

UltraColor Survey Spray – Non Fluoro Colours

Chemwatch Hazard Alert Code: 4

Issue Date: 27/06/2017 Print Date: 05/12/2017 L.GHS.AUS.EN

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

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

Product Identifier

Chemwatch: 4503-75

Product name	Ultracolor Aerosol Survey Marker-Non 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

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

Details of the supplier of the safety data sheet

Registered company name	Zeus Chemical Products
Address	3 Anderson Place South Windsor NSW 2756 Australia
Telephone	+61 2 4577 4866
Fax	+61 2 4577 6919
Website	www.ultracolor.com.au
Email	admin@ultracolor.com.au

Emergency telephone number

Association / Organisatio	Not Available	
Emergency telephone number	+61 2 4577 4866 (Mon-Fri, 8am-5pm)	
Other emergency telephon number	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]	Gas under Pressure (Compressed gas), Acute Toxicity (Oral) Category 4, Acute Toxicity (Dermal) Category 5, Acute Toxicity (Inhalation) Category 5, Skin Corrosion/Irritation Category 2, Serious Eye Damage Category 1, Reproductive Toxicity Category 2, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), 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 DANGE

Hazard statement(s)

nazara otatomom(o)	
H280	Contains gas under pressure; may explode if heated.
H302	Harmful if swallowed.
H313	May be harmful in contact with skin
H333	May be harmful if inhaled.
H315	Causes skin irritation.
H318	Causes serious eye damage.
H361	Suspected of damaging fertility or the unborn child.
H335	May cause respiratory irritation.

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H336	May cause drowsiness or dizziness.
H373	May cause damage to organs through prolonged or repeated exposure.
Precautionary statement(s) Pr	evention
P201	Obtain special instructions before use.

P201	Obtain special instructions before use.
P260	Do not breathe dust/fume/gas/mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P281	Use personal protective equipment as required.
P270	Do not eat, drink or smoke when using this product.

Precautionary statement(s) Response

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.
P310	Immediately call a POISON CENTER or doctor/physician.
P362	Take off contaminated clothing and wash before reuse.
P304+P312	IF INHALED: Call a POISON CENTER or doctor/physician if you feel unwell.
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	P405 Store locked up.	
P410+P403	Protect from sunlight. Store in a well-ventilated place.	
P403+P233	Store in a well-ventilated place. Keep container tightly closed.	

Precautionary statement(s) Disposal

Dispose of contents/container in accordance with local regulations.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
471-34-1	30-60	calcium carbonate
108-88-3	10-30	<u>toluene</u>
1332-58-7	<10	kaolin
Not Available	<10	hydrocarbon resin
Not Available	<10	pigments unregulated
68476-85-7.	30-60	hydrocarbon propellant
		NOTE: Manufacturer has supplied full ingredient
		information to allow CHEMWATCH assessment.
		Less than 0.1% benzene content

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	If aerosols come in contact with the eyes:
Skin Contact	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. DO NOT use solvents. Seek medical attention in the event of irritation.
Inhalation	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.

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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.
- Figure phrine (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):

Sampling Time Comments o-Cresol in urine 0.5 mg/L End of shift 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	Fire Incompatibility • Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result			
Advice for firefighters				
Fire Fighting	Alert Fire Brigade and tell them location and nature of hazard. 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. 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	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). Combustion products include: , carbon monoxide (CO) , other pyrolysis products typical of burning organic material. Contains low boiling substance: Closed containers may rupture due to pressure buildup under fire conditions.			

SECTION 6 ACCIDENTAL RELEASE MEASURES

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Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Methods and material for cont	annient 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	1
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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. 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)

Source Ingredient	Material name	TWA	STEL	Peak	Notes
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Australia Exposure Standards	calcium carbonate	Calcium carbonate	10 mg/m3	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 (liquified petroleum gas)	1800 mg/m3 / 1000 ppm	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
calcium carbonate	Limestone; (Calcium carbonate; Dolomite)	45 mg/m3	500 mg/m3	3,000 mg/m3
calcium carbonate	Carbonic acid, calcium salt	45 mg/m3	210 mg/m3	1,300 mg/m3
toluene	Toluene	Not Available	Not Available	Not Available
hydrocarbon propellant	Liquified petroleum gas; (L.P.G.)	65,000 ppm	2.30E+05 ppm	4.00E+05 ppm

Ingredient	Original IDLH	Revised IDLH
calcium carbonate	Not Available	Not Available
toluene	500 ppm	Not Available
kaolin	Not Available	Not Available
hydrocarbon resin	Not Available	Not Available
pigments unregulated	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 calcium carbonate:

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.

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. Foetbotxicity 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

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.

The basic types of engineering controls are:

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

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

Employers may need to use multiple types of controls to prevent employee overexposure.

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.

Appropriate engineering controls

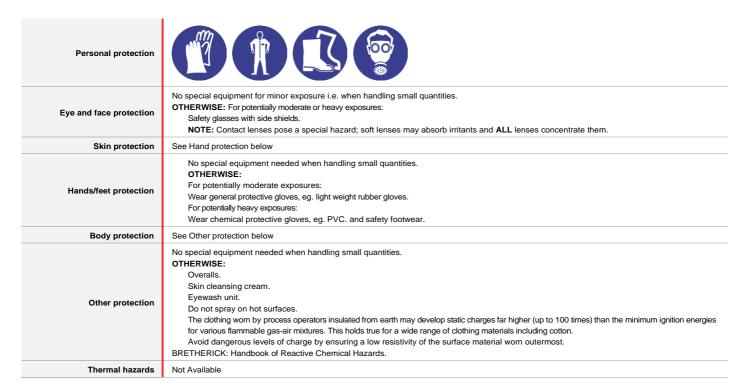
Type of Contaminant:	Speed:
aerosols, (released at low velocity into zone of active generation)	0.5-1 m/s
direct spray, spray painting in shallow booths, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)

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.

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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	СРІ
BUTYL	С
CPE	С
NEOPRENE	С
NEOPRENE/NATURAL	С
NITRILE	С
NITRILE+PVC	С
PE/EVAL/PE	С
PVA	С
PVC	С
SARANEX-23	С
SARANEX-23 2-PLY	С
TEFLON	С
VITON	С
VITON/CHLOROBUTYL	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.

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-fac

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

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. Liquid with aromatic solvent odour; does not mix with water.		
Physical state	Liquid	Relative density (Water = 1)	Not Available

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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 BuAC = 1	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)	>1	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. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination

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. 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.

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

Inhaled

 $\label{lem:common} \textbf{Common, generalised symptoms associated with toxic gas inhalation include:} \\$

- 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), and abdominal pain. 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. 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

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.

Symptoms of asphyxia (suffocation) may include headache, dizziness, shortness of breath, muscular weakness, drowsiness and ringing in the ears. If the asphyxia is allowed to progress, there may be nausea and vomiting, further physical weakness and unconsciousness and, finally, convulsions, coma and death. Significant concentrations of the non-toxic gas reduce the oxygen level in the air. As the amount of oxygen is reduced from 21 to 14 volume %, the pulse rate accelerates and the rate and volume of breathing increase. The ability to maintain attention and think clearly is diminished and muscular coordination is somewhat disturbed. As oxygen decreases from 14-10% judgement becomes faulty; severe injuries may cause no pain. Muscular exertion leads to rapid fatigue. Further reduction to 6% may produce nausea and vomiting and the ability to move may be lost. Permanent brain damage may result even after resuscitation at exposures to this lower oxygen level. Below 6% breathing is in gasps and convulsions may occur. Inhalation of a mixture containing no oxygen may result in unconsciousness from the first breath and death will follow in a few minutes.

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

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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.

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

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.

Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result.

Signs and symptoms of chemical (aspiration) pneumonitis may include coupling gasning choking burning of the mouth difficult breathing and h

Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cvanosis).

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 lethal chemical pneumonitis

Skin Contact

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. Comeal 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. 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.

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.

Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.

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.

Chronic

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.

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

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Ultracolor Aerosol Survey Marker Non Fluoro

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".

Jitracolor Non-Fluoro Aerosol	TOXICITY	IRRITATION
Survey Marker	Not Available	Not Available
	TOXICITY	IRRITATION
calcium carbonate	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 0.75 mg/24h - SEVERE
	Oral (rat) LD50: >2000 mg/kg ^[1]	Skin (rabbit): 500 mg/24h-moderate
	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
toluene	Oral (rat) LD50: 636 mg/kg ^[2]	Eye (rabbit):100 mg/30sec - mild
		Skin (rabbit):20 mg/24h-moderate
		Skin (rabbit):500 mg - moderate
	TOXICITY	IRRITATION
kaolin	Not Available	Not Available
	TOXICITY	IRRITATION
hydrocarbon propellant	Inhalation (rat) LC50: 84.684 mg/l15 min ^[1]	Not Available
	Inhalation (rat) LC50: 90.171125 mg/l15 min ^[1]	
Legend:	4. Value obtained from Frigure FCUA Positored Substances As	cute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified

CALCIUM CARBONATE

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

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

data extracted from RTECS - Register of Toxic Effect of chemical Substances

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 endogint 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:

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).

HYDROCARBON PROPELLANT

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) \\ = C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) \\ = C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) \\ = C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) \\ = C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) \\ = C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) \\ = C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) \\ = C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) \\ = C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) \\ = C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) \\ = C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) \\ = C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) \\ = C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) \\ = C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) \\ = C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) \\ = C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) \\ = C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) \\ = C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) \\ = C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) \\ = C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) \\ = C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) \\ = C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) \\ = C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) \\ = C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) \\ = C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,000 \ ppm) > C1-C4 \ HCs \ (LOAEL = 9,0$ to be 100% isobutane) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen)

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Ultracolor Non-Fluoro Aerosol Survey Marker & 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 symptons within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to sever 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 (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

Ultracolor Non-Fluoro Aerosol Survey Marker & HYDROCARBON PROPELL ANT

No significant acute toxicological data identified in literature search.

For toluene:

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 autopsv

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

Ultracolor Non-Fluoro Aerosol Survey 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 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.

CALCIUM CARBONATE & **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.

Acute Toxicity	✓	Carcinogenicity	0
Skin Irritation/Corrosion	✓	Reproductivity	✓
Serious Eye Damage/Irritation	✓	STOT - Single Exposure	✓
Respiratory or Skin sensitisation	0	STOT - Repeated Exposure	~
Mutagenicity	0	Aspiration Hazard	0

Leaend:

★ - Data available but does not fill the criteria for classification

- Data available to make classification

Data Not Available to make classification

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Toxicity

Ultracolor Non-Fluoro Aerosol Survey Marker	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURC
	Not Available	Not Available	Not Available	Not Available	Not Available
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURC
	LC50	96	Fish	>56000mg/L	4
calcium carbonate	EC50	72	Algae or other aquatic plants	>14mg/L	2
	NOEC	72	Algae or other aquatic plants	14mg/L	2
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURC
	LC50	96	Fish	0.0073mg/L	4
talaana	EC50	48	Crustacea	3.78mg/L	5
toluene	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
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURC
kaolin	Not Available	Not Available	Not Available	Not Available	Not Availabl
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURC
hydrocarbon propellant	Not Available	Not Available	Not Available	Not Available	Not Availabl

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.

(Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data

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.

Anthrocene 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 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)

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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)

Bioaccumulative potential

Ingredient	Bioaccumulation
toluene	LOW (BCF = 90)

Mobility in soil

Ingredient	Mobility
toluene	LOW (KOC = 268)

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



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 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; A1 A145 A167 A802 203 150 kg 203; Forbidden 75 kg; Forbidden Y203; Forbidden 30 kg G; Forbidden	

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Sea transport (IMDG-Code / GGVSee)

UN number	1950		
UN proper shipping name	AEROSOLS		
Transport hazard class(es)	IMDG Class 2 IMDG Subrisk N	.1 lot 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 1000ml	

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

CALCIUM CARBONATE(471-34-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards Australia Inventory of Chemical Substances (AICS)

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)

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	Υ
Canada - DSL	Y
Canada - NDSL	N (toluene; kaolin; hydrocarbon propellant)
China - IECSC	Υ
Europe - EINEC / ELINCS / NLP	Υ
Japan - ENCS	N (kaolin; hydrocarbon propellant)
Korea - KECI	Υ
New Zealand - NZIoC	Υ
Philippines - PICCS	Υ
USA - TSCA	Υ
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
calcium carbonate	471-34-1, 13397-26-7, 15634-14-7, 1317-65-3, 72608-12-9, 878759-26-3, 63660-97-9, 459411-10-0, 198352-33-9, 146358-95-4
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

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

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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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