

RocX Mine Mark Paint– Fluoro Colours

RocX Mine Mark Paint

Chemwatch: 47249
Version No: 4.1.1.1
Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 4

Issue Date: 27/06/2017
Print Date: 26/10/2017
L.GHS.AUS.EN

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

Product Identifier

Product name	RocX Mine Mark Paint Fluoro
Synonyms	spray paint
Proper shipping name	AEROSOLS
Other means of identification	Not Available

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

Relevant identified uses	Application is by spray atomisation from a hand held aerosol pack Use according to manufacturer's directions. Spray paint.
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Details of the supplier of the safety data sheet

Registered company name	Dynamics G-Ex Pty Ltd.
Address	37 Langton Road, GYMPIE QLD 4570
Telephone	+61 7 5482 6649
Fax	Not Available
Website	www.dynamicsgex.com.au
Email	customerservice@dynamicsgex.com.au

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	+61 7 5482 6649 (Mon-Fri, 7am-5pm)
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.

Poisons Schedule	Not Applicable
Classification ^[1]	Aerosols Category 1, Gas under Pressure (Compressed gas), Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Carcinogenicity Category 1A, Reproductive Toxicity Category 2, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Specific target organ toxicity - repeated exposure Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HSIS ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	
SIGNAL WORD	DANGER

Hazard statement(s)

H222	Extremely flammable aerosol.
H280	Contains gas under pressure; may explode if heated.
H302	Harmful if swallowed.
H315	Causes skin irritation.
H319	Causes serious eye irritation.
H350	May cause cancer.
H361	Suspected of damaging fertility or the unborn child.
H336	May cause drowsiness or dizziness.

Continued...

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H373	May cause damage to organs through prolonged or repeated exposure.
AUH044	Risk of explosion if heated under confinement

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat/sparks/open flames/hot surfaces. - No smoking.
P211	Do not spray on an open flame or other ignition source.
P251	Pressurized container: Do not pierce or burn, even after use.
P260	Do not breathe dust/fume/gas/mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P281	Use personal protective equipment as required.
P270	Do not eat, drink or smoke when using this product.
P280	Wear protective gloves/protective clothing/eye protection/face protection.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/attention.
P362	Take off contaminated clothing and wash before reuse.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P337+P313	If eye irritation persists: Get medical advice/attention.
P301+P312	IF SWALLOWED: Call a POISON CENTER or doctor/physician if you feel unwell.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.
P330	Rinse mouth.
P332+P313	If skin irritation occurs: Get medical advice/attention.

Precautionary statement(s) Storage

P405	Store locked up.
P410+P403	Protect from sunlight. Store in a well-ventilated place.
P410+P412	Protect from sunlight. Do not expose to temperatures exceeding 50 °C/122 °F.
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.
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SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
75-09-2	30-60	<u>methylen chloride</u>
108-88-3	10-30	<u>toluene</u>
1332-58-7	1-10	<u>kaolin</u>
Not Available	1-10	hydrocarbon resin (aromatic)
Not Available	1-10	fluorescent pigment
68476-85-7.	30-60	<u>hydrocarbon propellant</u>
		NOTE: Manufacturer has supplied full ingredient information to allow CHEMWATCH assessment.

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	<p>If aerosols come in contact with the eyes: Immediately hold the eyelids apart and flush the eye continuously for at least 15 minutes with fresh running water. Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.</p> <ul style="list-style-type: none"> ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If solids or aerosol mists are deposited upon the skin: Flush skin and hair with running water (and soap if available). Remove any adhering solids with industrial skin cleansing cream.</p> <ul style="list-style-type: none"> ▶ DO NOT use solvents. ▶ Seek medical attention in the event of irritation.

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Inhalation	<p>If aerosols, fumes or combustion products are inhaled: Remove to fresh air. Lay patient down. Keep warm and rested. Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. If breathing is shallow or has stopped, ensure clear airway and apply resuscitation, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. Transport to hospital, or doctor.</p>
Ingestion	<p>Avoid giving milk or oils. Avoid giving alcohol. Not considered a normal route of entry. For advice, contact a Poisons Information Centre or a doctor. If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.</p>

Indication of any immediate medical attention and special treatment needed

for intoxication due to Freons/ Halons;

A: Emergency and Supportive Measures

- ▶ Maintain an open airway and assist ventilation if necessary
- ▶ Treat coma and arrhythmias if they occur. Avoid (adrenaline) epinephrine or other sympathomimetic amines that may precipitate ventricular arrhythmias. Tachyarrhythmias caused by increased myocardial sensitisation may be treated with propranolol, 1-2 mg IV or esmolol 25-100 microgm/kg/min IV.
- ▶ Monitor the ECG for 4-6 hours

B: Specific drugs and antidotes:

- ▶ There is no specific antidote

C: Decontamination

- ▶ Inhalation; remove victim from exposure, and give supplemental oxygen if available.
- ▶ Ingestion; (a) Prehospital: Administer activated charcoal, if available. **DO NOT** induce vomiting because of rapid absorption and the risk of abrupt onset CNS depression. (b) Hospital: Administer activated charcoal, although the efficacy of charcoal is unknown. Perform gastric lavage only if the ingestion was very large and recent (less than 30 minutes)

D: Enhanced elimination:

- ▶ There is no documented efficacy for diuresis, haemodialysis, haemoperfusion, or repeat-dose charcoal.

POISONING and DRUG OVERDOSE, Californian Poison Control System Ed. Kent R Olson; 3rd Edition

- ▶ Do not administer sympathomimetic drugs unless absolutely necessary as material may increase myocardial irritability.
- ▶ No specific antidote.
- ▶ Because rapid absorption may occur through lungs if aspirated and cause systematic effects, the decision of whether to induce vomiting or not should be made by an attending physician.
- ▶ If lavage is performed, suggest endotracheal and/or esophageal control.
- ▶ Danger from lung aspiration must be weighed against toxicity when considering emptying the stomach.
- ▶ Treatment based on judgment of the physician in response to reactions of the patient

Treat symptomatically.

Following acute or short term repeated exposures to toluene:

- ▶ Toluene is absorbed across the alveolar barrier, the blood/air mixture being 11.2/15.6 (at 37 degrees C.) The concentration of toluene, in expired breath, is of the order of 18 ppm following sustained exposure to 100 ppm. The tissue/blood proportion is 1/3 except in adipose where the proportion is 8/10.
- ▶ Metabolism by microsomal mono-oxygenation, results in the production of hippuric acid. This may be detected in the urine in amounts between 0.5 and 2.5 g/24 hr which represents, on average 0.8 gm/gm of creatinine. The biological half-life of hippuric acid is in the order of 1-2 hours.
- ▶ Primary threat to life from ingestion and/or inhalation is respiratory failure.
- ▶ Patients should be quickly evaluated for signs of respiratory distress (eg cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO2 <50 mm Hg or pCO2 > 50 mm Hg) should be intubated.
- ▶ Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial damage 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 (adrenaline) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.
- ▶ Lavage is indicated in patients who require decontamination; ensure use.

BIOLOGICAL EXPOSURE INDEX - BEI

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

Determinant	Index	Sampling Time	Comments
o-Cresol in urine	0.5 mg/L	End of shift	B
Hippuric acid in urine	1.6 g/g creatinine	End of shift	B, NS
Toluene in blood	0.05 mg/L	Prior to last shift of workweek	

NS: Non-specific determinant; also observed after exposure to other material

B: Background levels occur in specimens collected from subjects NOT exposed

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

SMALL FIRE:

- ▶ Water spray, dry chemical or CO2

LARGE FIRE:

- ▶ Water spray or fog.

Special hazards arising from the substrate or mixture

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

Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ May be violently or explosively reactive. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ If safe, switch off electrical equipment until vapour fire hazard removed. ▶ Use water delivered as a fine spray to control fire and cool adjacent area. ▶ DO NOT approach containers suspected to be hot.
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	<ul style="list-style-type: none"> ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire. ▶ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ Liquid and vapour are highly flammable. ▶ Severe fire hazard when exposed to heat or flame. ▶ Vapour forms an explosive mixture with air. ▶ Severe explosion hazard, in the form of vapour, when exposed to flame or spark. ▶ Vapour may travel a considerable distance to source of ignition. ▶ Heating may cause expansion or decomposition with violent container rupture. ▶ Aerosol cans may explode on exposure to naked flames. ▶ Rupturing containers may rocket and scatter burning materials. ▶ Hazards may not be restricted to pressure effects. ▶ May emit acrid, poisonous or corrosive fumes. ▶ On combustion, may emit toxic fumes of carbon monoxide (CO). <p>Combustion products include:</p> <ul style="list-style-type: none"> , carbon monoxide (CO) , carbon dioxide (CO2) , hydrogen chloride , phosgene , other pyrolysis products typical of burning organic material. <p>Contains low boiling substance: Closed containers may rupture due to pressure buildup under fire conditions.</p>
HAZCHEM	2Y

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	<p>Clean up all spills immediately. Avoid breathing vapours and contact with skin and eyes. Wear protective clothing, impervious gloves and safety glasses. Shut off all possible sources of ignition and increase ventilation. Wipe up. If safe, damaged cans should be placed in a container outdoors, away from all ignition sources, until pressure has dissipated. Undamaged cans should be gathered and stowed safely.</p>
Major Spills	<p>DO NOT exert excessive pressure on valve; DO NOT attempt to operate damaged valve. Clear area of personnel and move upwind. Alert Fire Brigade and tell them location and nature of hazard. May be violently or explosively reactive. Wear breathing apparatus plus protective gloves. Prevent, by any means available, spillage from entering drains or water courses No smoking, naked lights or ignition sources. Increase ventilation. Stop leak if safe to do so. Water spray or fog may be used to disperse / absorb vapour. Absorb or cover spill with sand, earth, inert materials or vermiculite. If safe, damaged cans should be placed in a container outdoors, away from ignition sources, until pressure has dissipated. Undamaged cans should be gathered and stowed safely. Collect residues and seal in labelled drums for disposal. Remove leaking cylinders to a safe place if possible. Release pressure under safe, controlled conditions by opening the valve.</p>

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ Avoid smoking, naked lights or ignition sources. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ DO NOT incinerate or puncture aerosol cans. ▶ DO NOT spray directly on humans, exposed food or food utensils. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.
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	Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	<p>Store below 38 deg. C. Keep dry to avoid corrosion of cans. Corrosion may result in container perforation and internal pressure may eject contents of can Store in original containers in approved flammable liquid storage area. DO NOT store in pits, depressions, basements or areas where vapours may be trapped. No smoking, naked lights, heat or ignition sources. Keep containers securely sealed. Contents under pressure. Store away from incompatible materials. Store in a cool, dry, well ventilated area. Avoid storage at temperatures higher than 40 deg C. Store in an upright position. Protect containers against physical damage. Check regularly for spills and leaks. Observe manufacturer's storage and handling recommendations contained within this SDS.</p>

Conditions for safe storage, including any incompatibilities

Suitable container	<p>DO NOT use aluminium or galvanised containers Aerosol dispenser. Check that containers are clearly labelled.</p>
Storage incompatibility	<p>Methylene chloride is a combustible liquid under certain circumstances even though there is no measurable flash point and it is difficult to ignite its is flammable in ambient air in the range 12-23%; increased oxygen content can greatly enhance fire and explosion potential contact with hot surfaces and elevated temperatures can form fumes of hydrogen chloride and phosgene reacts violently with active metals, aluminium, lithium, methanol., peroxydisulfuryl difluoride, potassium, potassium tert-butoxide, sodium forms explosive mixtures with nitric acid is incompatible with strong oxidisers, strong caustics, alkaline earths and alkali metals attacks some plastics, coatings and rubber may generate electrostatic charge due to low conductivity Avoid reaction with oxidising agents</p>

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	methylene chloride	Methylene chloride	174 mg/m3 / 50 ppm	Not Available	Not Available	Not Available
Australia Exposure Standards	toluene	Toluene	191 mg/m3 / 50 ppm	574 mg/m3 / 150 ppm	Not Available	Not Available
Australia Exposure Standards	kaolin	Kaolin	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	hydrocarbon propellant	LPG (liquefied petroleum gas)	1800 mg/m3 / 1000 ppm	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
methylene chloride	Methylene chloride; (Dichloromethane)	Not Available	Not Available	Not Available
toluene	Toluene	Not Available	Not Available	Not Available
hydrocarbon propellant	Liquefied petroleum gas; (L.P.G.)	65,000 ppm	2.30E+05 ppm	4.00E+05 ppm

Ingredient	Original IDLH	Revised IDLH
methylene chloride	2,000 ppm	Not Available
toluene	500 ppm	Not Available
kaolin	Not Available	Not Available
hydrocarbon resin (aromatic)	Not Available	Not Available
fluorescent pink pigment	Not Available	Not Available
pink pigment	Not Available	Not Available
hydrocarbon propellant	2,000 [LEL] ppm	Not Available

MATERIAL DATA

For kaolin:

Kaolin dust appears to have fibrogenic potential even in the absence of crystalline silica. Kaolinosis can exist as simple and complicated forms with the latter often associated with respiratory symptoms. Crystalline silica enhances the severity of the pneumoconiosis.

For methylene chloride

Odour Threshold Value: 158 ppm (detection), 227 ppm (recognition)

NOTE: Detector tubes for methylene chloride, measuring in excess of 25 ppm are commercially available. Long-term measurements (4 hrs) may be conducted to detect concentrations exceeding 13 ppm.

Exposure at or below the recommended TLV-TWA (and in the absence of occupational exposure to carbon monoxide) is thought to minimise the potential for liver injury and to provide protection against the possible weak carcinogenic effects which have been demonstrated in laboratory rats and mice. Enhancement of tumours of the lung, liver, salivary glands and mammary tissue in rodent studies has lead NIOSH to recommend a more conservative outcome. The ACGIH however concludes that in the absence of documentation of health-related injuries at higher exposures after a long history of methylene chloride use and a number of epidemiologic studies, the recommended TLV-TWA provides an adequate margin of safety.

Concentration effects:

Concentration	Clinical effects
>300 ppm	Sweet odour
500-1000 ppm (1-2 h)	Unpleasant odour, slight anaesthetic effects, headache, light-headedness, eye irritation and elevated COHb concentration
2300 ppm (5 min.)	Odour strong, intensely irritating; dizziness
7200 ppm (8-16 min)	Paraesthesia, tachycardia
>50000 ppm	Immediately life-threatening

Animals exposed by inhalation to 10 mg/m³ titanium dioxide show no significant fibrosis, possibly reversible tissue reaction. The architecture of lung air spaces remains intact.

For toluene:

Odour Threshold Value: 0.16-6.7 (detection), 1.9-69 (recognition)

NOTE: Detector tubes measuring in excess of 5 ppm, are available.

High concentrations of toluene in the air produce depression of the central nervous system (CNS) in humans. Intentional toluene exposure (glue-sniffing) at maternally-intoxicating concentration has also produced birth defects. Foetotoxicity appears at levels associated with CNS narcosis and probably occurs only in those with chronic toluene-induced kidney failure. Exposure at or below the recommended TLV-TWA is thought to prevent transient headache and irritation, to provide a measure of safety for possible disturbances to human reproduction, the prevention of reductions in cognitive responses reported amongst humans inhaling greater than 40 ppm, and the significant risks of hepatotoxic, behavioural and nervous system effects (including impaired reaction time and incoordination). Although toluene/ethanol interactions are well recognised, the degree of protection afforded by the TLV-TWA among drinkers is not known.

Odour Safety Factor(OSF)

OSF=17 (TOLUENE)

NOTE K: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.1%/w/w 1,3-butadiene (EINECS No 203-450-8). - 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

<p>Appropriate engineering controls</p>	<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>General exhaust is adequate under normal conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection.</p> <p>Provide adequate ventilation in warehouse or closed storage areas.</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" data-bbox="389 920 1485 1016"> <thead> <tr> <th>Type of Contaminant:</th> <th>Speed:</th> </tr> </thead> <tbody> <tr> <td>aerosols, (released at low velocity into zone of active generation)</td> <td>0.5-1 m/s</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, 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> <p>Within each range the appropriate value depends on:</p> <table border="1" data-bbox="389 1077 1485 1240"> <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> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>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.</p>	Type of Contaminant:	Speed:	aerosols, (released at low velocity into zone of active generation)	0.5-1 m/s	direct spray, spray painting in shallow booths, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)	Lower end of the range	Upper end of the range	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity	3: Intermittent, low production.	3: High production, heavy use	4: Large hood or large air mass in motion	4: Small hood-local control only
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<p>Personal protection</p>																	
<p>Eye and face protection</p>	<p>Safety glasses with side shields.</p> <ul style="list-style-type: none"> ▶ Chemical goggles. ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent] 																
<p>Skin protection</p>	<p>See Hand protection below</p>																
<p>Hands/feet protection</p>	<ul style="list-style-type: none"> ▶ No special equipment needed when handling small quantities. ▶ OTHERWISE: ▶ For potentially moderate exposures: ▶ Wear general protective gloves, eg. light weight rubber gloves. ▶ For potentially heavy exposures: ▶ Wear chemical protective gloves, eg. PVC. and safety footwear. 																
<p>Body protection</p>	<p>See Other protection below</p>																
<p>Other protection</p>	<p>No special equipment needed when handling small quantities.</p> <p>OTHERWISE:</p> <ul style="list-style-type: none"> ▶ Overalls. ▶ Skin cleansing cream. ▶ Eyewash unit. ▶ Do not spray on hot surfaces. ▶ The clothing worn by process operators insulated from earth may develop static charges far higher (up to 100 times) than the minimum ignition energies for various flammable gas-air mixtures. This holds true for a wide range of clothing materials including cotton. ▶ Avoid dangerous levels of charge by ensuring a low resistivity of the surface material worn outermost. 																

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	BREThERICK: Handbook of Reactive Chemical Hazards.
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:

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Material	CPI
BUTYL	C
CPE	C
NATURAL RUBBER	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVA	C
PVC	C
SARANEX-23	C
SARANEX-23 2-PLY	C
TEFLON	C
VITON	C
VITON/BUTYL	C
VITON/CHLOROBUTYL	C
VITON/NEOPRENE	C

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

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 10 x ES	AX-AUS	-	AX-PAPR-AUS / Class 1
up to 50 x ES	-	AX-AUS / Class 1	-
up to 100 x ES	-	AX-2	AX-PAPR-2 ^

^ - Full-face

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

Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content. The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Supplied as an aerosol pack. Contents under PRESSURE . Contains highly flammable hydrocarbon propellant. Pink liquid with solvent odour; does not mix with water.		
Physical state	Liquid	Relative density (Water = 1)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Applicable	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	-81 propellant	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	HIGHLY FLAMMABLE.	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water (g/L)	Immiscible	pH as a solution (1%)	Not Applicable
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
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Chemical stability	Elevated temperatures. Presence of open flame. Product is considered stable. Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

	<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>Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual. Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>The acute toxicity of inhaled alkylbenzenes is best described by central nervous system depression. As a rule, these compounds may also act as general anaesthetics.</p> <p>Systemic poisoning produced by general anaesthesia is characterised by lightheadedness, nervousness, apprehension, euphoria, confusion, dizziness, drowsiness, tinnitus, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness and respiratory depression and arrest. Cardiac arrest may result from cardiovascular collapse. Bradycardia, and hypotension may also be produced.</p> <p>Inhaled alkylbenzene vapours cause death in animals at air levels that are relatively similar (typically LC50s are in the range 5000 -8000 ppm for 4 to 8 hour exposures). It is likely that acute inhalation exposure to alkylbenzenes resembles that to general anaesthetics.</p> <p>Alkylbenzenes are not generally toxic other than at high levels of exposure. This may be because their metabolites have a low order of toxicity and are easily excreted. There is little or no evidence to suggest that metabolic pathways can become saturated leading to spillover to alternate pathways. Nor is there evidence that toxic reactive intermediates, which may produce subsequent toxic or mutagenic effects, are formed</p> <p>Common, generalised symptoms associated with toxic gas inhalation include:</p> <ul style="list-style-type: none"> central nervous system effects such as depression, headache, confusion, dizziness, progressive stupor, coma and seizures; respiratory system complications may include acute pulmonary oedema, dyspnoea, stridor, tachypnoea, bronchospasm, wheezing and other reactive airway symptoms, and respiratory arrest; cardiovascular effects may include cardiovascular collapse, arrhythmias and cardiac arrest; gastrointestinal effects may also be present and may include mucous membrane irritation, nausea and vomiting (sometimes bloody), and abdominal pain. <p>Inhalation hazard is increased at higher temperatures.</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>Inhaled</p> <p>Material is highly volatile and may quickly form a concentrated atmosphere in confined or unventilated areas. The vapour may displace and replace air in breathing zone, acting as a simple asphyxiant. This may happen with little warning of overexposure.</p> <p>Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination</p> <p>WARNING: Intentional misuse by concentrating/inhaling contents may be lethal.</p> <p>High inhaled concentrations of mixed hydrocarbons may produce narcosis characterised by nausea, vomiting and lightheadedness. Inhalation of aerosols may produce severe pulmonary oedema, pneumonitis and pulmonary haemorrhage. Inhalation of petroleum hydrocarbons consisting substantially of low molecular weight species (typically C2-C12) may produce irritation of mucous membranes, incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and anaesthetic stupor. Massive exposures may produce central nervous system depression with sudden collapse and deep coma; fatalities have been recorded. Irritation of the brain and/or apnoeic anoxia may produce convulsions. Although recovery following overexposure is generally complete, cerebral micro-haemorrhage of focal post-inflammatory scarring may produce epileptiform seizures some months after the exposure. Pulmonary episodes may include chemical pneumonitis with oedema and haemorrhage. The lighter hydrocarbons may produce kidney and neurotoxic effects. Pulmonary irritancy increases with carbon chain length for paraffins and olefins. Alkenes produce pulmonary oedema at high concentrations. Liquid paraffins may produce anaesthesia and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce polyneuropathy. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue and vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons are cardiac sensitisers and may cause ventricular fibrillations.</p> <p>Acute intoxication by halogenated aliphatic hydrocarbons appears to take place over two stages. Signs of a reversible narcosis are evident in the first stage and in the second stage signs of injury to organs may become evident, a single organ alone is (almost) never involved.</p> <p>Inhalation exposure may cause susceptible individuals to show change in heart beat rhythm i.e. cardiac arrhythmia. Exposures must be terminated. Hydrocarbons may sensitise the heart to adrenalin and other circulatory catecholamines; as a result cardiac arrhythmias and ventricular fibrillation may occur. Abrupt collapse may produce traumatic injury. Central nervous system (CNS) depression may be evident early. Symptoms of moderate poisoning may include giddiness, headache, dizziness and nausea. Serious poisonings may result in respiratory depression and may be fatal.</p> <p>The paraffin gases C1-4 are practically non-toxic below their lower flammability limits (18000-50000 ppm). Above this level, incidental effects include CNS depression and irritation but these are reversible upon cessation of the exposure. The C3 and iso-C5 hydrocarbons show increasing narcotic properties; branching of the chain also enhances the effect. The C4 hydrocarbons appear to be more highly neurotoxic than the C3 and C5 members. Several fatalities due to voluntary inhalation of butane have been reported, possibly due to central, respiratory and circulatory effects resulting from anaesthesia, laryngeal oedema, chemical pneumonia or the combined effects of cardiac toxicity and increased sympathomimetic effects.</p> <p>Inhalation of petroleum gases may produce narcosis, due in part to olefinic impurities. Displacement of oxygen in the air may cyanosis. If present in sufficient quantity these gases may reduce the oxygen level to below 18% producing asphyxiation. Symptoms include rapid respiration, mental dullness, lack of coordination, poor judgement, nausea and vomiting. The onset of cyanosis may lead to unconsciousness and death.</p>
Ingestion	<p>Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.</p> <p>Not normally a hazard due to physical form of product.</p> <p>Considered an unlikely route of entry in commercial/industrial environments</p> <p>Considered an unlikely route of entry in commercial/industrial environments. The liquid may produce gastrointestinal discomfort and may be harmful if swallowed. Ingestion may result in nausea, pain and vomiting. Vomit entering the lungs by aspiration may cause potentially lethal chemical pneumonitis</p>

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<p>Skin Contact</p>	<p>The material produces severe skin irritation; evidence exists, or practical experience predicts, that the material either:</p> <ul style="list-style-type: none"> ▶ produces severe inflammation of the skin in a substantial number of individuals following direct contact, and/or ▶ produces significant and severe inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. ▶ Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. <p>NOTE: Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration. Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. Spray mist may produce discomfort. Open cuts, abraded or irritated skin should not be exposed to this material. Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>
<p>Eye</p>	<p>Direct contact with the eye may not cause irritation because of the extreme volatility of the gas; however concentrated atmospheres may produce irritation after brief exposures..</p> <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>The liquid produces a high level of eye discomfort and is capable of causing pain and severe conjunctivitis. Corneal injury may develop, with possible permanent impairment of vision, if not promptly and adequately treated.</p>
<p>Chronic</p>	<p>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. Harmful: danger of serious damage to health by prolonged exposure through inhalation. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.</p> <p>Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>There is some evidence to provide a presumption that human exposure to the material may result in impaired fertility on the basis of: some evidence in animal studies of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects but which is not a secondary non-specific consequence of other toxic effects. Principal route of occupational exposure to the gas is by inhalation. The health hazards associated with bentonite, kaolin, and common clay, which are commercially important clay products, as well as the related phyllosilicate minerals montmorillonite, kaolinite, and illite, have an extensive literature. Fibrous clay minerals, such as sepiolite, attapulgite, and zeolites, have a separate literature. The biological effects of clay minerals are influenced by their mineral composition and particle size. The decreasing rank order of the potencies of quartz, kaolinite, and montmorillonite to produce lung damage is consistent with their known relative active surface areas and surface chemistry. Clays are chemically all described as aluminosilicates; these are further classified as bentonite, kaolin and common clays. Bentonite is a rock formed of highly colloidal and plastic clays composed mainly of montmorillonite, a clay mineral of the smectite group. Kaolin or china clay is a mixture of different minerals. Its main component is kaolinite; in addition, it frequently contains quartz, mica, feldspar, illite, and montmorillonite. The main components of common clay and shale are illite and chlorite. Illite is also a component of ball clays. Illite closely resembles micas, From the limited data available from studies on bentonite-exposed persons, retained montmorillonite appears to effect only mild nonspecific tissue changes, which are similar to those that have been described in the spectrum of changes of the "small airways mineral dust disease" (nodular peribronchiolar dust accumulations containing refractile material [montmorillonite] in association with limited interstitial fibrosis). In some of the studies, radiological abnormalities have also been reported. Long-term occupational exposures to bentonite dust may cause structural and functional damage to the lungs. However, available data are inadequate to conclusively establish a dose-response relationship or even a cause-and-effect relationship due to limited information on period and intensity of exposure and to confounding factors, such as exposure to silica and tobacco smoke. Long-term exposure to kaolin may lead to a relatively benign pneumoconiosis, in an exposure-related fashion, known as kaolinos. Deterioration of lung function has been observed only in cases with prominent radiological alterations. Based on data from china clay workers in the United Kingdom, it can be very roughly estimated that kaolin is at least an order of magnitude less potent than quartz.. Clearcut deterioration of respiratory function and related symptoms have been reported only in cases with prominent radiological findings. The composition of the clay - i.e., quantity and quality of minerals other than kaolinite — is an important determinant of the effects. Bentonite, kaolin, and other clays often contain quartz, and exposure to quartz is causally related to silicosis and lung cancer. Statistically significant increases in the incidence of or mortality from chronic bronchitis and pulmonary emphysema have been reported after exposure to quartz. The removal of clay particles from the lungs takes place by solubilisation in situ and by physical clearance. In humans, there was a rapid initial clearance of 8% and 40% of aluminosilicate particles that were, respectively, 1.9 and 6.1 um in aerodynamic diameter from the lung region over 6 days. Thereafter, 4% and 11% of the two particle sizes were removed following a half-time of 20 days, and the rest with half-times of 330 and 420 days. Ultrafine particles (<100 nm) have a high deposition in the nasal area; they can penetrate the alveolar/capillary barrier. Epidemiological studies have indicated an increase in morbidity and mortality associated with an increase in airborne particulate matter, particularly in the ultrafine size range. An important determinant of the toxicity of clays is the content of quartz. The presence of quartz in the clays studied hampers reliable independent estimation of the fibrogenicity of other components of clays. Single intratracheal injection into rodents of bentonite and montmorillonite with low content of quartz produced dose- and particle size-dependent cytotoxic effects, as well as transient local inflammation, the signs of which included oedema and, consequently, increased lung weight. After high doses of intratracheal kaolin (containing 8-65% quartz), fibrosis has been described in some studies, whereas at lower kaolin doses, no fibrosis has been observed in the few available studies. There are limited data on the effects of multiple exposures of experimental animals to montmorillonite or bentonite. Mice maintained on diets containing 10% or 25% bentonite but otherwise adequate to support normal growth displayed slightly reduced growth rates, whereas mice maintained on a similar diet with 50% bentonite showed minimal growth and developed fatty livers and eventually fibrosis of the liver and benign hepatomas. In vitro studies of the effects of bentonite on a variety of mammalian cell types usually indicated a high degree of cytotoxicity. Concentrations below 1.0 mg/ml of bentonite and montmorillonite particles less than 5 um in diameter caused membrane damage and even cell lysis, as well as functional changes in several types of cells. No adequate studies are available on the carcinogenicity of bentonite. In an inhalation study and in a study using intrapleural injection, kaolin did not induce</p>

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tumours in rats. No studies are available on the genotoxicity of clays.
Single, very limited studies did not demonstrate developmental toxicity in rats after oral exposure to bentonite or kaolin.
Chronic dust inhalation of kaolin, as experienced in mineral extraction, has caused kaolinosis with heavy lung marking, emphysema, and nodular pneumoconiosis.
Evidence of kaolinosis (pneumoconiosis) was found in 9% of 553 Cornish china clay workers who had been exposed to kaolin dust for periods exceeding 5 years, whereas no kaolinosis was observed in workers exposed for less than 5 years. Workers in more heavily exposed jobs of milling, bagging and loading showed a prevalence of kaolinosis rising from 6% in those within between 5 and 15 years exposure to 23% in those exposed for more than 15 years. Workers intermittently and less heavily exposed in the older, outdated drying plants required 25 years of massive exposure before reaching the highest prevalence of 17%. Massive fibrosis was seen in four workers, and six workers needed antituberculosis chemotherapy. Preventative measures instituted include preemployment chest examination and approaches to the problem of dust control.
Sheer, G.; Brit. Jnl. Ind. Med. 21, pp 218-225, 1964
Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/or memory loss, tremor in the fingers and tongue, vertigo, olfactory disorders, constriction of visual field, paraesthesias of the extremities, weight loss and anaemia and degenerative changes in the liver and kidney. Chronic exposure by petroleum workers, to the lighter hydrocarbons, has been associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness and paraesthesias), psychological and neurophysiological deficits, bone marrow toxicities (including hypoplasia possibly due to benzene) and hepatic and renal involvement. Chronic dermal exposure to petroleum hydrocarbons may result in defatting which produces localised dermatoses. Surface cracking and erosion may also increase susceptibility to infection by microorganisms. One epidemiological study of petroleum refinery workers has reported elevations in standard mortality ratios for skin cancer along with a dose-response relationship indicating an association between routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have been unable to confirm this finding.
Methylene chloride is stored in body fat and is metabolised to carbon monoxide which increases and sustains carboxyhaemoglobin levels in the blood, reducing its oxygen carrying capacity. Smokers with already high carboxyhaemoglobin levels may show increased effects of exposure.
Methylene chloride exposures cause liver and kidney damage in animals and this justifies consideration before exposing persons with a history of impaired liver function and/or renal disorders.
Chronic exposure may produce central nervous system damage including confusion, delusions, slurred speech, memory impairment, anxiety, focal seizures, encephalopathy and visual and auditory hallucinations. These effects are probably due to chronic carbon monoxide poisoning resulting from methylene chloride metabolism.
Two epidemiological studies of workers exposed to methylene chloride have been published. An excess in pancreatic tumours was noted in one study. Chronic exposure to methylene chloride (approximately 30-120 ppm TWA) did not appear to increase the risk of deaths arising from lung cancer or cardiovascular disease. A study from Zeneca's Central Toxicology Laboratory added further support to the claim that solvent methylene chloride is not a human carcinogen. This study supported a previous finding by the European Centre of Ecology and Toxicology (ECETOC) that methylene chloride induced-cancers, previously identified in mice, were a consequence of a unique metabolic pathway found only in mice.

RocX Mine Mark Paint Fluoro	TOXICITY	IRRITATION
	Not Available	Not Available
methylene chloride	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye(rabbit): 162 mg - moderate
	Inhalation (rat) LC50: 76 mg/l/4H ^[2]	Eye(rabbit): 500 mg/24hr - mild
	Oral (rat) LD50: 985 mg/kg ^[2]	Skin (rabbit): 100mg/24hr-moderate Skin (rabbit): 810 mg/24hr-SEVERE
toluene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 12124 mg/kg ^[2]	Eye (rabbit): 2mg/24h - SEVERE
	Inhalation (rat) LC50: 49 mg/l/4H ^[2]	Eye (rabbit):0.87 mg - mild
	Oral (rat) LD50: 636 mg/kg ^[2]	Eye (rabbit):100 mg/30sec - mild Skin (rabbit):20 mg/24h-moderate Skin (rabbit):500 mg - moderate
kaolin	TOXICITY	IRRITATION
	Not Available	Not Available
hydrocarbon propellant	TOXICITY	IRRITATION
	Inhalation (rat) LC50: 84.684 mg/l15 min ^[1] Inhalation (rat) LC50: 90.171125 mg/l15 min ^[1]	Not Available

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

METHYLENE CHLORIDE	WARNING: This substance has been classified by the IARC as Group 2A: Probably Carcinogenic to Humans. Inhalation (human) TCLo: 500 ppm/ 1 y - I Eye(rabbit): 10 mg - mild
TOLUENE	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.
HYDROCARBON PROPELLANT	for Petroleum Hydrocarbon Gases: In many cases, there is more than one potentially toxic constituent in a refinery gas. In those cases, the constituent that is most toxic for a particular endpoint in an individual refinery stream is used to characterize the endpoint hazard for that stream. The hazard potential for each mammalian endpoint for each of the petroleum hydrocarbon gases is dependent upon each petroleum hydrocarbon gas constituent endpoint toxicity values (LC50, LOAEL, etc.) and the relative concentration of the constituent present in that gas. It should also be noted that for an individual petroleum hydrocarbon gas, the constituent characterizing toxicity may be different for different mammalian endpoints, again, being dependent upon the concentration of the different constituents in each, distinct petroleum hydrocarbon gas. All Hydrocarbon Gases Category members contain primarily hydrocarbons (i.e., alkanes and alkenes) and occasionally asphyxiant gases like hydrogen. The inorganic components of the petroleum hydrocarbon gases are less toxic than the C1 - C4 and C5 - C6 hydrocarbon components to both mammalian

	<p>and aquatic organisms. Unlike other petroleum product categories (e.g. gasoline, diesel fuel, lubricating oils, etc.), the inorganic and hydrocarbon constituents of hydrocarbon gases can be evaluated for hazard individually to then predict the screening level hazard of the Category members</p> <p>Acute toxicity: No acute toxicity LC50 values have been derived for the C1 -C4 and C5- C6 hydrocarbon (HC) fractions because no mortality was observed at the highest exposure levels tested (~ 5 mg/l) for these petroleum hydrocarbon gas constituents. The order of acute toxicity of petroleum hydrocarbon gas constituents from most to least toxic is:</p> <p>C5-C6 HCs (LC50 > 1063 ppm) > C1-C4 HCs (LC50 > 10,000 ppm) > benzene (LC50 = 13,700 ppm) > butadiene (LC50 = 129,000 ppm) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen).</p> <p>Repeat dose toxicity: With the exception of the asphyxiant gases, repeated dose toxicity has been observed in individual selected petroleum hydrocarbon gas constituents. Based upon LOAEL values, the order of order of repeated-dose toxicity of these constituents from most toxic to the least toxic is: Benzene (LOAEL .>=10 ppm) >C1-C4 HCs (LOAEL = 5,000 ppm; assumed to be 100% 2-butene) > C5-C6 HCs (LOAEL = 6,625 ppm) > butadiene (LOAEL = 8,000 ppm) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen).</p> <p>Genotoxicity:</p> <p>In vitro: The majority of the Petroleum Hydrocarbon Gases Category components are negative for <i>in vitro</i> genotoxicity. The exceptions are: benzene and 1,3-butadiene, which are genotoxic in bacterial and mammalian <i>in vitro</i> test systems.</p> <p>In vivo: The majority of the Petroleum Hydrocarbon Gases Category components are negative for <i>in vivo</i> genotoxicity. The exceptions are benzene and 1,3-butadiene, which are genotoxic in <i>in vivo</i> test systems</p> <p>Developmental toxicity: Developmental effects were induced by two of the petroleum hydrocarbon gas constituents, benzene and the C5 -C6 hydrocarbon fraction. No developmental toxicity was observed at the highest exposure levels tested for the other petroleum hydrocarbon gas constituents tested for this effect. The asphyxiant gases have not been tested for developmental toxicity. Based on LOAEL and NOAEL values, the order of acute toxicity of these constituents from most to least toxic is:</p> <p>Benzene (LOAEL = 20 ppm) > butadiene (NOAEL .>=1,000 ppm) > C5-C6 HCs (LOAEL = 3,463 ppm) > C1-C4 HCs (NOAEL >=5,000 ppm; assumed to be 100% 2-butene) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen).</p> <p>Reproductive toxicity: Reproductive effects were induced by only two petroleum hydrocarbon gas constituents, benzene and isobutane (a constituent of the C1-C4 hydrocarbon fraction). No reproductive toxicity was observed at the highest exposure levels tested for the other petroleum hydrocarbon gas constituents tested for this effect. The asphyxiant gases have not been tested for reproductive toxicity. Based on LOAEL and NOAEL values, the order of reproductive toxicity of these constituents from most to least toxic is:</p> <p>Benzene (LOAEL = 300 ppm) > butadiene (NOAEL .>=6,000 ppm) > C5-C6 HCs (NOAEL .>=6,521 ppm) > C1-C4 HCs (LOAEL = 9,000 ppm; assumed to be 100% isobutane) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen)</p>
RocX Mine Mark Paint & HYDROCARBON PROPELLANT	No significant acute toxicological data identified in literature search.
RocX Mine Mark Paint & METHYLENE CHLORIDE	
RocX Mine Mark Paint & METHYLENE CHLORIDE	The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
RocX Mine Mark Paint & METHYLENE CHLORIDE	The material may produce severe 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) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.
RocX Mine Mark Paint & TOLUENE	<p>For toluene:</p> <p>Acute Toxicity</p> <p>Humans exposed to intermediate to high levels of toluene for short periods of time experience adverse central nervous system effects ranging from headaches to intoxication, convulsions, narcosis, and death. Similar effects are observed in short-term animal studies.</p> <p>Humans - Toluene ingestion or inhalation can result in severe central nervous system depression, and in large doses, can act as a narcotic. The ingestion of about 60 mL resulted in fatal nervous system depression within 30 minutes in one reported case.</p> <p>Constriction and necrosis of myocardial fibers, markedly swollen liver, congestion and haemorrhage of the lungs and acute tubular necrosis were found on autopsy.</p> <p>Central nervous system effects (headaches, dizziness, intoxication) and eye irritation occurred following inhalation exposure to 100 ppm toluene 6 hours/day for 4 days.</p> <p>Exposure to 600 ppm for 8 hours resulted in the same and more serious symptoms including euphoria, dilated pupils, convulsions, and nausea. Exposure to 10,000-30,000 ppm has been reported to cause narcosis and death</p> <p>Toluene can also strip the skin of lipids causing dermatitis</p> <p>Animals - The initial effects are instability and incoordination, lachrymation and snuffles (respiratory exposure), followed by narcosis. Animals die of respiratory failure from severe nervous system depression. Cloudy swelling of the kidneys was reported in rats following inhalation exposure to 1600 ppm, 18-20 hours/day for 3 days</p> <p>Subchronic/Chronic Effects:</p> <p>Repeat doses of toluene cause adverse central nervous system effects and can damage the upper respiratory system, the liver, and the kidney. Adverse effects occur as a result from both oral and the inhalation exposures. A reported lowest-observed-effect level in humans for adverse neurobehavioral effects is 88 ppm.</p> <p>Humans - Chronic occupational exposure and incidences of toluene abuse have resulted in hepatomegaly and liver function changes. It has also resulted in nephrotoxicity and, in one case, was a cardiac sensitiser and fatal cardiotoxin.</p> <p>Neural and cerebellar dystrophy were reported in several cases of habitual "glue sniffing." An epidemiological study in France on workers chronically exposed to toluene fumes reported leukopenia and neutropenia. Exposure levels were not given in the secondary reference; however, the average urinary excretion of hippuric acid, a metabolite of toluene, was given as 4 g/L compared to a normal level of 0.6 g/L</p> <p>Animals - The major target organs for the subchronic/chronic toxicity of toluene are the nervous system, liver, and kidney. Depressed immune response has been reported in male mice given doses of 105 mg/kg/day for 28 days. Toluene in corn oil administered to F344 male and female rats by gavage 5 days/week for 13 weeks, induced prostration, hypoactivity, ataxia, piloerection, lachrymation, excess salivation, and body tremors at doses 2500 mg/kg. Liver, kidney, and heart weights were also increased at this dose and histopathologic lesions were seen in the liver, kidneys, brain and urinary bladder. The no-observed-adverse effect level (NOAEL) for the study was 312 mg/kg (223 mg/kg/day) and the lowest-observed-adverse effect level (LOAEL) for the study was 625 mg/kg (446 mg/kg/day) .</p> <p>Developmental/Reproductive Toxicity</p> <p>Exposures to high levels of toluene can result in adverse effects in the developing human fetus. Several studies have indicated that high levels of toluene can also adversely effect the developing offspring in laboratory animals.</p> <p>Humans - Variable growth, microcephaly, CNS dysfunction, attentional deficits, minor craniofacial and limb abnormalities, and developmental delay were seen in three children exposed to toluene in utero as a result of maternal solvent abuse before and during pregnancy</p> <p>Animals - Sternebral alterations, extra ribs, and missing tails were reported following treatment of rats with 1500 mg/m3 toluene 24 hours/day during days 9-14 of gestation. Two of the dams died during the exposure. Another group of rats received 1000 mg/m3 8 hours/day during days 1-21 of gestation. No maternal deaths or toxicity occurred, however, minor skeletal retardation was present in the exposed fetuses. CFLP Mice were exposed to 500 or 1500 mg/m3 toluene continuously during days 6-13 of pregnancy. All dams died at the high dose during the first 24 hours of exposure, however none died at 500 mg/m3. Decreased foetal weight was reported, but there were no differences in the incidences of skeletal malformations or anomalies between the treated and control offspring.</p> <p>Absorption - Studies in humans and animals have demonstrated that toluene is readily absorbed via the lungs and the gastrointestinal tract. Absorption</p>

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through the skin is estimated at about 1% of that absorbed by the lungs when exposed to toluene vapor.

Dermal absorption is expected to be higher upon exposure to the liquid; however, exposure is limited by the rapid evaporation of toluene .

Distribution - In studies with mice exposed to radiolabeled toluene by inhalation, high levels of radioactivity were present in body fat, bone marrow, spinal nerves, spinal cord, and brain white matter. Lower levels of radioactivity were present in blood, kidney, and liver. Accumulation of toluene has generally been found in adipose tissue, other tissues with high fat content, and in highly vascularised tissues .

Metabolism - The metabolites of inhaled or ingested toluene include benzyl alcohol resulting from the hydroxylation of the methyl group. Further oxidation results in the formation of benzaldehyde and benzoic acid. The latter is conjugated with glycine to yield hippuric acid or reacted with glucuronic acid to form benzoyl glucuronide. o-cresol and p-cresol formed by ring hydroxylation are considered minor metabolites

Excretion - Toluene is primarily (60-70%) excreted through the urine as hippuric acid. The excretion of benzoyl glucuronide accounts for 10-20%, and excretion of unchanged toluene through the lungs also accounts for 10-20%. Excretion of hippuric acid is usually complete within 24 hours after exposure.

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 available to make classification
 ⊖ – Data Not Available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

RocX Mine Mark Paint Fluoro	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
methylene chloride	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	=13.1mg/L	1
	EC50	48	Crustacea	=108.5mg/L	1
	EC50	72	Algae or other aquatic plants	242mg/L	4
	NOEC	96	Algae or other aquatic plants	56mg/L	4
toluene	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.0073mg/L	4
	EC50	48	Crustacea	3.78mg/L	5
	EC50	72	Algae or other aquatic plants	12.5mg/L	4
	BCF	24	Algae or other aquatic plants	10mg/L	4
	NOEC	168	Crustacea	0.74mg/L	5
kaolin	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
hydrocarbon propellant	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available

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

Within an aromatic series, acute toxicity increases with increasing alkyl substitution on the aromatic nucleus. For example, there is an increase in toxicity as alkylation of the naphthalene structure increases. The order of most toxic to least in a study using grass shrimp (*Palaemonetes pugio*) and brown shrimp (*Penaeus aztecus*) was dimethylnaphthalenes > methylnaphthalenes > naphthalenes.

Studies conclude that the toxicity of an oil appears to be a function of its di-aromatic and tri-aromatic hydrocarbons, which includes three-ring hydrocarbons such as phenanthrene.

The heavier (4-, 5-, and 6-ring) PAHs are more persistent than the lighter (2- and 3-ring) PAHs and tend to have greater carcinogenic and other chronic impact potential. PAHs in general are more frequently associated with chronic risks. These risks include cancer and often are the result of exposures to complex mixtures of chronic-risk aromatics (such as PAHs, alkyl PAHs, benzenes, and alkyl benzenes), rather than exposures to low levels of a single compound.

Anthracene is a phototoxic PAH. UV light greatly increases the toxicity of anthracene to bluegill sunfish. Benchmarks developed in the absence of UV light may be under-protective, and biological resources in strong sunlight are at more risk than those that are not.

For methylene chloride:

log Kow: 1.25

log Koc: 1.68

log Kom: 1.44

Henry's atm m³/mol: 2.68E-03

BCF: 5

Environmental fate:

Methylene chloride is a volatile liquid, and tends to volatilise to the atmosphere from water and soil. The half-life of methylene chloride volatilisation from water has been found to be 21 minutes under experimental conditions but actual volatilisation from natural waters will depend on the rate of mixing, wind speed, temperature, and other factors. The Henry's law constant value (H) of 0.002 atm/m³/mol indicates that methylene chloride will volatilise rapidly from moist soil and water surfaces.

Methylene chloride is not strongly sorbed to soils or sediments. Based on its low soil organic carbon partitioning coefficient (Koc) of 25, methylene chloride is likely to be very highly mobile in soils and may be expected to leach from soils into groundwater.

Continued...

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Based on a reported log octanol/water partition coefficient (Kow) of 1.3 an estimated bioconcentration factor (BCF) of 2.3 was derived. There is no evidence of biomagnification, but because the estimated BCF is low, significant biomagnification of methylene chloride in aquatic food chains is not expected.

Air: The main degradation pathway for methylene chloride in air is its reaction with photochemically generated hydroxyl radicals. Thus, the atmospheric lifetime of methylene chloride may be predicted from the hydroxyl radical concentration in air and the rate of reaction. Most reported rates for hydroxyl radical reaction with methylene chloride range from 1.0 x10⁻¹³ to 1.5 x10⁻¹³ cm³/mol/sec, and estimates of average atmospheric hydroxyl radical concentration range from 2.5 x10⁺⁵ to 1x10⁺⁶ mol/cm³ Using this information, an average atmospheric lifetime for methylene chloride may be calculated to be 130 days. Because this degradation pathway is relatively slow, methylene chloride may become widely dispersed but is not likely to accumulate in the atmosphere. The small amount of methylene chloride which reaches the stratosphere (about 1%) may undergo direct photolytic degradation; however, photolysis in the troposphere is not expected. Reactions of methylene chloride with ozone or other common atmospheric species (e.g., oxygen atoms, chlorine atoms, and nitrate radicals) are not believed to contribute to its breakdown.

Water: Methylene chloride undergoes slow hydrolysis in water. The experimental half-life reported for the hydrolysis reaction, at neutral conditions, is approximately 18 months at 25 C. However, the rate of reaction varies greatly with changes in temperature and pH. A hydrolytic half-life of 14 days was reported for methylene chloride in acidic solutions at 80-150 C. This experimental value, when extrapolated to 25 C, is about 700 years. Different mechanisms of hydrolyses may be responsible for these two widely different values.

Both aerobic and anaerobic biodegradation may be an important fate process for methylene chloride in water. Methylene chloride has been observed to undergo degradation at a rapid rate under aerobic conditions. Reported total methylene chloride loss was 100% after 7 days in a static culture flask biodegradability screening test.

Sediment and Soil: The rate of biodegradation was found to be dependent on soil type, substrate concentration, and redox state of the soil. Methylene chloride biodegradation has been reported to occur under both aerobic conditions and anaerobic conditions. The biodegradation of methylene chloride appears to be accelerated by the presence of elevated levels of organic carbon. Methylene chloride has a low tendency to absorb to soil; therefore, there is a potential for leaching to groundwater. Also, because of the high vapor pressure, volatilisation to air is also a likely fate process from dry soil. Its high Henry's law constant (0.002 atm/m³/mol) indicates that volatilization from moist soil is also likely.

For toluene:

- log Kow : 2.1-3
- log Koc : 1.12-2.85
- Koc : 37-260
- log Kom : 1.39-2.89
- Half-life (hr) air : 2.4-104
- Half-life (hr) H2O surface water : 5.55-528
- Half-life (hr) H2O ground : 168-2628
- Half-life (hr) soil : <48-240
- Henry's Pa m³/mol: 518-694
- Henry's atm m³/mol: 5.94E-03
- BOD 5 0.86-2.12, 5%
- COD : 0.7-2.52,21-27%
- ThOD : 3.13
- BCF : 1.67-380
- log BCF : 0.22-3.28

Environmental fate:

Transport: The majority of toluene evaporates to the atmosphere from the water and soil. It is moderately retarded by adsorption to soils rich in organic material (Koc = 259), therefore, transport to ground water is dependent on the soil composition. In unsaturated topsoil containing organic material, it has been estimated that 97% of the toluene is adsorbed to the soil and only about 2% is in the soil-water phase and transported with flowing groundwater. There is little retardation in sandy soils and 2-13% of the toluene was estimated to migrate with flowing water; the remainder was volatilised, biodegraded, or unaccounted for. In saturated deep soils with no soil-air phase, about 48% may be transported with flowing groundwater.

Transformation/Persistence:

Air - The main degradation pathway for toluene in the atmosphere is reaction with photochemically produced hydroxyl radicals. The estimated atmospheric half life for toluene is about 13 hours. Toluene is also oxidised by reactions with atmospheric nitrogen dioxide, oxygen, and ozone, but these are minor degradation pathways. Photolysis is not considered a significant degradative pathway for toluene

Soil - In surface soil, volatilisation to air is an important fate process for toluene. Biodegradation of toluene has been demonstrated in the laboratory to occur with a half life of about 1 hour. In the environment, biodegradation of toluene to carbon dioxide occurs with a typical half life of 1-7 days.

Water - An important fate process for toluene is volatilization, the rate of which depends on the amount of turbulence in the surface water. The volatilisation of toluene from static water has a half life of 1-16 days, whereas from turbulent water the half life is 5-6 hours. Degradation of toluene in surface water occurs primarily by biodegradation with a half life of less than one day under favorable conditions (presence of microorganisms, microbial adaptation, and optimum temperature). Biodegradation also occurs in shallow groundwater and in salt water at a reduced rate. No data are available on anaerobic degradation of toluene in deep ground water conditions where aerobic degradation would be minimal.

Biota - Bioaccumulation in most organisms is limited by the metabolism of toluene into more polar compounds that have greater water solubility and a lower affinity for lipids. Bioaccumulation in the food chain is predicted to be low.

Ecotoxicity:

- Toluene has moderate acute toxicity to aquatic organisms; several toxicity values are in the range of greater than 1 mg/L and 100 mg/L.
- Fish LC50 (96 h): fathead minnow (*Pimephales promelas*) 12.6-72 mg/l; *Lepomis macrochirus* 13-24 mg/l;
- guppy (*Poecilia reticulata*) 28.2-59.3 mg/l; channel catfish (*Ictalurus punctatus*) 240 mg/l; goldfish (*Carassius auratus*); 22.8-57.68 mg/l
- Crustaceans LC50 (96 h): grass shrimp (*Palaemonetes pugio*) 9.5 ppm, crab larvae stage (*Cancer magister*) 28 ppm; shrimp (*Crangon franciscorum*) 4.3 ppm; daggerblade grass shrimp (*Palaemonetes pugio*) 9.5 mg/l
- Algae EC50 (24 h): green algae (*Chlorella vulgaris*) 245 mg/l (growth); (72 h) green algae (*Selenastrum capricornutum*) 12.5 mg/l (growth)

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
methylene chloride	LOW (Half-life = 56 days)	HIGH (Half-life = 191 days)
toluene	LOW (Half-life = 28 days)	LOW (Half-life = 4.33 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
methylene chloride	LOW (BCF = 40)
toluene	LOW (BCF = 90)

Mobility in soil

Ingredient	Mobility
methylene chloride	LOW (KOC = 23.74)
toluene	LOW (KOC = 268)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

- Product / Packaging disposal ▶ **DO NOT** allow wash water from cleaning or process equipment to enter drains.

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- ▶ It may be necessary to collect all wash water for treatment before disposal.
- ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.
- ▶ Where in doubt contact the responsible authority.
- ▶ Consult State Land Waste Management Authority for disposal.
- ▶ Discharge contents of damaged aerosol cans at an approved site.
- ▶ Allow small quantities to evaporate.
- ▶ **DO NOT incinerate or puncture aerosol cans.**
- ▶ Bury residues and emptied aerosol cans at an approved site.

SECTION 14 TRANSPORT INFORMATION

Labels Required

	
Marine Pollutant	NO
HAZCHEM	2Y

Land transport (ADG)

UN number	1950	
UN proper shipping name	AEROSOLS	
Transport hazard class(es)	Class	2.1
	Subrisk	Not Applicable
Packing group	Not Applicable	
Environmental hazard	Not Applicable	
Special precautions for user	Special provisions	63 190 277 327 344
	Limited quantity	1000ml

Air transport (ICAO-IATA / DGR)

UN number	1950	
UN proper shipping name	Aerosols, flammable; Aerosols, flammable (engine starting fluid)	
Transport hazard class(es)	ICAO/IATA Class	2.1
	ICAO / IATA Subrisk	Not Applicable
	ERG Code	10L
Packing group	Not Applicable	
Environmental hazard	Not Applicable	
Special precautions for user	Special provisions	A145 A167 A802; A1 A145 A167 A802
	Cargo Only Packing Instructions	203
	Cargo Only Maximum Qty / Pack	150 kg
	Passenger and Cargo Packing Instructions	203; Forbidden
	Passenger and Cargo Maximum Qty / Pack	75 kg; Forbidden
	Passenger and Cargo Limited Quantity Packing Instructions	Y203; Forbidden
	Passenger and Cargo Limited Maximum Qty / Pack	30 kg G; Forbidden

Sea transport (IMDG-Code / GGVSee)

UN number	1950	
UN proper shipping name	AEROSOLS	
Transport hazard class(es)	IMDG Class	2.1
	IMDG Subrisk	Not Applicable
Packing group	Not Applicable	
Environmental hazard	Not Applicable	
Special precautions for user	EMS Number	F-D, S-U
	Special provisions	63 190 277 327 344 381 959
	Limited Quantities	1000ml

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

Not Applicable

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SECTION 15 REGULATORY INFORMATION

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

METHYLENE CHLORIDE(75-09-2) 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

TOLUENE(108-88-3) 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

KAOLIN(1332-58-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
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HYDROCARBON PROPELLANT(68476-85-7.) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists	International Air Transport Association (IATA) Dangerous Goods Regulations - Prohibited List Passenger and Cargo Aircraft

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (toluene; kaolin; hydrocarbon propellant; methylene chloride)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	N (kaolin; hydrocarbon propellant)
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	Y
Legend:	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION

Other information

Ingredients with multiple cas numbers

Name	CAS No
kaolin	1332-58-7, 71888-52-3, 1026990-70-4, 12198-85-5, 12199-11-0, 190086-05-6, 290817-34-4, 384842-32-4, 39406-22-9, 52624-41-6, 849104-81-0, 903527-69-5, 90803-81-9, 944250-63-9, 95077-05-7
hydrocarbon propellant	68476-85-7., 68476-86-8.

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.

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

Definitions and abbreviations

- PC – TWA: Permissible Concentration-Time Weighted Average
- PC – STEL: Permissible Concentration-Short Term Exposure Limit
- IARC: International Agency for Research on Cancer
- ACGIH: American Conference of Governmental Industrial Hygienists
- STEL: Short Term Exposure Limit
- TEEL: Temporary Emergency Exposure Limit.
- IDLH: Immediately Dangerous to Life or Health Concentrations
- OSF: Odour Safety Factor
- NOAEL :No Observed Adverse Effect Level
- LOAEL: Lowest Observed Adverse Effect Level
- TLV: Threshold Limit Value
- LOD: Limit Of Detection
- OTV: Odour Threshold Value
- BCF: BioConcentration Factors
- BEI: Biological Exposure Index

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