

Ultracolor Survey / Linemarking Aerosol Paint

Zeus Chemical Products

Chemwatch: 11424

Version No: 7.1

Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Chemwatch Hazard Alert Code: 4

Issue Date: 10/03/2023

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

Product Identifier

Product name	Ultracolor Survey / Linemarking Aerosol Paint
Chemical Name	Not Applicable
Synonyms	white aerosol line marking 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

Relevant identified uses	Line marking paint. Applied as a hand held aerosol. Application is by spray atomisation from a hand held aerosol pack Use according to manufacturer's directions.
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Details of the manufacturer or 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 / Organisation	Zeus Chemical Products	CHEMWATCH EMERGENCY RESPONSE (24/7)
Emergency telephone numbers	+61 2 4577 4866 (Mon-Fri, 8am-5pm)	+61 1800 951 288
Other emergency telephone numbers	Not Available	+61 3 9573 3188

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

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 2A, Acute Toxicity (Inhalation) Category 4, 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

Ultracolor Survey / Linemarking Aerosol Paint

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.
H315	Causes skin irritation.
H319	Causes serious eye irritation.
H332	Harmful if inhaled.
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. If more than 15 mins from Doctor, INDUCE VOMITING (if conscious).
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P337+P313	If eye irritation persists: Get medical advice/attention.
P301+P312	IF SWALLOWED: Call a POISON CENTER/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.
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SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
13463-67-7	30-60	<u>titanium dioxide</u>
108-88-3	10-30	<u>toluene</u>
1332-58-7	1-10	<u>kaolin</u>
64742-16-1	1-10	<u>hydrocarbon resin, postpolymerised with maleic anhydride</u>
Not Available	1-10	vinyl toluene acrylate resin unspecified
63449-39-8	1-10	<u>chlorinated paraffin, long chain grades</u>
Not Available		propellant as
68476-85-7	10-30	<u>hydrocarbon propellant</u>
Not Available		No other ingredient information supplied.

Legend: 1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	<p>If aerosols come in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Immediately hold the eyelids apart and flush the eye continuously for at least 15 minutes with fresh running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If solids or aerosol mists are deposited upon the skin:</p> <ul style="list-style-type: none"> ▶ 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	<p>If aerosols, fumes or combustion products are inhaled:</p> <ul style="list-style-type: none"> ▶ Remove to fresh air. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ If breathing is shallow or has stopped, ensure clear airway and apply resuscitation, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor.
Ingestion	<ul style="list-style-type: none"> ▶ Avoid giving milk or oils. ▶ Avoid giving alcohol. ▶ 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.

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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	<ul style="list-style-type: none"> Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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Advice for firefighters

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

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	<ul style="list-style-type: none"> 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.
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	<ul style="list-style-type: none">▶ Undamaged cans should be gathered and stowed safely.
Major Spills	<ul style="list-style-type: none">▶ 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	<ul style="list-style-type: none">▶ Avoid all personal contact, including inhalation.▶ Wear protective clothing when risk of exposure occurs.▶ Use in a well-ventilated area.▶ Prevent concentration in hollows and sumps.▶ DO NOT enter confined spaces until atmosphere has been checked.▶ Avoid smoking, naked lights or ignition sources.▶ Avoid contact with incompatible materials.▶ When handling, DO NOT eat, drink or smoke.▶ DO NOT incinerate or puncture aerosol cans.▶ DO NOT spray directly on humans, exposed food or food utensils.▶ Avoid physical damage to containers.▶ Always wash hands with soap and water after handling.▶ Work clothes should be laundered separately.▶ Use good occupational work practice.▶ Observe manufacturer's storage and handling recommendations contained within this SDS.▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	<ul style="list-style-type: none">▶ 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	<ul style="list-style-type: none">▶ Aerosol dispenser.▶ Check that containers are clearly labelled.
Storage incompatibility	<ul style="list-style-type: none">▶ 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	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.

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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	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
titanium dioxide	30 mg/m3	330 mg/m3	2,000 mg/m3
toluene	Not Available	Not Available	Not Available
hydrocarbon propellant	65,000 ppm	2.30E+05 ppm	4.00E+05 ppm

Ingredient	Original IDLH	Revised IDLH
titanium dioxide	5,000 mg/m3	Not Available
toluene	500 ppm	Not Available
kaolin	Not Available	Not Available
hydrocarbon resin, postpolymerised with maleic anhydride	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.

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."

May act as a simple asphyxiants; these are gases which, when present in high concentrations, reduce the oxygen content in air below that required to support breathing, consciousness and life; loss of consciousness, with death by suffocation may rapidly occur in an oxygen deficient atmosphere.

CARE: Most simple asphyxiants are odourless or possess low odour and there is no warning on entry into an oxygen deficient atmosphere. If there is any doubt, oxygen content can be checked simply and quickly. It may not be appropriate to only recommend an exposure standard for simple asphyxiants rather it is essential that sufficient oxygen be maintained. Air normally has 21 percent oxygen by volume, with 18 percent regarded as minimum under normal atmospheric pressure to maintain consciousness / life. At pressures significantly higher or lower than normal atmospheric pressure, expert guidance should be sought.

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 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.
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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.

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.

Individual protection measures, such as personal protective equipment



Eye and face protection

- ▶ Safety glasses with side shields.
- ▶ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent]
- ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].

Skin protection

See Hand protection below

Hands/feet protection

- ▶ No special equipment needed when handling small quantities.
- ▶ OTHERWISE:
- ▶ For potentially moderate exposures:
- ▶ Wear general protective gloves, eg. light weight rubber gloves.
- ▶ For potentially heavy exposures:
- ▶ Wear chemical protective gloves, eg. PVC. and safety footwear.

Body protection

See Other protection below

Other protection

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.

BRETHERRICK: 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:

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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.

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

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	AX-AUS	-	AX-PAPR-AUS / Class 1
up to 50 x ES	-	AX-AUS / Class 1	-
up to 100 x ES	-	AX-2	AX-PAPR-2 ^

^ - Full-face

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

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

Appearance	White or coloured liquid with a strong 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)	0.9-1.0 approx
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 (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	-81 propellant	Taste	Not Available
Evaporation rate	Fast	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)	>50
Vapour pressure (kPa)	UNDER PRESSURE	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (1%)	Not Applicable
Vapour density (Air = 1)	> 2	VOC g/L	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ▶ Elevated temperatures. ▶ Presence of open flame.

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	<ul style="list-style-type: none"> ▶ Product is considered stable. ▶ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

	<p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p> <p>Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>The acute toxicity of inhaled alkylbenzene is best described by central nervous system depression. These compounds may also act as general anaesthetics. Whole body symptoms of poisoning include light-headedness, nervousness, apprehension, a feeling of well-being, confusion, dizziness, drowsiness, ringing in the ears, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness, depression of breathing, and arrest. Heart stoppage may result from cardiovascular collapse. A slow heart rate and low blood pressure may also occur.</p> <p>Alkylbenzenes are not generally toxic except at high levels of exposure. Their breakdown products have low toxicity and are easily eliminated from the body.</p> <p>Common, generalised symptoms associated with toxic gas inhalation include:</p> <ul style="list-style-type: none"> ▶ central nervous system effects such as depression, headache, confusion, dizziness, progressive stupor, coma and seizures; ▶ respiratory system complications may include acute pulmonary oedema, dyspnoea, stridor, tachypnoea, bronchospasm, wheezing and other reactive airway symptoms, and respiratory arrest; ▶ cardiovascular effects may include cardiovascular collapse, arrhythmias and cardiac arrest; ▶ gastrointestinal effects may also be present and may include mucous membrane irritation, nausea and vomiting (sometimes bloody), and abdominal pain. <p>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.</p> <p>WARNING: Intentional misuse by concentrating/inhaling contents may be lethal.</p> <p>Hydrocarbons may sensitise the heart to adrenalin and other circulatory catecholamines; as a result cardiac arrhythmias and ventricular fibrillation may occur. Abrupt collapse may produce traumatic injury. Central nervous system (CNS) depression may be evident early. Symptoms of moderate poisoning may include giddiness, headache, dizziness and nausea. Serious poisonings may result in respiratory depression and may be fatal.</p> <p>The paraffin gases C1-4 are practically non-toxic below their lower flammability limits (18000-50000 ppm). Above this level, incidental effects include CNS depression and irritation but these are reversible upon cessation of the exposure. The C3 and iso-C5 hydrocarbons show increasing narcotic properties; branching of the chain also enhances the effect. The C4 hydrocarbons appear to be more highly neurotoxic than the C3 and C5 members. Several fatalities due to voluntary inhalation of butane have been reported, possibly due to central, respiratory and circulatory effects resulting from anaesthesia, laryngeal oedema, chemical pneumonia or the combined effects of cardiac toxicity and increased sympathomimetic effects.</p> <p>Inhalation of petroleum gases may produce narcosis, due in part to olefinic impurities. Displacement of oxygen in the air may cyanosis. If present in sufficient quantity these gases may reduce the oxygen level to below 18% producing asphyxiation. Symptoms include rapid respiration, mental dullness, lack of coordination, poor judgement, nausea and vomiting. The onset of cyanosis may lead to unconsciousness and death.</p>
Inhaled	
Ingestion	<p>Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.</p> <p>Not normally a hazard due to physical form of product.</p> <p>Considered an unlikely route of entry in commercial/industrial environments</p>

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	<p>Considered an unlikely route of entry in commercial/industrial environments. The liquid may produce gastrointestinal discomfort and may be harmful if swallowed. Ingestion may result in nausea, pain and vomiting. Vomit entering the lungs by aspiration may cause potentially lethal chemical pneumonitis</p>
Skin Contact	<p>Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. Spray mist may produce discomfort</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p> <p>The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either</p> <ul style="list-style-type: none"> ▶ 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. <p>Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p>
Eye	<p>Direct contact with the eye may not cause irritation because of the extreme volatility of the gas; however concentrated atmospheres may produce irritation after brief exposures..</p> <p>Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p> <p>The liquid produces a high level of eye discomfort and is capable of causing pain and severe conjunctivitis. Corneal injury may develop, with possible permanent impairment of vision, if not promptly and adequately treated.</p>
Chronic	<p>Harmful: danger of serious damage to health by prolonged exposure through inhalation.</p> <p>Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.</p> <p>Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>Principal route of occupational exposure to the gas is by inhalation.</p> <p>The health hazards associated with bentonite, kaolin, and common clay, which are commercially important clay products, as well as the related phyllosilicate minerals montmorillonite, kaolinite, and illite, have an extensive literature. Fibrous clay minerals, such as sepiolite, attapulgite, and zeolites, have a separate literature.</p> <p>The biological effects of clay minerals are influenced by their mineral composition and particle size. The decreasing rank order of the potencies of quartz, kaolinite, and montmorillonite to produce lung damage is consistent with their known relative active surface areas and surface chemistry.</p> <p>Clays are chemically all described as aluminosilicates; these are further classified as bentonite, kaolin and common clays. Bentonite is a rock formed of highly colloidal and plastic clays composed mainly of montmorillonite, a clay mineral of the smectite group.</p> <p>Kaolin or china clay is a mixture of different minerals. Its main component is kaolinite; in addition, it frequently contains quartz, mica, feldspar, illite, and montmorillonite.</p> <p>The main components of common clay and shale are illite and chlorite. Illite is also a component of ball clays. Illite closely resembles micas,</p> <p>From the limited data available from studies on bentonite-exposed persons, retained montmorillonite appears to effect only mild nonspecific tissue changes, which are similar to those that have been described in the spectrum of changes of the "small airways mineral dust disease" (nodular peribronchiolar dust accumulations containing refractile material [montmorillonite] in association with limited interstitial fibrosis). In some of the studies, radiological abnormalities have also been reported</p> <p>Long-term occupational exposures to bentonite dust may cause structural and functional damage to the lungs. However, available data are inadequate to conclusively establish a dose-response relationship or even a cause-and-effect relationship due to limited information on period and intensity of exposure and to confounding factors, such as exposure to silica and tobacco smoke.</p> <p>Long-term exposure to kaolin may lead to a relatively benign pneumoconiosis, in an exposure-related fashion. known as kaolinosiis. Deterioration of lung function has been observed only in cases with prominent radiological alterations. Based on data from china clay workers in the United Kingdom, it can be very roughly estimated that kaolin is at least an order of magnitude less potent than quartz.. Clearcut deterioration of respiratory function and related symptoms have been reported only in cases with prominent radiological findings.</p> <p>The composition of the clay - i.e., quantity and quality of minerals other than kaolinite — is an important determinant of the effects. Bentonite, kaolin, and other clays often contain quartz, and exposure to quartz is causally related to silicosis and lung cancer. Statistically significant increases in the incidence of or mortality from chronic bronchitis and pulmonary emphysema have been reported after exposure to quartz.</p> <p>The removal of clay particles from the lungs takes place by solubilisation in situ and by physical clearance.</p> <p>In humans, there was a rapid initial clearance of 8% and 40% of aluminosilicate particles that were, respectively, 1.9 and 6.1 um in aerodynamic diameter from the lung region over 6 days. Thereafter, 4% and 11% of the two particle sizes were removed following a halftime of 20 days, and the rest with half-times of 330 and 420 days.</p> <p>Ultrafine particles (<100 nm) have a high deposition in the nasal area; they can penetrate the alveolar/capillary barrier.</p> <p>Epidemiological studies have indicated an increase in morbidity and mortality associated with an increase in airborne particulate matter, particularly in the ultrafine size range</p>

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An important determinant of the toxicity of clays is the content of quartz. The presence of quartz in the clays studied hampers reliable independent estimation of the fibrogenicity of other components of clays.

Single intratracheal injection into rodents of bentonite and montmorillonite with low content of quartz produced dose- and particle size-dependent cytotoxic effects, as well as transient local inflammation, the signs of which included oedema and, consequently, increased lung weight. After high doses of intratracheal kaolin (containing 8-65% quartz), fibrosis has been described in some studies, whereas at lower kaolin doses, no fibrosis has been observed in the few available studies.

There are limited data on the effects of multiple exposures of experimental animals to montmorillonite or bentonite. Mice maintained on diets containing 10% or 25% bentonite but otherwise adequate to support normal growth displayed slightly reduced growth rates, whereas mice maintained on a similar diet with 50% bentonite showed minimal growth and developed fatty livers and eventually fibrosis of the liver and benign hepatomas.

In vitro studies of the effects of bentonite on a variety of mammalian cell types usually indicated a high degree of cytotoxicity. Concentrations below 1.0 mg/ml of bentonite and montmorillonite particles less than 5 um in diameter caused membrane damage and even cell lysis, as well as functional changes in several types of cells.

No adequate studies are available on the carcinogenicity of bentonite. In an inhalation study and in a study using intrapleural injection, kaolin did not induce tumours in rats. No studies are available on the genotoxicity of clays.

Single, very limited studies did not demonstrate developmental toxicity in rats after oral exposure to bentonite or kaolin.

Chronic dust inhalation of kaolin, as experienced in mineral extraction, has caused kaolinosis with heavy lung marking, emphysema, and nodular pneumoconiosis.

Evidence of kaolinosis (pneumoconiosis) was found in 9% of 553 Cornish china clay workers who had been exposed to kaolin dust for periods exceeding 5 years, whereas no kaolinosis was observed in workers exposed for less than 5 years. Workers in more heavily exposed jobs of milling, bagging and loading showed a prevalence of kaolinosis rising from 6% in those within between 5 and 15 years exposure to 23% in those exposed for more than 15 years. Workers intermittently and less heavily exposed in the older, outdated drying plants required 25 years of massive exposure before reaching the highest prevalence of 17%. Massive fibrosis was seen in four workers, and six workers needed antituberculosis chemotherapy. Preventative measures instituted include preemployment chest examination and approaches to the problem of dust control.

Sheer, G.; Brit. Jnl. Ind. Med. 21, pp 218-225, 1964

On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.

Long term exposure to the dusts of titanium and several of its compounds produces chronic lung disease (fibrosis) in animals. Radiological evidence exists amongst titanium dioxide workers suggesting chronic lung changes which resemble a slight form of silicosis. Workers chronically exposed to titanium or titanium dioxide dusts show a high incidence of chronic bronchitis (endobronchitis and peribronchitis). Early stages of this disease are characterised by impaired pulmonary respiration and ventilatory capacity and by reduced blood alkalinity. Cardiac changes characteristic of pulmonary disease (with hypertrophy of the right auricle) have also been observed amongst workers.

Titanium employed in implants has provoked immune responses which occur locally as metallosis and systemically as raised serum levels of activated T-lymphocytes. Some concern has been expressed about the potential for generating bone-resorbing mediators associated with titanium wear-debris.

The largest of the cohort studies was among white male production workers in the titanium dioxide industry in six European countries. The study indicated a slightly increased risk for lung cancer compared with the general population. However, there was no evidence of an exposure-response relationship within the cohort. No increase in the mortality rates for kidney cancer was found when the cohort was compared with the general population, but there was a suggestion of an exposure-response relationship in internal analyses. The other cohort studies, both of which were conducted in the USA, did not report an increased risk for lung cancer or cancer at any other site; no results for kidney cancer were reported, presumably because there were few cases.

One population-based case-control study conducted in Montreal did not indicate an increased risk for lung or kidney cancer. In summary, the studies do not suggest an association between occupational exposure to titanium dioxide as it occurred in recent decades in western Europe and North America and risk for cancer.

All the studies had methodological limitations; misclassification of exposure could not be ruled out. None of the studies was designed to assess the impact of particle size (fine or ultrafine) or the potential effect of the coating compounds on the risk for lung cancer.

An increased incidence of lung adenomas in rats of both sexes and of cystic keratinising lesions, diagnosed as squamous cell carcinomas in female rats, was seen in animals subject to high doses of inhaled titanium dioxide. Intratracheal delivery of titanium dioxide in combination with benz[a]pyrene produced an increase in benign and malignant tumours of the larynx, trachea and lungs in hamsters.

Squamous cell carcinomas developed after exposure to 250 mg/m3 for 6 hours/day, 5 days/week for 2 years in rats; the type of carcinoma that developed was considered to be a unique experimentally induced tumour and to be of questionable relevance for extrapolation of the results to humans. Given the extremely high level of dust in the lungs, the carcinomas were postulated to be the result of saturation of the normal pulmonary clearance mechanisms. At 50 mg/m3, massive accumulations of dust-laden macrophages, foamy dust cells and free particles were considered indicative of such overload.

Ultracolor Survey / Linemarking Aerosol Paint	TOXICITY	IRRITATION
	Not Available	Not Available
titanium dioxide	TOXICITY	IRRITATION
	dermal (hamster) LD50: >=10000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation (Rat) LC50: >2.28 mg/4h ^[1]	Skin (human): 0.3 mg /3D (int)-mild *
	Oral (Rat) LD50: >=2000 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
toluene	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

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	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]
kaolin	TOXICITY	IRRITATION
	Not Available	Not Available
hydrocarbon resin, postpolymerised with maleic anhydride	TOXICITY	IRRITATION
	Oral (Rat) LD50: >2000 mg/kg ^[1]	Not Available
chlorinated paraffin, long chain grades	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >10000 mg/kg ^[2]	Not Available
	Oral (Mouse) LD50: 21800 mg/kg ^[2]	
hydrocarbon propellant	TOXICITY	IRRITATION
	Inhalation (Rat) LC50: 658 mg/l4h ^[2]	Not Available
Legend:	1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances	

TITANIUM DIOXIDE	<p>* IUCLID</p> <p>Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.</p> <p>Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. 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. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.</p>
TOLUENE	<p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p>
KAOLIN	<p>for bentonite clays:</p> <p>Bentonite (CAS No. 1302-78-9) consists of a group of clays formed by crystallisation of vitreous volcanic ashes that were deposited in water.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>Ingestion of bentonite without adequate liquids may result in intestinal obstruction in humans.</p> <p>Hypokalaemia and microcytic iron-deficiency anaemia may occur in patients after repeat doses of clay. Chronic ingestion has been reported to cause myositis.</p>
HYDROCARBON RESIN, POSTPOLYMERISED WITH MALEIC ANHYDRIDE	Oral (-) LD50: 7000-10000 mg/kg Nil reported. [Manufacturer]
CHLORINATED PARAFFIN, LONG CHAIN GRADES	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.

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	<p>The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
HYDROCARBON PROPELLANT	<p>for Petroleum Hydrocarbon Gases:</p> <p>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.</p> <p>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</p> <p>Acute toxicity: No acute toxicity LC50 values have been derived for the C1 -C4 and C5- C6 hydrocarbon (HC) fractions because no mortality was observed at the highest exposure levels tested (~ 5 mg/l) for these petroleum hydrocarbon gas constituents. The order of acute toxicity of petroleum hydrocarbon gas constituents from most to least toxic is: C5-C6 HCs (LC50 > 1063 ppm) > C1-C4 HCs (LC50 > 10,000 ppm) > benzene (LC50 = 13,700 ppm) > butadiene (LC50 = 129,000 ppm) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen).</p> <p>Repeat dose toxicity: With the exception of the asphyxiant gases, repeated dose toxicity has been observed in individual selected petroleum hydrocarbon gas constituents. Based upon LOAEL values, the order of order of repeated-dose toxicity of these constituents from most toxic to the least toxic is: Benzene (LOAEL .>=10 ppm) >C1-C4 HCs (LOAEL = 5,000 ppm; assumed to be 100% 2-butene) > C5-C6 HCs (LOAEL = 6,625 ppm) > butadiene (LOAEL = 8,000 ppm) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen).</p> <p>Genotoxicity:</p> <p>In vitro: The majority of the Petroleum Hydrocarbon Gases Category components are negative for <i>in vitro</i> genotoxicity. The exceptions are: benzene and 1,3-butadiene, which are genotoxic in bacterial and mammalian <i>in vitro</i> test systems.</p> <p>In vivo: The majority of the Petroleum Hydrocarbon Gases Category components are negative for <i>in vivo</i> genotoxicity. The exceptions are benzene and 1,3-butadiene, which are genotoxic in <i>in vivo</i> test systems</p> <p>Developmental toxicity: Developmental effects were induced by two of the petroleum hydrocarbon gas constituents, benzene and the C5 -C6 hydrocarbon fraction. No developmental toxicity was observed at the highest exposure levels tested for the other petroleum hydrocarbon gas constituents tested for this effect. The asphyxiant gases have not been tested for developmental toxicity. Based on LOAEL and NOAEL values, the order of acute toxicity of these constituents from most to least toxic is: Benzene (LOAEL = 20 ppm) > butadiene (NOAEL .>=1,000 ppm) > C5-C6 HCs (LOAEL = 3,463 ppm) > C1-C4 HCs (NOAEL .>=5,000 ppm; assumed to be 100% 2-butene) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen).</p> <p>Reproductive toxicity: Reproductive effects were induced by only two petroleum hydrocarbon gas constituents, benzene and isobutane (a constituent of the 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)</p>
Ultracolor Survey / Linemarking Aerosol Paint & TITANIUM DIOXIDE & KAOLIN & HYDROCARBON PROPELLANT	<p>No significant acute toxicological data identified in literature search.</p>
Ultracolor Survey / Linemarking Aerosol Paint & TITANIUM DIOXIDE	<p>The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often 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.</p> <p>For titanium dioxide:</p> <p>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 size-dependent 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.</p> <p>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.</p> <p>No data were available on genotoxic effects in titanium dioxide-exposed humans.</p> <p>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-</p>

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	<p>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.</p> <p>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.</p> <p>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.</p> <p>Animal carcinogenicity data</p> <p>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.</p> <p>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.</p> <p>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.</p>
<p>Ultracolor Survey / Linemarking Aerosol Paint & CHLORINATED PARAFFIN, LONG CHAIN GRADES</p>	<p>NOTE: C12, 60% chlorinated paraffin [CAS RN 108171-26-2] is classified by IARC as Group 2B. Possibly carcinogenic to humans.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>The Regulatory regime in various countries differs with respect to chlorinated paraffins.</p> <p>In the USA, the short-chain (C12), 58% chlorine product has been classified and labelled as a carcinogen.</p> <p>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.</p> <p>All EU Member States are required to classify short chain chlorinated paraffins as Category 3 carcinogens.</p>
<p>Ultracolor Survey / Linemarking Aerosol Paint & TOLUENE</p>	<p>For toluene:</p> <p>Acute Toxicity</p> <p>Humans exposed to intermediate to high levels of toluene for short periods of time experience adverse central nervous system effects ranging from headaches to intoxication, convulsions, narcosis, and death. Similar effects are observed in short-term animal studies.</p> <p>Humans - Toluene ingestion or inhalation can result in severe central nervous system depression, and in large doses, can act as a narcotic. The ingestion of about 60 mL resulted in fatal nervous system depression within 30 minutes in one reported case. Constriction and necrosis of myocardial fibers, markedly swollen liver, congestion and haemorrhage of the lungs and acute tubular necrosis were found on autopsy.</p> <p>Central nervous system effects (headaches, dizziness, intoxication) and eye irritation occurred following inhalation exposure to 100 ppm toluene 6 hours/day for 4 days.</p> <p>Exposure to 600 ppm for 8 hours resulted in the same and more serious symptoms including euphoria, dilated pupils, convulsions, and nausea. Exposure to 10,000-30,000 ppm has been reported to cause narcosis and death.</p> <p>Toluene can also strip the skin of lipids causing dermatitis.</p> <p>Animals - The initial effects are instability and incoordination, lachrymation and 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.</p> <p>Subchronic/Chronic Effects:</p> <p>Repeat doses of toluene cause adverse central nervous system effects and can damage the upper respiratory system, the liver, and the kidney. Adverse effects occur as a result from both oral and the inhalation exposures. A reported lowest-observed-effect level in humans for adverse neurobehavioral effects is 88 ppm.</p> <p>Humans - Chronic occupational exposure and incidences of toluene abuse have resulted in hepatomegaly and liver function changes. It has also resulted in nephrotoxicity and, in one case, was a cardiac sensitiser and fatal cardiotoxin.</p> <p>Neural and cerebellar dystrophy were reported in several cases of habitual "glue sniffing." An epidemiological study in France on workers chronically exposed to toluene fumes reported leukopenia and neutropenia. Exposure levels were not given in the secondary reference; however, the average urinary excretion of hippuric acid, a metabolite of toluene, was given as 4 g/L compared to a normal level of 0.6 g/L.</p> <p>Animals - The major target organs for the subchronic/chronic toxicity of toluene are the nervous system, liver, and kidney. Depressed immune response has been reported in male mice given doses of 105 mg/kg/day for 28 days. Toluene in corn oil administered to F344 male and female rats by gavage 5 days/week for 13 weeks, induced prostration, hypoactivity, ataxia, piloerection, lachrymation, excess salivation, and body tremors at doses 2500 mg/kg. Liver, kidney, and heart weights were also increased at this dose and histopathologic lesions were seen in the liver, kidneys, brain and urinary bladder. The no-observed-</p>

Ultracolor Survey / Linemarking Aerosol Paint

	<p>adverse effect level (NOAEL) for the study was 312 mg/kg (223 mg/kg/day) and the lowest-observed-adverse effect level (LOAEL) for the study was 625 mg/kg (446 mg/kg/day) .</p> <p>Developmental/Reproductive Toxicity</p> <p>Exposures to high levels of toluene can result in adverse effects in the developing human foetus. Several studies have indicated that high levels of toluene can also adversely effect the developing offspring in laboratory animals.</p> <p>Humans - Variable growth, microcephaly, CNS dysfunction, attentional deficits, minor craniofacial and limb abnormalities, and developmental delay were seen in three children exposed to toluene in utero as a result of maternal solvent abuse before and during pregnancy</p> <p>Animals - Sternebral alterations, extra ribs, and missing tails were reported following treatment of rats with 1500 mg/m3 toluene 24 hours/day during days 9-14 of gestation. Two of the dams died during the exposure. Another group of rats received 1000 mg/m3 8 hours/day during days 1-21 of gestation. No maternal deaths or toxicity occurred, however, minor skeletal retardation was present in the exposed fetuses. CFLP Mice were exposed to 500 or 1500 mg/m3 toluene continuously during days 6-13 of pregnancy. All dams died at the high dose during the first 24 hours of exposure, however none died at 500 mg/m3. Decreased foetal weight was reported, but there were no differences in the incidences of skeletal malformations or anomalies between the treated and control offspring.</p> <p>Absorption - Studies in humans and animals have demonstrated that toluene is readily absorbed via the lungs and the gastrointestinal tract. Absorption through the skin is estimated at about 1% of that absorbed by the lungs when exposed to toluene vapor.</p> <p>Dermal absorption is expected to be higher upon exposure to the liquid; however, exposure is limited by the rapid evaporation of toluene .</p> <p>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 .</p> <p>Metabolism - The metabolites of inhaled or ingested toluene include benzyl alcohol resulting from the hydroxylation of the methyl group. Further oxidation results in the formation of benzaldehyde and benzoic acid. The latter is conjugated with glycine to yield hippuric acid or reacted with glucuronic acid to form benzoyl glucuronide. o-cresol and p-cresol formed by ring hydroxylation are considered minor metabolites</p> <p>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.</p>
TITANIUM DIOXIDE & CHLORINATED PARAFFIN, LONG CHAIN GRADES	<p>WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.</p>

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

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

SECTION 12 Ecological information

Toxicity

Ultracolor Survey / Linemarking Aerosol Paint	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
titanium dioxide	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	1.85-3.06mg/l	4
	BCF	1008h	Fish	<1.1-9.6	7
	EC50	72h	Algae or other aquatic plants	3.75-7.58mg/l	4
	EC50	48h	Crustacea	1.9mg/l	2
	NOEC(ECx)	672h	Fish	>=0.004mg/L	2
	EC50	96h	Algae or other aquatic plants	179.05mg/l	2
toluene	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	12.5mg/L	4
	LC50	96h	Fish	5-35mg/l	4
	EC50	48h	Crustacea	3.78mg/L	5
	NOEC(ECx)	168h	Crustacea	0.74mg/l	2

Continued...

Ultracolor Survey / Linemarking Aerosol Paint

	EC50	96h	Algae or other aquatic plants	>376.71mg/L	4
kaolin	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
hydrocarbon resin, postpolymerised with maleic anhydride	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	72h	Algae or other aquatic plants	<1mg/l	2
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
chlorinated paraffin, long chain grades	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	>0.011mg/L	4
	NOEC(ECx)	504h	Crustacea	~0.002mg/L	2
	EC50	72h	Algae or other aquatic plants	>3.2mg/l	2
	EC50	96h	Algae or other aquatic plants	>3.2mg/l	2
hydrocarbon propellant	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	24.11mg/l	2
	EC50(ECx)	96h	Algae or other aquatic plants	7.71mg/l	2
	EC50	96h	Algae or other aquatic plants	7.71mg/l	2
Legend:	Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data				

For Aromatic Substances Series:

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

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

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

The term chlorinated paraffins is usually taken to encompass a wide range of liquids and solids from C10 to >C24 and containing 30-72% chlorine content.

Properties differ significantly across this range and for this reason they are considered in three separate groups.

1. The C10-13 liquid products from 40-72% Cl₂ content
2. The C14-17, C18-20 and chlorinated paraffin wax liquids (average C25) from 40-60% Cl₂ content
3. The powdered chlorinated paraffin waxes of >69% Cl₂ content

Liquid grades of chlorinated paraffin are produced from paraffins and waxes while solid grades are produced from waxes with a 70-72% chlorine content.

Increasing chlorine content results in higher viscosity and density.

- ▶ Chlorinated paraffins have very low vapour pressure with the most volatile (C10-13 types) < 10-3 mbar. They are chemically stable but dehydrochlorinated on heating at high temperatures (for prolonged periods). Dehydrochlorination also occurs on prolonged exposure to light.
- ▶ All have low solubility in water but C10-13 types are significantly more soluble than other classes which show decreased solubility with increasing chain length.
- ▶ Studies confirm that they adsorb strongly onto suspended materials or sediments in an aqueous environment. True solutions (at the low solubility limit) do degrade without added reagents.
- ▶ Laboratory studies often fail to indicate biodegradation occurring, but longer term studies in biological effluent treatment plants do reveal substantial degradation and the undegraded residue is removed by adsorption onto biological sludge.
- ▶ The short chain grades have been shown in laboratory tests, to have toxic effects on fish and other forms of aquatic life after long-term exposure to concentrations close to their water solubility, and significantly higher than those found typically in the environment.
- ▶ Mid-chain forms show a significantly reduced spectrum of toxicity compared to short-chain grades, as would be expected from their lower bioaccumulation. No measurable short or long term toxicity has been found in studies with numerous species of fish. Only one of several aquatic invertebrate species that has been been tested showed any sensitivity but at levels higher than those found in the environment. Similarly, certain soil and sediment organisms are affected at levels of hundreds of parts per million (ppm) whilst others are affected at 1000s of ppm.
- ▶ Long chain grades, because of their large molecular size and their very low solubility, have shown no toxicity to fish and other forms of aquatic life at and above their solubility limit.

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 : 5.94;

Continued...

E-03BOD 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.

Atmospheric Fate: The majority of toluene evaporates to the atmosphere from the water and soil. 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 oxidized 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.

Terrestrial Fate: Toluene is moderately retarded by adsorption to soils rich in organic material, therefore, transport to ground water is dependent on 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 volatilized, biodegraded, or unaccounted for. In saturated deep soils with no soil-air phase, about 48% may be transported with flowing groundwater. In surface soil, volatilization to air is an important fate process for toluene. In the environment, biodegradation of toluene to carbon dioxide occurs with a typical half life of 1-7 days.

Aquatic Fate: An important fate process for toluene is volatilization, the rate of which depends on the amount of turbulence in the surface water. The volatilization 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.

Ecotoxicity: Bioaccumulation in the food chain is predicted to be low. Toluene has moderate acute toxicity to aquatic organisms. Toluene is, on the average, slightly toxic to fathead minnow, guppies and goldfish and not acutely toxic to bluegill or channel catfish and crab. Toluene, on the average, is slightly toxic to crustaceans specifically, shrimp species including grass shrimp and daggerblade grass shrimp. Toluene has a negative effect on green algae during their growth phase.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
titanium dioxide	HIGH	HIGH
toluene	LOW (Half-life = 28 days)	LOW (Half-life = 4.33 days)

Bioaccumulative potential

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

Mobility in soil

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

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none">▶ DO NOT allow wash water from cleaning or process equipment to enter drains.▶ It may be necessary to collect all wash water for treatment before disposal.▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.▶ Where in doubt contact the responsible authority.▶ 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.
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SECTION 14 Transport information

Labels Required

	
Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (ADG)

14.1. UN number or ID number	1950	
14.2. UN proper shipping name	AEROSOLS	
14.3. Transport hazard class(es)	Class	2.1
	Subsidiary Hazard	Not Applicable
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Special provisions	63 190 277 327 344 381
	Limited quantity	1000ml

Air transport (ICAO-IATA / DGR)

14.1. UN number	1950	
14.2. UN proper shipping name	Aerosols, flammable	
14.3. Transport hazard class(es)	ICAO/IATA Class	2.1
	ICAO / IATA Subsidiary Hazard	Not Applicable
	ERG Code	10L
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Special provisions	A145 A167 A802
	Cargo Only Packing Instructions	203
	Cargo Only Maximum Qty / Pack	150 kg
	Passenger and Cargo Packing Instructions	203
	Passenger and Cargo Maximum Qty / Pack	75 kg
	Passenger and Cargo Limited Quantity Packing Instructions	Y203
	Passenger and Cargo Limited Maximum Qty / Pack	30 kg G

Sea transport (IMDG-Code / GGVSee)

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

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

Not Applicable

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

Product name	Group
titanium dioxide	Not Available
toluene	Not Available
kaolin	Not Available
hydrocarbon resin, postpolymerised with maleic anhydride	Not Available

Product name	Group
chlorinated paraffin, long chain grades	Not Available
hydrocarbon propellant	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
titanium dioxide	Not Available
toluene	Not Available
kaolin	Not Available
hydrocarbon resin, postpolymerised with maleic anhydride	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

titanium dioxide is found on the following regulatory lists
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
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)
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 - Not Classified as Carcinogenic
kaolin is found on the following regulatory lists
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
hydrocarbon resin, postpolymerised with maleic anhydride is found on the following regulatory lists
Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
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
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
hydrocarbon propellant 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

Additional Regulatory Information

Not Applicable

National Inventory Status

Ultracolor Survey / Linemarking Aerosol Paint

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (toluene; kaolin; hydrocarbon resin, postpolymerised with maleic anhydride; 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:	<p>Yes = All CAS declared ingredients are on the inventory</p> <p>No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.</p>

SECTION 16 Other information

Revision Date	10/03/2023
Initial Date	07/04/2005

SDS Version Summary

Version	Date of Update	Sections Updated
6.1	01/11/2019	One-off system update. NOTE: This may or may not change the GHS classification
7.1	10/03/2023	Classification change due to full database hazard calculation/update.

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
- 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
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration

- AIIC: Australian Inventory of Industrial Chemicals
- DSL: Domestic Substances List
- NDSL: Non-Domestic Substances List
- IECSC: Inventory of Existing Chemical Substance in China

Continued...

Ultracolor Survey / Linemarking Aerosol Paint

- 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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