain, shellac solutions, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)	
<b>Environmental hazard</b>	Not Applicable	
<b>Transport hazard class(es)</b>	IMDG Class	3
	IMDG Subrisk	Not Applicable
<b>Special precautions for user</b>	EMS Number	F-E, S-E
	Special provisions	163 367
	Limited Quantities	5 L

**Transport in bulk according to Annex II of MARPOL 73 / 78 and the IBC code**

Source	Ingredient	Pollution Category
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	xylene	Y
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	n-butyl acetate	Y
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	propylene glycol monomethyl ether acetate, alpha-isomer	Z
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	ethylbenzene	Y
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	ethylene glycol monobutyl ether acetate	Y
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	2-ethoxyethyl acetate	Y

**SECTION 15 REGULATORY INFORMATION****Safety, health and environmental regulations / legislation specific for the substance or mixture****XYLENE(1330-20-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards  
Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)  
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

**N-BUTYL ACETATE(123-86-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

## DNA BaZecoats (HeliX/Metallic/Profile/Camo)

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
------------------------------	---

Australia Hazardous Substances Information System - Consolidated Lists

**PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER(108-65-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
------------------------------	---

Australia Hazardous Substances Information System - Consolidated Lists

**PETROLEUM ETHER(8032-32-4.) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

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

**ETHYLBENZENE(100-41-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
------------------------------	---

Australia Hazardous Substances Information System - Consolidated Lists

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

**N-BUTANOL(71-36-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
------------------------------	---

Australia Hazardous Substances Information System - Consolidated Lists

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

**CARBON BLACK(1333-86-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
------------------------------	---

Australia Hazardous Substances Information System - Consolidated Lists

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

**ETHYLENE GLYCOL MONOBUTYL ETHER ACETATE(112-07-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
------------------------------	---

Australia Hazardous Substances Information System - Consolidated Lists

**2-ETHOXYETHYL ACETATE(111-15-9) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
------------------------------	---

Australia Hazardous Substances Information System - Consolidated Lists

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (propylene glycol monomethyl ether acetate, alpha-isomer; n-butanol; xylene; n-butyl acetate; petroleum ether; ethylbenzene; ethylene glycol monobutyl ether acetate; carbon black; 2-ethoxyethyl acetate)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	N (petroleum ether)
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	Y
<b>Legend:</b>	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

**SECTION 16 OTHER INFORMATION****Other information****Ingredients with multiple cas numbers**

Name	CAS No
propylene glycol monomethyl ether acetate, alpha-isomer	108-65-6, 142300-82-1, 84540-57-8

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

A list of reference resources used to assist the committee may be found at: [www.chemwatch.net](http://www.chemwatch.net)

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

**Definitions and abbreviations**

PC – TWA: Permissible Concentration-Time Weighted Average  
 PC – STEL: Permissible Concentration-Short Term Exposure Limit  
 IARC: International Agency for Research on Cancer  
 ACGIH: American Conference of Governmental Industrial Hygienists  
 STEL: Short Term Exposure Limit  
 TEEL: Temporary Emergency Exposure Limit,  
 IDLH: Immediately Dangerous to Life or Health Concentrations  
 OSF: Odour Safety Factor  
 NOAEL :No Observed Adverse Effect Level  
 LOAEL: Lowest Observed Adverse Effect Level

Continued...

**DNA BaZecoats (HeliX/Metallic/Profile/Camo)**

TLV: Threshold Limit Value  
LOD: Limit Of Detection  
OTV: Odour Threshold Value  
BCF: BioConcentration Factors  
BEI: Biological Exposure Index

This document is copyright.

Apart from any fair dealing for the purposes of private study, research, review or criticism, as permitted under the Copyright Act, no part may be reproduced by any process without written permission from CHEMWATCH.

TEL (+61 3) 9572 4700.