FOUNTAIN AEROSOL LINE MARKER - COLOUR RANGE

Fountain Linemarking

Chemwatch: 35762 Version No: 6.1

Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Chemwatch Hazard Alert Code: 4

Issue Date: **01/11/2019**Print Date: **22/02/2022**L.GHS.AUS.EN

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

Product Identifier		
Product name	Fountain Aerosol Line Marker	
Chemical Name	Not Applicable	
Synonyms	Aerosol Spray Paint	
Proper shipping name	AEROSOLS	
Chemical formula	Not Applicable	
Other means of identification	Not Available	

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

	Aerosol spray paint.
Relevant identified uses	Application is by spray atomisation from a hand held aerosol pack
	Use according to manufacturer's directions.

Details of the supplier of the safety data sheet

Registered company name	Fountain Linemarking Co
	3 Production Avenue Kogarah
Address	2217 NSW Australia
Telephone	+61 2 9587 0232
Fax	+61 2 9587 8443
Website	Not Available
Email	Not Available

Emergency telephone number

Association / Organisation	Not available	CHEMWATCH EMERGENCY RESPONSE
Emergency telephone numbers	+61 2 9587 0232 (Mon-Fri, 8am-5pm)	+61 1800 951 288
Other emergency telephone numbers	Not Available	+61 2 9186 1132

Once connected and if the message is not in your preferred language then please dial 01

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, Acute Toxicity (Oral) Category 4, Aspiration Hazard Category 1, Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Germ Cell Mutagenicity Category 2, Carcinogenicity Category 1A, Reproductive Toxicity Category 2, Specific Target Organ Toxicity - Repeated Exposure Category 2		
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI		

Label elements

Hazard pictogram(s)









Signal word | Danger

Hazard statement(s)

H222+H229	Extremely flammable aerosol. Pressurized container: may burst if heated.
H302	Harmful if swallowed.
H304	May be fatal if swallowed and enters airways.

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H315	Causes skin irritation.
H318	Causes serious eye damage.
H335	May cause respiratory irritation.
H336	May cause drowsiness or dizziness.
H341	Suspected of causing genetic defects.
H350	May cause cancer.
H361d	Suspected of damaging the unborn child.
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, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P211	Do not spray on an open flame or other ignition source.
P251	Do not pierce or burn, even after use.
P260	Do not breathe mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.
P331	Do NOT induce vomiting.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P330	Rinse mouth.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

P405	Store locked up.
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 to authorised hazardous or special waste collection point in accordance with any local regulation.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
108-88-3	10-30	toluene
13463-67-7	10-30	titanium dioxide
471-34-1	10-30	calcium carbonate
1332-58-7	1-10	kaolin
Not Available	1-10	hydrocarbon resin (aromatic)
Not Available	1-10	vinyl toluene acrylate resin
63449-39-8	1-10	chlorinated paraffin, long chain grades
68476-85-7.	30-60	hydrocarbon propellant
Not Available		NOTE: Manufacturer has supplied full ingredient
Not Available		information to allow CHEMWATCH assessment.
Leg	,	nwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI;

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SECTION 4 First aid measures

Description of first aid measures If aerosols come in contact with the eves: 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 **Eve Contact** upper and lower lids. Transport to hospital or doctor without delay. Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. If solids or aerosol mists are deposited upon the skin: Flush skin and hair with running water (and soap if available). Skin Contact Remove any adhering solids with industrial skin cleansing cream. DO NOT use solvents. Seek medical attention in the event of irritation. If aerosols, fumes or combustion products are inhaled: Remove to fresh air. Lay patient down. Keep warm and rested. Inhalation 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. Avoid giving milk or oils. Avoid giving alcohol. Ingestion Not considered a normal route of entry. If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

Indication of any immediate medical attention and special treatment needed

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

Advice for firefighters

- ▶ 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.
- Fire Fighting 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.
 - 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.

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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. Fire/Explosion Hazard May emit acrid, poisonous or corrosive fumes. On combustion, may emit toxic fumes of carbon monoxide (CO). Combustion products include: carbon monoxide (CO) carbon dioxide (CO2) hydrogen chloride phosgene other pyrolysis products typical of burning organic material. Contains low boiling substance: Closed containers may rupture due to pressure buildup under fire conditions. HAZCHEM

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	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.
Major Spills	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.

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

SECTION 7 Handling and storage

Precautions for safe handling	
Safe handling	 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. Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	 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.

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- 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.

Conditions for safe storage, including any incompatibilities

Suitable container	Aerosol dispenser. Check that containers are clearly labelled.
Storage incompatibility	Avoid reaction with oxidising agents

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	toluene	Toluene	50 ppm / 191 mg/m3	574 mg/m3 / 150 ppm	Not Available	Not Available
Australia Exposure Standards	titanium dioxide	Titanium dioxide	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	calcium carbonate	Calcium carbonate	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	kaolin	Kaolin	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	hydrocarbon propellant	LPG (liquified petroleum gas)	1000 ppm / 1800 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
toluene	Not Available	Not Available	Not Available
titanium dioxide	30 mg/m3	330 mg/m3	2,000 mg/m3
calcium carbonate	45 mg/m3	210 mg/m3	1,300 mg/m3
hydrocarbon propellant	65,000 ppm	2.30E+05 ppm	4.00E+05 ppm

Ingredient	Original IDLH	Revised IDLH
toluene	500 ppm	Not Available
titanium dioxide	5,000 mg/m3	Not Available
calcium carbonate	Not Available	Not Available
kaolin	Not Available	Not Available
chlorinated paraffin, long chain		
grades	Not Available	Not Available
hydrocarbon propellant	2,000 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.

The TLV-TWA is thought to be protective against the significant risk of physical irritation associated with exposure.

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. The label on a package containing 1% or more of titanium oxide with aerodynamic diameter equal or below 10 microns shall bear the following statement: EUH211 "Warning! Hazardous respirable droplets may be formed when sprayed. Do NOT breathe spray or mist

·The label on the packaging of solid mixtures containing 1% or more of titanium dioxide shall bear the following statement: EUH212" "Warning! Hazardous respirable dust may be formed when used. Do not breathe dust".

In addition, the label on the packaging of liquid and solid mixtures not intended for the general public and not classified as hazardous which are labelled EUH211 or EU212 shall bear statement EUH210: "Safety data sheet available on request." 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

Appropriate engineering

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls car

be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are:

Process controls which involve changing the way a job activity or process is done to reduce the risk.

Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure.

General exhaust is adequate under normal conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection.

Provide adequate ventilation in warehouse or closed storage areas.

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.

controls

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.)

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range	
1: Room air currents minimal or favourable to capture	1: Disturbing room air 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	

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

No special equipment for minor exposure i.e. when handling small quantities.

OTHERWISE: For potentially moderate or heavy exposures

- Safety glasses with side shields
- NOTE: Contact lenses pose a special hazard; soft lenses may absorb irritants and ALL lenses concentrate them.

Skin protection

See Hand protection below

No special equipment needed when handling small quantities.

- Hands/feet protection

 OTHERWISE:
 For potentially
 - 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.

Body protection

Other protection

See Other protection below

No special equipment needed when handling small quantities.

OTHERWISE:

- 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.

BRETHERICK: Handbook of Reactive Chemical Hazards.

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:

Fountain Aerosol Line Marker

Material	СРІ
PE/EVAL/PE	A
PVA	A
VITON	A
VITON/CHLOROBUTYL	A
TEFLON	В
BUTYL	С
CPE	С
NEOPRENE	С
NEOPRENE/NATURAL	С

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 =

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NITRILE	С
NITRILE+PVC	С
PVC	С
SARANEX-23	С
SARANEX-23 2-PLY	С
VITON/NEOPRENE	С

* 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.

Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

	ance

White liquid with aromatic solvent odour; does not mix with water.

	Supplied as an aerosol pack. Contents under PRESSURE. Contains highly flammable hydrocarbon propellant.			
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	Immiscible	pH as a solution (Not Available%)	Not Applicable	
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available	

SECTION 10 Stability and reactivity

Reactivity	See section 7
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

Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.

Inhaled

Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.

Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.

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The acute toxicity of inhaled alkylbenzenes is best described by central nervous system depression. As a rule, these compounds may also act

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.

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

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

Common, generalised symptoms associated with toxic gas inhalation include:

- 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),

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.

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.

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 WARNING: Intentional misuse by concentrating/inhaling contents may be lethal.

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 postinflammatory 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. 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.

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.

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

Ingestion

Skin Contact

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.

Not normally a hazard due to physical form of product.

Considered an unlikely route of entry in commercial/industrial environments

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

The material may accentuate any pre-existing dermatitis condition

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.

The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either

- produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or
- 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.

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.

Eve

When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation. 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...

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. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.

Chronic

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

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become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.

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. On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body 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.

Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

Principal route of occupational exposure to the gas is by inhalation.

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.

Hydrocarbon solvents are liquid hydrocarbon fractions derived from petroleum processing streams, containing only carbon and hydrogen atoms, with carbon numbers ranging from approximately C5-C20 and boiling between approximately 35-370 deg C. Many of the hydrocarbon solvents have complex and variable compositions with constituents of 4 types, alkanes (normal paraffins, isoparaffins, and cycloparaffins) and aromatics (primarily alkylated one- and two-ring species). Despite the compositional complexity, most hydrocarbon solvent constituents have similar toxicological properties, and the overall toxicological hazards can be characterized in generic terms. Hydrocarbon solvents can cause chemical pneumonitis if aspirated into the lung, and those that are volatile can cause acute CNS effects and/or ocular and respiratory irritation at exposure levels exceeding occupational recommendations. Otherwise, there are few toxicologically important effects. The exceptions, n-hexane and naphthalene, have unique toxicological properties

Animal studies:

No deaths or treatment related signs of toxicity were observed in rats exposed to light alkylate naphtha (paraffinic hydrocarbons) at concentrations of 668, 2220 and 6646 ppm for 6 hrs/day, 5 days/wk for 13 weeks. Increased liver weights and kidney toxicity (male rats) was observed in high dose animals. Exposure to pregnant rats at concentrations of 137, 3425 and 6850 ppm did not adversely affect reproduction or cause maternal or foetal toxicity. Lifetime skin painting studies in mice with similar naphthas have shown weak or no carcinogenic activity following prolonged and repeated exposure. Similar

naphthas/distillates, when tested at nonirritating dose levels, did not show any significant carcinogenic activity indicating that this tumorigenic response is likely related to chronic irritation and not to dose. The mutagenic potential of naphthas has been reported to be largely negative in a variety of mutagenicity tests. The exact relationship between these results and human health is not known. Some components of this product have been shown to produce a species specific, sex hormonal dependent kidney lesion in male rats from repeated oral or inhalation exposure. Subsequent research has shown that the kidney damage develops via the formation of a alpha-2u-globulin, a mechanism unique to the male rat. Humans do not form alpha-2u-globulin, therefore, the kidney effects resulting from this mechanism are not relevant in human. Chronic toluene habituation occurs following intentional abuse (glue sniffing) or from occupational exposure. Ataxia, incoordination and tremors of the hands and feet (as a consequence of diffuse cerebral atrophy), headache, abnormal speech, transient memory loss, convulsions, coma, drowsiness, reduced colour perception, frank blindness, nystagmus (rapid, involuntary eye-movements), hearing loss leading to deafness and mild dementia have all been associated with chronic abuse. Peripheral nerve damage, encephalopathy, giant axonopathy electrolyte disturbances in the cerebrospinal fluid and abnormal computer tomographic (CT scans) are common amongst toluene addicts. Although toluene abuse has been linked with kidney disease, this does not commonly appear in cases of occupational toluene exposures. Cardiac and haematological toxicity are however associated with chronic toluene exposures. Cardiac arrhythmia, multifocal and premature ventricular contractions and supraventricular tachycardia are present in 20% of patients who abused toluene-containing paints. Previous suggestions that chronic toluene inhalation produced human peripheral neuropathy have been discounted. However central nervous system (CNS) depression is well documented where blood toluene exceeds 2.2 mg%. Toluene abusers can achieve transient circulating concentrations of 6.5 %. Amongst workers exposed for a median time of 29 years, to toluene, no subacute effects on neurasthenic complaints and psychometric test results could be established.

The prenatal toxicity of very high toluene concentrations has been documented for several animal species and man. Malformations indicative of specific teratogenicity have not generally been found. Neonatal toxicity, described in the literature, takes the form of embryo death or delayed foetal growth and delayed skeletal system development. Permanent damage of children has been seen only when mothers have suffered from chronic intoxication as a result of "sniffing".

toluene

titanium dioxide

TOXICITY	IRRITATION
Not Available	Not Available
TOXICITY	IRRITATION
Dermal (rabbit) LD50: 12124 mg/kg ^[2]	Eye (rabbit): 2mg/24h - SEVERE
Inhalation(Rat) LC50; >13350 ppm4h ^[2]	Eye (rabbit):0.87 mg - mild
Oral (Rat) LD50; 636 mg/kg ^[2]	Eye (rabbit):100 mg/30sec - mild
	Eye: adverse effect observed (irritating) ^[1] Skin (rabbit):20 mg/24h-moderate
	Skin (rabbit):500 mg - moderate
	Skin: adverse effect observed (irritating) ^[1]
	Skin: no adverse effect observed (not irritating) ^[1]
TOXICITY	IRRITATION
dermal (hamster) LD50: >=10000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
Inhalation(Rat) LC50; >2.28 mg/l4h ^[1]	Skin (human): 0.3 mg /3D (int)-mild *
Oral (Rat) LD50; >=2000 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]

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	TOXICITY	IRRITATION	
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 0.75 mg/24h - SEVERE	
calcium carbonate	Inhalation(Rat) LC50; >3 mg/l4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]	
	Oral (Rat) LD50; >2000 mg/kg ^[1]	Skin (rabbit): 500 mg/24h-moderate	
		Skin: no adverse effect observed (not irritating) ^[1]	
	TOXICITY	IRRITATION	
kaolin	Not Available	Not Available	
	TOXICITY	IRRITATION	
chlorinated paraffin, long chain grades	Dermal (rabbit) LD50: >10000 mg/kg ^[2]	Not Available	
chain grades	Oral (Rat) LD50; >5000 mg/kg ^[1]		
	TOXICITY	IRRITATION	
hydrocarbon propellant	Inhalation(Rat) LC50; 658 mg/l4h ^[2]	Not Available	
Legend:	Nalue 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		

Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of

appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.

For titanium dioxide:

Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals. (General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) With regard to inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral ingestion of fine titanium dioxide showed particle sizedependent absorption by the gastrointestinal tract and large interindividual variations in blood levels of titanium dioxide. Studies on the application of sunscreens containing ultrafine titanium dioxide to healthy skin of human volunteers revealed that titanium dioxide particles only penetrate into the outermost layers of the stratum corneum, suggesting that healthy skin is an effective barrier to titanium dioxide. There are no studies on penetration of titanium dioxide in compromised skin.

Respiratory effects that have been observed among groups of titanium dioxide-exposed workers include decline in lung function, pleural disease with plaques and pleural thickening, and mild fibrotic changes. However, the workers in these studies were also exposed to asbestos and/or silica

No data were available on genotoxic effects in titanium dioxide-exposed humans.

Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain. Clearance of titanium dioxide is also affected by pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols. Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide are more slowly cleared than their fine counterparts.

TITANIUM DIOXIDE

Titanium dioxide causes varying degrees of inflammation and associated pulmonary effects including lung epithelial cell injury, cholesterol granulomas and fibrosis. Rodents experience stronger pulmonary effects after exposure to ultrafine titanium dioxide particles compared with fine particles on a mass basis. These differences are related to lung burden in terms of particle surface area, and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium.

Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the generation of reactive oxygen species by both particle types. This effect is stronger for ultrafine than for fine titanium oxide, and is markedly enhanced by exposure to simulated sunlight/ultraviolet light.

Animal carcinogenicity data

Pigmentary and ultrafine titanium dioxide were tested for carcinogenicity by oral administration in mice and rats, by inhalation in rats and female mice, by intratracheal administration in hamsters and female rats and mice, by subcutaneous injection in rats and by intraperitoneal administration in male mice and female rats.

In one inhalation study, the incidence of benign and malignant lung tumours was increased in female rats. In another inhalation study, the incidences of lung adenomas were increased in the high-dose groups of male and female rats. Cystic keratinizing lesions that were diagnosed as squamous-cell carcinomas but re-evaluated as non-neoplastic pulmonary keratinizing cysts were also observed in the high-dose groups of female rats. Two inhalation studies in rats and one in female mice were negative.

Intratracheally instilled female rats showed an increased incidence of both benign and malignant lung tumours following treatment with two types of titanium dioxide. Tumour incidence was not increased in intratracheally instilled hamsters and female mice.

In-vivo studies have shown enhanced micronucleus formation in bone marrow and peripheral blood lymphocytes of intraperitoneally instilled mice. Increased Hprt mutations were seen in lung epithelial cells isolated from titanium dioxide-instilled rats. In another study, no enhanced oxidative DNA damage was observed in lung tissues of rats that were intratracheally instilled with titanium dioxide. The results of most in-vitro genotoxicity studies with titanium dioxide were negative.

The material may produce moderate eye irritation leading to 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.

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

No evidence of carcinogenic properties. No evidence of mutagenic or teratogenic effects.

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

CALCIUM CARBONATE

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produce conjunctivitis.

for bentonite clays:

KAOLIN

CHLORINATED PARAFFIN,

LONG CHAIN GRADES

HYDROCARBON

PROPELLANT

Bentonite (CAS No. 1302-78-9) consists of a group of clays formed by crystallisation of vitreous volcanic ashes that were deposited in water. The expected acute oral toxicity of bentonite in humans is very low (LD50>15 g/kg). However, severe anterior segment inflammation, uveitis and retrocorneal abscess from eye exposure were reported when bentonite had been used as a prophypaste.

In a 33 day dietary (2 and 6%) and a 90 day dietary (1, 3 and 5%) studies in chickens, no changes in behaviour, overall state, clinical and biochemical parameters and electrolytic composition of the blood. Repeat dietary administration of bentonite did not affect calcium or phosphorus metabolism. However, larger amounts caused decreased growth, muscle weakness, and death with marked changes in both calcium and phosphorus metabolism.

Bentonite did not cause fibrosis after 1 year exposure of 60 mg dust (<5 um) in a rat study. However, in a second rat study, where 5 um particles were intratracheally instilled at 5, 15 and 45 mg/rat, dose-related fibrosis was observed. Bentonite clay dust is believed to be responsible for bronchial asthma in workers at a processing plant in USA.

Ingestion of bentonite without adequate liquids may result in intestinal obstruction in humans.

Hypokalaemia and microcytic iron-deficiency anaemia may occur in patients after repeat doses of clay. Chronic ingestion has been reported to cause myositis.

Oral (rat) LD50: >4000 mg/kg [I.C.I.] Cereclor range: Chlorinated paraffin waxes represents a family of substances which vary in molecular weight. Studies using the C12, 59% chlorinated variant (in combination with corn oil) caused tumors when force fed at very high doses over long periods of time. The C24, 43% chlorinated paraffin under the same conditions caused an increase in tumors only in the male mouse. A 13 week dietary, range finding study was conducted on rats with a C24, 70% chlorinated paraffin. This study established a no effect level of 900 mg/kg/day. Pregnant rats fed C16, 52% chlorinated paraffin had offspring which died during weaning.

The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

NOTE: C12, 60% chlorinated paraffin ICAS RN 108171-26-21 is classified by IARC as Group 2B. Possibly carcinogenic to humans. Studies using the C12, 59% chlorinated variant (in combination with corn oil) caused tumors when force fed at very high doses over long periods of time. Pregnant rats fed C16, 52% chlorinated paraffin had offspring which died during weaning.

High molecular weight liquid chloroparaffins are considered to be practically non-harmful. Special consideration should be given to solid grades of the material (eg Cereclor 70) because of relatively high levels of carbon tetrachloride remaining as a residual reactant. Vapours are readily absorbed through intact skin, requiring additional precautions in handling. Lifetime studies have been carried out with two grades of chlorinated paraffins. A short-chain grade with 58% chlorine caused tumours in rats

and mice. Male mice exposed to long-chain grades with 40% chlorine showed an excess of tumours at one site. It has been shown that the mechanisms by which short-term paraffins cause tumours are specific to rodents and may not have relevance to human health. Furthermore, chlorinated paraffins have been shown to non-genotoxic.

The Regulatory regime in various countries differs with respected to chlorinated paraffins.

In the USA, the short-chain (C12), 58% chlorine product has been classified and labelled as a carcinogen.

In Germany the MAK Commission has classified most chlorinated paraffins as Category IIIB (suspect carcinogens). They are not however included in the list of substances (TRGS 905) required to be labelled.

All EU Member States are required to classify short chain chlorinated paraffins as Category 3 carcinogens.

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 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

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:

 $\text{C5-C6 HCs (LC50} > 1063 \text{ ppm}) > \text{C1-C4 HCs (LC50} > 10,000 \text{ ppm}) > \text{benzene (LC50} = 13,700 \text{ ppm}) > \text{butadiene (LC50} = 129,000 \text{ ppm}) > \text{benzene (LC50} = 13,700 \text{ ppm}) > \text{benzene (LC50} = 129,000 \text{$ ppm) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen).

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). Genotoxicity:

In vitro: The majority of the Petroleum Hydrocarbon Gases Category components are negative for in vitro genotoxicity. The exceptions are: benzene and 1,3-butadiene, which are genotoxic in bacterial and mammalian in vitro test systems

In vivo: The majority of the Petroleum Hydrocarbon Gases Category components are negative for in vivo genotoxicity.

The exceptions are benzene and 1,3-butadiene, which are genotoxic in in vivo test systems

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:

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).

Reproductive toxicity: Reproductive effects were induced by only two petroleum hydrocarbon gas constituents, benzene and isobutane (a constituent of the 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:

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)

Fountain Aerosol Line Marker & TITANIUM DIOXIDE & **CALCIUM CARBONATE**

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 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

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Fountain Aerosol Line
Marker & TITANIUM DIOXIDE
& KAOLIN & HYDROCARBON

PROPELLANT

No significant acute toxicological data identified in literature search.

For toluene:

production

Acute Toxicity

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.

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.

Constriction and necrosis of myocardial fibers, markedly swollen liver, congestion and haemorrhage of the lungs and acute tubular necrosis were found on autopsy.

Central nervous system effects (headaches, dizziness, intoxication) and eye irritation occurred following inhalation exposure to 100 ppm toluene 6 hours/day for 4 days.

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

Toluene can also strip the skin of lipids causing dermatitis

Animals - The initial effects are instability and incoordination, lachrymation and sniffles (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

Subchronic/Chronic Effects:

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.

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.

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

Fountain Aerosol Line Marker & TOLUENE

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).

Developmental/Reproductive Toxicity

Exposures to high levels of toluene can result in adverse effects in the developing human foetus. Several studies have indicated that high levels of toluene can also adversely effect the developing offspring in laboratory animals.

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

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.

Absorption - Studies in humans and animals have demonstrated that toluene is readily absorbed via the lungs and the gastrointestinal tract. Absorption 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 glycuronic acid to form benzoyl glycuronide. 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.

TOLUENE & CALCIUM CARBONATE

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.

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

Legend:

X - Data either not available or does not fill the criteria for classification

Data available to make classification

SECTION 12 Ecological information

Toxicity

Fountain Aerosol Line Marker	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available

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	Endpoint	Test Duration (hr)	Species		١	/alue	Sourc
	NOEC(ECx)	168h	Crustacea		C).74mg/L	5
toluene	LC50	96h	Fish		5	5-35mg/l	4
	EC50	48h	Crustacea		3	3.78mg/L	5
	EC50	96h	Algae or other ag	uatic plants	>	-376.71mg/L	4
	Endpoint	Test Duration (hr)	Species		V	alue	Sourc
	BCF	1008h	Fish		<	1.1-9.6	7
	NOEC(ECx)	504h	Crustacea		0	.02mg/l	4
titanium dioxide	LC50	96h	Fish		1.	.85-3.06mg/l	4
	EC50	72h	Algae or other aq	uatic plants	3	.75-7.58mg/l	4
	EC50	48h	Crustacea		1.	.9mg/l	2
	EC50	96h	Algae or other aq	uatic plants	1	79.05mg/l	2
	Endpoint	Test Duration (hr)	Species		V	alue	Source
	NOEC(ECx)	1h	Fish			-320mg/l	4
calcium carbonate	LC50	96h		Fish		165200mg/L	4
	EC50	72h	Algae or other ac	uatic plants		14mg/l	2
	Endpoint	Test Duration (hr)	Species			Value	Source
kaolin	Not Available	Not Available	Not Available			Not Available	Not Availab
	Endpoint	Test Duration (hr)	Species		Value	•	Source
	EC0(ECx)	48h	Crustacea		>0.02	26-0.877mg/l	2
abladantad sasatta lasas							4
chlorinated paraffin, long chain grades	LC50	96h	Fish		0.06-	0.08mg/l	4
chlorinated paraffin, long chain grades	LC50 EC50	96h 72h		ic plants			
•			Fish Algae or other aquat Algae or other aquat		0.06- >3.2r >3.2r	ng/l	2
•	EC50 EC50	72h 96h	Algae or other aquat		>3.2r	ng/l	2
	EC50 EC50 Endpoint	72h 96h Test Duration (hr)	Algae or other aquat Algae or other aquat Species	ic plants	>3.2r	ng/l Value	2 2 Source
•	EC50 EC50	72h 96h	Algae or other aquat	ic plants	>3.2r	value 7.71mg/l	2
•	EC50 Endpoint EC50(ECx)	72h 96h Test Duration (hr) 96h	Algae or other aquat Algae or other aquat Species Algae or other a	ic plants	>3.2r	ng/l Value	2 2 Source
chain grades	EC50 EC50 Endpoint EC50(ECx)	72h 96h Test Duration (hr) 96h 96h	Algae or other aquat Algae or other aquat Species Algae or other a	aquatic plants	>3.2r	value 7.71mg/l 24.11mg/l	2 2 Source 2 2
chain grades	EC50 EC50 Endpoint EC50(ECx) LC50 EC50	72h 96h Test Duration (hr) 96h 96h	Algae or other aquat Algae or other aquat Species Algae or other a Fish Algae or other a	aquatic plants	>3.2r	value 7.71mg/l 24.11mg/l 7.71mg/l	2 2 Source 2 2 2

For aromatic hydrocarbons:

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 chronicrisk 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 underprotective, and biological resources in strong sunlight are at more risk than those that are not.

Volatile furandiones and aldehydes are significant atmospheric oxidation products of aromatic compounds. Highly acidic dicarboxylic acids produced by the reactions between furandiones and water were shown to rapidly acidify an aqueous phase 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

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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
toluene	LOW (Half-life = 28 days)	LOW (Half-life = 4.33 days)
titanium dioxide	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
toluene	LOW (BCF = 90)
titanium dioxide	LOW (BCF = 10)

Mobility in soil

Ingredient	Mobility
toluene	LOW (KOC = 268)
titanium dioxide	LOW (KOC = 23.74)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal

- 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



warme	POIII	utant

NO

HAZCHEM

Not Applicable

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 63 190 277 327 344 381 Limited quantity 1000ml	

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

Sea transport (IMDG-Code / GGVSee)

UN number	1950		
UN proper shipping name	AEROSOLS		
Transport hazard class(es)	IMDG Class 2.1 IMDG Subrisk No	1ot Applicable	
Packing group	Not Applicable		
Environmental hazard	Not Applicable		
Special precautions for user	EMS Number Special provisions Limited Quantities	F-D , S-U 63 190 277 327 344 381 959 1000 ml	

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

Not Applicable

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

Product name	Group
toluene	Not Available
titanium dioxide	Not Available
calcium carbonate	Not Available
kaolin	Not Available
chlorinated paraffin, long chain grades	Not Available
hydrocarbon propellant	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
toluene	Not Available
titanium dioxide	Not Available
calcium carbonate	Not Available
kaolin	Not Available
chlorinated paraffin, long chain grades	Not Available
hydrocarbon propellant	Not Available

SECTION 15 Regulatory information

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

toluene is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP)

- Schedule 5

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP)

- Schedule 6

Australian Inventory of Industrial Chemicals (AIIC) Chemical

Footprint Project - Chemicals of High Concern List

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

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Australian Inventory of Industrial Chemicals (AIIC) Chemical

Footprint Project - Chemicals of High Concern List

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

Zeus

calcium carbonate is found on the following regulatory

lists Australian Inventory of Industrial Chemicals (AIIC)

kaolin is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

chlorinated paraffin, long chain grades is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals Australian Inventory of Industrial Chemicals (AIIC) Chemical Footprint

Project - Chemicals of High Concern List

hydrocarbon propellant is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals Australian Inventory of Industrial Chemicals (AIIC)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans

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

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

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

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

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans Stockholm Convention on Persistent Organic Pollutants - Annex A - Elimination

Chemical Footprint Project - Chemicals of High Concern List

National Inventory Status

National Inventory	Status	
Australia - AIIC / Australia Non-Industrial Use	Yes	
Canada - DSL	Yes	
Canada - NDSL	No (toluene; kaolin; chlorinated paraffin, long chain grades; hydrocarbon propellant)	
China - IECSC	Yes	
Europe - EINEC / ELINCS / NLP	Yes	
Japan - ENCS	No (kaolin)	
Korea - KECI	Yes	
New Zealand - NZIoC	Yes	
Philippines - PICCS	Yes	
USA - TSCA	Yes	
Taiwan - TCSI	Yes	
Mexico - INSQ	Yes	
Vietnam - NCI	Yes	
Russia - FBEPH	Yes	
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.	

SECTION 16 Other information

Revision Date	01/11/2019
Initial Date	28/03/2002

SDS Version Summary

Version	Date of Update	Sections Updated
4.1	27/06/2017	Acute Health (eye), Acute Health (inhaled), Acute Health (skin), Acute Health (swallowed), Advice to Doctor, Chronic Health, Classification, Engineering Control, Environmental, Exposure Standard, Fire Fighter (extinguishing media), Fire Fighter (fire/explosion hazard), Fire Fighter (fire incompatibility), First Aid (eye), First Aid (inhaled), First Aid (swallowed), Personal Protection (other), Personal Protection (Respirator), Personal Protection (eye), Personal Protection (hands/feet), Physical Properties, Spills (major), Storage (storage incompatibility), Storage (storage requirement), Supplier Information, Synonyms, Toxicity and Irritation (Other), Use
6.1	01/11/2019	One-off system update. NOTE: This may or may not change the GHS classification

Other information

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

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STEL: Short Term Exposure Limit

TEEL: Temporary Emergency Exposure Limit。

IDLH: Immediately Dangerous to Life or Health Concentrations

ES: Exposure Standard OSF: Odour Safety Factor

NOAEL :No Observed Adverse Effect Level LOAEL: Lowest Observed Adverse Effect Level

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

AIIC: Australian Inventory of Industrial Chemicals

DSL: Domestic Substances List

NDSL: Non-Domestic Substances List

IECSC: Inventory of Existing Chemical Substance in China EINECS: European INventory of Existing Commercial chemical Substances ELINCS: European List of Notified Chemical Substances

NLP: No-Longer Polymers

ENCS: Existing and New Chemical Substances Inventory

KECI: Korea Existing Chemicals Inventory NZIoC: New Zealand Inventory of Chemicals

PICCS: Philippine Inventory of Chemicals and Chemical Substances

TSCA: Toxic Substances Control Act TCSI: Taiwan Chemical Substance Inventory

INSQ: Inventario Nacional de Sustancias Químicas

NCI: National Chemical Inventory

FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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