Fromm Packaging Metal Marking Texpen/Dalo - All Colours FROMM Packaging Australia Pty Ltd

Chemwatch: **5240-10** Version No: **5.1**

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

Chemwatch Hazard Alert Code: 3

Issue Date: 19/01/2023 Print Date: 19/01/2023 L.GHS.AUS.EN.E

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

Product Identifier		
Product name	Fromm Packaging Metal Marking Texpen/Dalo - All Colours	
Chemical Name	Not Applicable	
Synonyms	J3070 (Black), J2143 (Blue), Y916 (Green), A451M (Orange), J3076 (Red), J1694 (White), A419M (Yellow); Texpen - Fine, Medium and Broad; Dalo- Medium and Broad; Solvent based marker	
Proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)	
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 Solvent based marker.
Use according to manufacturer's directions.

Details of the manufacturer or supplier of the safety data sheet

Registered company name	FROMM Packaging Australia Pty Ltd	
Address	Unit 1, 6 Yatala Road Mount Kuring Gai NSW 2080 Australia	
Telephone	1800 023 890	
Fax	1800 246 551	
Website	www.fromm-pack.com.au	
Email	sales@fromm-pack.com.au	

Emergency telephone number

Association / Organisation	FROMM Packaging Australia Pty Ltd
Emergency telephone numbers	+61 733753477
Other emergency telephone numbers	+61 412 822 725

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable	
Classification [1]	Flammable Liquids Category 3, Aspiration Hazard Category 1, Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2A, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Carcinogenicity Category 1B, Hazardous to the Aquatic Environment Long-Term Hazard Category 2	
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI	

Label elements

Hazard pictogram(s)









Signal word

Dangei

Hazard statement(s)

H226	Flammable liquid and vapour.
H304	May be fatal if swallowed and enters airways.
H315	Causes skin irritation.
H319	Causes serious eye irritation.
H335	May cause respiratory irritation.
H336	May cause drowsiness or dizziness.

Chemwatch: 5240-10 Page 2 of 21 Issue Date: 19/01/2023

Fromm Packaging Metal Marking Texpen/Dalo - All Colours

Version No: 5.1 Print Date: 19/01/2023

H350	May cause cancer.
H411	Toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

Obtain special instructions before use.
Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
Use only outdoors or in a well-ventilated area.
Wear protective gloves, protective clothing, eye protection and face protection.
Ground and bond container and receiving equipment.
Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
Use non-sparking tools.
Take action to prevent static discharges.
Avoid breathing mist/vapours/spray.
Avoid release to the environment.
Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.	
P331	Do NOT induce vomiting.	
P308+P313	IF exposed or concerned: Get medical advice/ attention.	
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam to extinguish.	
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.	
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.	
P337+P313	If eye irritation persists: Get medical advice/attention.	
P391	Collect spillage.	
P302+P352	IF ON SKIN: Wash with plenty of water.	
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].	
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.	
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

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

Precautionary statement(s) Disposal

P501 Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mivturos

%[v	%[weight]	Name
-7 <30	<30	<u>kaolin</u>
7-7 <30	<30	titanium dioxide
5-6. <30	<30	naphtha petroleum, light aromatic solvent
<30	<30	1.2.4-trimethyl benzene
-4 <5	<5	carbon black
-9 <5	<5	silica amorphous
3 <5	<5	1.3.5-trimethyl benzene
-3. <5	<5	Stoddard Solvent
1-2 <5	<5	aluminium hydroxide
-7 <5	<5	xylene
lable <5	<5	isopropyl benzene - cumene
· <1	<1	<u>ethylbenzene</u>
Legend: 1. 0		ssification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 12

SECTION 4 First aid measures

Chemwatch: 5240-10 Page 3 of 21 Issue Date: 19/01/2023 Version No: 5.1

Fromm Packaging Metal Marking Texpen/Dalo - All Colours

Print Date: 19/01/2023

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: Wash out immediately 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. Seek medical attention without delay; if pain persists or recurs seek medical attention. Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: Immediately remove all contaminated clothing, including footwear. Flush skin and hair with running water (and soap if available). Seek medical attention in event of irritation.
Inhalation	 If fumes or combustion products are inhaled remove from contaminated area. 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. Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. Transport to hospital, or doctor, without delay.
Ingestion	 If swallowed do NOT induce vomiting. If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. Observe the patient carefully. Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. Seek medical advice. Avoid giving milk or oils. Avoid giving alcohol. 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

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

SECTION 5 Firefighting measures

Extinguishing media

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

Special hazards arising from the substrate or mixture

Fire Incompatibility	Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
dvice for firefighters	
Fire Fighting	 Alert Fire Brigade and tell them location and nature of hazard. May be violently or explosively reactive. Wear breathing apparatus plus protective gloves. Prevent, by any means available, spillage from entering drains or water course. If safe, switch off electrical equipment until vapour fire hazard removed. Use water delivered as a fine spray to control fire and cool adjacent area. Avoid spraying water onto liquid pools. 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.
Fire/Explosion Hazard	 Liquid and vapour are flammable. Moderate fire hazard when exposed to heat or flame. Vapour forms an explosive mixture with air. Moderate explosion hazard when exposed to heat or flame. Vapour may travel a considerable distance to source of ignition. Heating may cause expansion or decomposition leading to violent rupture of containers. On combustion, may emit toxic fumes of carbon monoxide (CO). Combustion products include: carbon dioxide (CO2) nitrogen oxides (NOx) other pyrolysis products typical of burning organic material. May emit clouds of acrid smoke
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SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Issue Date: 19/01/2023 Chemwatch: 5240-10 Page 4 of 21 Version No: 5.1

Print Date: 19/01/2023 Fromm Packaging Metal Marking Texpen/Dalo - All Colours

Methods and material for containment and cleaning up

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Minor Spills	 Remove all ignition sources. Clean up all spills immediately. Avoid breathing vapours and contact with skin and eyes. Control personal contact with the substance, by using protective equipment. Contain and absorb small quantities with vermiculite or other absorbent material. Wipe up. Collect residues in a flammable waste container.
Major Spills	 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 course. Consider evacuation (or protect in place). 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. Contain spill with sand, earth or vermiculite. Use only spark-free shovels and explosion proof equipment. Collect recoverable product into labelled containers for recycling. Absorb remaining product with sand, earth or vermiculite. Collect solid residues and seal in labelled drums for disposal. Wash area and prevent runoff into drains. If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling The conductivity of this material may make it a static accumulator., A liquid is typically considered nonconductive if its conductivity is below 100 pS/m and is considered semi-conductive if its conductivity is below 10 000 pS/m., Whether a liquid is nonconductive or semi-conductive, the precautions are the same., A number of factors, for example liquid temperature, presence of contaminants, and anti-static additives can greatly influence the conductivity of a liquid. ▶ Containers, even those that have been emptied, may contain explosive vapours. ▶ Do NOT cut, drill, grind, weld or perform similar operations on or near containers. DO NOT allow clothing wet with material to stay in contact with skin Avoid all personal contact, including inhalation. Wear protective clothing when risk of overexposure 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. Safe handling Avoid generation of static electricity. DO NOT use plastic buckets Earth all lines and equipment Use spark-free tools when handling. Avoid contact with incompatible materials. When handling, DO NOT eat, drink or smoke Keep containers securely sealed when not in use. 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. Store in original containers in approved flammable liquid storage area. Store away from incompatible materials in a cool, dry, well-ventilated area. DO NOT store in pits, depressions, basements or areas where vapours may be trapped. No smoking, naked lights, heat or ignition sources Storage areas should be clearly identified, well illuminated, clear of obstruction and accessible only to trained and authorised personnel adequate security must be provided so that unauthorised personnel do not have access.

Other information

- Store according to applicable regulations for flammable materials for storage tanks, containers, piping, buildings, rooms, cabinets, allowable quantities and minimum storage distances
- Use non-sparking ventilation systems, approved explosion proof equipment and intrinsically safe electrical systems.
- Have appropriate extinguishing capability in storage area (e.g. portable fire extinguishers dry chemical, foam or carbon dioxide) and flammable gas detectors.
- Keep adsorbents for leaks and spills readily available.
- Protect containers against physical damage and check regularly for leaks.
- Observe manufacturer's storage and handling recommendations contained within this SDS.

In addition, for tank storages (where appropriate):

- Store in grounded, properly designed and approved vessels and away from incompatible materials.
- For bulk storages, consider use of floating roof or nitrogen blanketed vessels; where venting to atmosphere is possible, equip storage tank vents with flame arrestors; inspect tank vents during winter conditions for vapour/ ice build-up.
- Storage tanks should be above ground and diked to hold entire contents.

Conditions for safe storage, including any incompatibilities

Suitable container

- Packing as supplied by manufacturer.
- Plastic containers may only be used if approved for flammable liquid.
- Check that containers are clearly labelled and free from leaks
- For low viscosity materials (i): Drums and jerry cans must be of the non-removable head type. (ii): Where a can is to be used as an inner package, the can must have a screwed enclosure.

Version No: **5.1**

Fromm Packaging Metal Marking Texpen/Dalo - All Colours

Issue Date: **19/01/2023**Print Date: **19/01/2023**

- ▶ For materials with a viscosity of at least 2680 cSt. (23 deg. C)
- ▶ For manufactured product having a viscosity of at least 250 cSt. (23 deg. C)
- Manufactured product that requires stirring before use and having a viscosity of at least 20 cSt (25 deg. C): (i) Removable head packaging; (ii) Cans with friction closures and (iii) low pressure tubes and cartridges may be used.
- Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages
- In addition, where inner packagings are glass and contain liquids of packing group I there must be sufficient inert absorbent to absorb any spillage, unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
- Avoid reaction with oxidising agents
- Storage incompatibility

 Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.
 - Avoid storage with reducing agents.
 - Avoid strong bases.

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	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	titanium dioxide	Titanium dioxide	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	carbon black	Carbon black	3 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Silica, fused	0.05 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Fume (thermally generated)(respirable dust)	2 mg/m3	Not Available	Not Available	(e) Containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Diatomaceous earth (uncalcined)	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Silica gel	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Precipitated silica	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Fumed silica (respirable dust)	2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	Stoddard Solvent	White spirits	790 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	xylene	Xylene (o-, m-, p- isomers)	80 ppm / 350 mg/m3	655 mg/m3 / 150 ppm	Not Available	Not Available
Australia Exposure Standards	ethylbenzene	Ethyl benzene	100 ppm / 434 mg/m3	543 mg/m3 / 125 ppm	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
naphtha petroleum, light aromatic solvent	1,200 mg/m3	6,700 mg/m3	40,000 mg/m3
1,2,4-trimethyl benzene	140 mg/m3	360 mg/m3	2,200 mg/m3
1,2,4-trimethyl benzene	Not Available	Not Available	480 ppm
carbon black	9 mg/m3	99 mg/m3	590 mg/m3
silica amorphous	18 mg/m3	200 mg/m3	1,200 mg/m3
silica amorphous	18 mg/m3	100 mg/m3	630 mg/m3
silica amorphous	120 mg/m3	1,300 mg/m3	7,900 mg/m3
silica amorphous	45 mg/m3	500 mg/m3	3,000 mg/m3
silica amorphous	18 mg/m3	740 mg/m3	4,500 mg/m3
1,3,5-trimethyl benzene	Not Available	Not Available	480 ppm
Stoddard Solvent	300 mg/m3	1,800 mg/m3	29500** mg/m3
aluminium hydroxide	8.7 mg/m3	73 mg/m3	440 mg/m3
xylene	Not Available	Not Available	Not Available
ethylbenzene	Not Available	Not Available	Not Available

Ingredient Original IDLH Revised IDLH

Chemwatch: 5240-10 Page 6 of 21 Issue Date: 19/01/2023 Version No: 5.1 Print Date: 19/01/2023

Fromm Packaging Metal Marking Texpen/Dalo - All Colours

Ingredient	Original IDLH	Revised IDLH
kaolin	Not Available	Not Available
titanium dioxide	5,000 mg/m3	Not Available
naphtha petroleum, light aromatic solvent	Not Available	Not Available
1,2,4-trimethyl benzene	Not Available	Not Available
carbon black	1,750 mg/m3	Not Available
silica amorphous	3,000 mg/m3	Not Available
1,3,5-trimethyl benzene	Not Available	Not Available
Stoddard Solvent	20,000 mg/m3	Not Available
aluminium hydroxide	Not Available	Not Available
xylene	900 ppm	Not Available
ethylbenzene	800 ppm	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
1,2,4-trimethyl benzene	Е	≤ 0.1 ppm
1,3,5-trimethyl benzene	E	≤ 0.1 ppm
aluminium hydroxide	Е	≤ 0.01 mg/m³

Notes:

Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.

MATERIAL DATA

NOTE P: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.01% w/w benzene (EINECS No 200-753-7). Note E shall also apply when the substance is classified as a carcinogen. This note applies only to certain complex oil-derived substances in Annex VI. European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

Exposure controls

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

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

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

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

- Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulated area.
 - Work should be undertaken in an isolated system such as a "glove-box". Employees should wash their hands and arms upon completion of the assigned task and before engaging in other activities not associated with the isolated system.
 - Within regulated areas, the carcinogen should be stored in sealed containers, or enclosed in a closed system, including piping systems, with any sample ports or openings closed while the carcinogens are contained within.
 - Open-vessel systems are prohibited.
 - Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the
 - Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system.
 - For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood.
 - Except for outdoor systems, regulated areas should be maintained under negative pressure (with respect to non-regulated areas).
 - Local exhaust ventilation requires make-up air be supplied in equal volumes to replaced air.
 - Laboratory hoods must be designed and maintained so as to draw air inward at an average linear face velocity of 0.76 m/sec with a minimum of 0.64 m/sec. Design and construction of the fume hood requires that insertion of any portion of the employees body, other than hands and arms, be disallowed.

Personal protection

Appropriate engineering

controls









- Safety glasses with side shields
- Chemical goggles

Eve and face protection

Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalentl

Skin protection

See Hand protection below

- Wear chemical protective gloves, e.g. PVC.
- Wear safety footwear or safety gumboots, e.g. Rubber

Hands/feet protection

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.

Chemwatch: 5240-10 Page 7 of 21 Issue Date: 19/01/2023 Version No: 5.1

Fromm Packaging Metal Marking Texpen/Dalo - All Colours

Print Date: 19/01/2023

The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.

Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:

- · frequency and duration of contact
- · chemical resistance of glove material,
- · glove thickness and
- dexterity

Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).

- · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374. AS/NZS 2161.10.1 or national equivalent) is recommended.
- · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374. AS/NZS 2161.10.1 or national equivalent) is recommended.
- · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
- · Contaminated gloves should be replaced.

As defined in ASTM F-739-96 in any application, gloves are rated as:

- · Excellent when breakthrough time > 480 min
- · Good when breakthrough time > 20 min
- · Fair when breakthrough time < 20 min
- · Poor when glove material degrades

For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.

It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.

Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task.

Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:

- Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.
- · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential

Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

Body protection

Other protection

See Other protection below

Figure 2 Employees working with confirmed human carcinogens should be provided with, and be required to wear, clean, full body protective clothing (smocks, coveralls, or long-sleeved shirt and pants), shoe covers and gloves prior to entering the regulated area. [AS/NZS ISO 6529:2006 or

- Employees engaged in handling operations involving carcinogens should be provided with, and required to wear and use half-face filter-type respirators with filters for dusts, mists and fumes, or air purifying canisters or cartridges. A respirator affording higher levels of protection may be substituted. [AS/NZS 1715 or national equivalent]
- Figure 2 Emergency deluge showers and eyewash fountains, supplied with potable water, should be located near, within sight of, and on the same level with locations where direct exposure is likely.
- Prior to each exit from an area containing confirmed human carcinogens, employees should be required to remove and leave protective clothing and equipment at the point of exit and at the last exit of the day, to place used clothing and equipment in impervious containers at the point of exit for purposes of decontamination or disposal. The contents of such impervious containers must be identified with suitable labels. For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood.
- Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood.
- Overalls.
 - PVC Apron.
 - ▶ PVC protective suit may be required if exposure severe.
 - Eyewash unit.
 - Ensure there is ready access to a safety shower.
 - Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity
 - For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets).
 - Non sparking safety or conductive footwear should be considered. Conductive footwear describes a boot or shoe with a sole made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground the foot an shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.

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 computergenerated selection:

Fromm Packaging Metal Marking Texpen/Dalo - All Colours

Material	СРІ
BUTYL	С
BUTYL/NEOPRENE	С
HYPALON	С
NAT+NEOPR+NITRILE	С
NATURAL+NEOPRENE	С
NEOPRENE	С
NEOPRENE/NATURAL	С
NITRILE	С

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required. Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

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

Issue Date: 19/01/2023
Print Date: 19/01/2023

NITRILE+PVC	С
PE/EVAL/PE	С
PVA	С
PVC	С
PVDC/PE/PVDC	С
TEFLON	С
VITON	С

- * CPI Chemwatch Performance Index
- A: Best Selection
- B: Satisfactory; may degrade after 4 hours continuous immersion
- C: Poor to Dangerous Choice for other than short term immersion

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

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

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	Opaque various coloured, thick viscosity flammable liquid with aromatic odour; partially miscible with water. Coloured		
Physical state	Liquid	Relative density (Water = 1)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Applicable	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	158.89-170	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	42.22	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Flammable.	Oxidising properties	Not Available
Upper Explosive Limit (%)	12.3	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	1.9	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Partly miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	>1	VOC g/L	<430

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	 Unstable in the presence of incompatible materials. Product is considered stable. Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

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

Inhaled

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

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

Inhalation hazard is increased at higher temperatures.

Version No: **5.1**

Fromm Packaging Metal Marking Texpen/Dalo - All Colours

Issue Date: 19/01/2023 Print Date: 19/01/2023

	Acute effects from inhalation of high concentrations of vapour are pulmor depression - characterised by headache and dizziness, increased reaction		
Ingestion	Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result. Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis). Accidental ingestion of the material may be damaging to the health of the individual.		
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. The material may accentuate any pre-existing dermatitis condition Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.		
Еуе	Evidence exists, or practical experience predicts, that the material may cause 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. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.		
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. On the basis, primarily, of animal experiments, the material may be regarded as carcinogenic to humans. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in cancer on the basis of: - appropriate long-term animal studies - other relevant information Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. There is some evidence that human exposure to the material may result in developmental toxicity. This evidence is based on animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.		
	TOXICITY	IRRITATION	
	Dermal (None) LD50: 8777 mg/kg ^[2]	Not Available	
Fromm Packaging Metal Marking Texpen/Dalo - All	Inhalation (None) LC50: 54 mg/L (vapour) ^[2]		
Colours	Inhalation (None) LC50: 8 mg/L ^[2]		
	Oral (None) LD50: 9951 mg/kg ^[2]		
	(······)· ····g···g		
kaolin	TOXICITY	IRRITATION	
	Not Available	Not Available	
	TOXICITY	IRRITATION	
	dermal (hamster) LD50: >=10000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]	
titanium dioxide	Inhalation(Rat) LC50: >2.28 mg/l4h ^[1]	Skin (human): 0.3 mg /3D (int)-mild *	
	Oral (Rat) LD50; >=2000 mg/kg ^[1]	, , , , ,	
		Skin. no adverse effect observed (not imtating). 1	
		Skin: no adverse effect observed (not irritating) ^[1]	
	тохісіту	IRRITATION	
naphtha petroleum, light	, , , .		
naphtha petroleum, light aromatic solvent	TOXICITY Dermal (rabbit) LD50: >1900 mg/kg ^[1] Inhalation(Rat) LC50: >4.42 mg/L4h ^[1]	IRRITATION	
	TOXICITY Dermal (rabbit) LD50: >1900 mg/kg ^[1]	IRRITATION Eye: no adverse effect observed (not irritating) ^[1]	
	TOXICITY Dermal (rabbit) LD50: >1900 mg/kg ^[1] Inhalation(Rat) LC50: >4.42 mg/L4h ^[1]	IRRITATION Eye: no adverse effect observed (not irritating) ^[1]	
	TOXICITY Dermal (rabbit) LD50: >1900 mg/kg ^[1] Inhalation(Rat) LC50: >4.42 mg/L4h ^[1] Oral (Rat) LD50; >4500 mg/kg ^[1]	IRRITATION Eye: no adverse effect observed (not irritating) ^[1] Skin: adverse effect observed (irritating) ^[1]	
	TOXICITY Dermal (rabbit) LD50: >1900 mg/kg ^[1] Inhalation(Rat) LC50: >4.42 mg/L4h ^[1] Oral (Rat) LD50; >4500 mg/kg ^[1] TOXICITY	IRRITATION Eye: no adverse effect observed (not irritating) ^[1] Skin: adverse effect observed (irritating) ^[1] IRRITATION	
aromatic solvent	TOXICITY	IRRITATION Eye: no adverse effect observed (not irritating) ^[1] Skin: adverse effect observed (irritating) ^[1] IRRITATION	
aromatic solvent	TOXICITY Dermal (rabbit) LD50: >1900 mg/kg ^[1] Inhalation(Rat) LC50: >4.42 mg/L4h ^[1] Oral (Rat) LD50; >4500 mg/kg ^[1] TOXICITY Dermal (rabbit) LD50: >3160 mg/kg ^[2] Inhalation(Rat) LC50: 18 mg/L4h ^[2] Oral (Rat) LD50; 6000 mg/kg ^[1]	IRRITATION Eye: no adverse effect observed (not irritating) ^[1] Skin: adverse effect observed (irritating) ^[1] IRRITATION Not Available	
aromatic solvent 1,2,4-trimethyl benzene	TOXICITY	IRRITATION Eye: no adverse effect observed (not irritating) ^[1] Skin: adverse effect observed (irritating) ^[1] IRRITATION Not Available IRRITATION	
aromatic solvent	TOXICITY	IRRITATION Eye: no adverse effect observed (not irritating) ^[1] Skin: adverse effect observed (irritating) ^[1] IRRITATION Not Available IRRITATION Eye: no adverse effect observed (not irritating) ^[1]	
aromatic solvent 1,2,4-trimethyl benzene	TOXICITY	IRRITATION Eye: no adverse effect observed (not irritating) ^[1] Skin: adverse effect observed (irritating) ^[1] IRRITATION Not Available IRRITATION	
aromatic solvent 1,2,4-trimethyl benzene	TOXICITY	IRRITATION Eye: no adverse effect observed (not irritating) ^[1] Skin: adverse effect observed (irritating) ^[1] IRRITATION Not Available IRRITATION Eye: no adverse effect observed (not irritating) ^[1]	
aromatic solvent 1,2,4-trimethyl benzene	TOXICITY	IRRITATION Eye: no adverse effect observed (not irritating) ^[1] Skin: adverse effect observed (irritating) ^[1] IRRITATION Not Available IRRITATION Eye: no adverse effect observed (not irritating) ^[1] Skin: no adverse effect observed (not irritating) ^[1] IRRITATION Eye (rabbit): non-irritating *	
aromatic solvent 1,2,4-trimethyl benzene	TOXICITY	IRRITATION Eye: no adverse effect observed (not irritating) ^[1] Skin: adverse effect observed (irritating) ^[1] IRRITATION Not Available IRRITATION Eye: no adverse effect observed (not irritating) ^[1] Skin: no adverse effect observed (not irritating) ^[1] IRRITATION IRRITATION	
1,2,4-trimethyl benzene	TOXICITY	IRRITATION Eye: no adverse effect observed (not irritating) ^[1] Skin: adverse effect observed (irritating) ^[1] IRRITATION Not Available IRRITATION Eye: no adverse effect observed (not irritating) ^[1] Skin: no adverse effect observed (not irritating) ^[1] IRRITATION Eye (rabbit): non-irritating *	

Chemwatch: 5240-10

Page 10 of 21 Issue Date: 19/01/2023 Version No: 5.1 Print Date: 19/01/2023 Fromm Packaging Metal Marking Texpen/Dalo - All Colours

	TOXICITY	IRRITATION	
	dermal (rat) LD50: >3460 mg/kg ^[1]	Eye (rabbit): 500 mg/24h mild	
1,3,5-trimethyl benzene	Inhalation(Rat) LC50: 24 mg/L4h ^[2]	Eye: adverse effect observed (irritating) ^[1]	
	Oral (Rat) LD50; 6000 mg/kg ^[1]	Skin (rabbit): 20 mg/24h moderate	
		Skin: adverse effect observed (irritating) ^[1]	
	TOXICITY	IRRITATION	
	Dermal (rabbit) LD50: >3000 mg/kg ^[1]	Eye (hmn) 470 ppm/15m irrit.	
	Inhalation(Rat) LC50: >5.5 mg/l4h ^[1]	Eye (rabbit) 500 mg/24h moderate	
Stoddard Solvent	Oral (Rat) LD50; >5000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]	
		Skin: adverse effect observed (irritating) ^[1]	
		Skin: no adverse effect observed (not irritating) ^[1]	
	TOXICITY	IRRITATION	
aluminium hydroxide	Inhalation(Rat) LC50: >2.3 mg/l4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]	
	Oral (Rat) LD50; >2000 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]	
	TOXICITY	IRRITATION	
	Dermal (rabbit) LD50: >1700 mg/kg ^[2]	Eye (human): 200 ppm irritant	
	Inhalation(Rat) LC50: 5000 ppm4h ^[2]	Eye (rabbit): 5 mg/24h SEVERE	
xylene	Oral (Mouse) LD50; 2119 mg/kg ^[2]	Eye (rabbit): 87 mg mild	
		Eye: adverse effect observed (irritating) ^[1]	
		Skin (rabbit):500 mg/24h moderate	
		Skin: adverse effect observed (irritating) ^[1]	
	TOXICITY	IRRITATION	
	Dermal (rabbit) LD50: 17800 mg/kg ^[2]	Eye (rabbit): 500 mg - SEVERE	
ethylbenzene	Inhalation(Rat) LC50: 17.2 mg/l4h ^[2]	Eye: no adverse effect observed (not irritating) ^[1]	
	Oral (Rat) LD50; 3500 mg/kg ^[2]	Skin (rabbit): 15 mg/24h mild	
		Skin: no adverse effect observed (not irritating) ^[1]	
Legend:	Value obtained from Europe ECHA Registered Substant		

for bentonite clays:

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

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

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

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

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

KAOLIN

TITANIUM DIOXIDE

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

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

For titanium dioxide:

Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals. (General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) With regard to inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral ingestion of fine titanium dioxide showed particle 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.

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

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

Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain. Clearance of titanium dioxide is also affected by

Continued...

Chemwatch: 5240-10 Page 11 of 21 Issue Date: 19/01/2023 Version No: 5.1

Fromm Packaging Metal Marking Texpen/Dalo - All Colours

Print Date: 19/01/2023

pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols. Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide are more slowly cleared than their fine counterparts.

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

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

Animal carcinogenicity data

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

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

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

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

The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce

Inhalation (rat) TCLo: 1320 ppm/6h/90D-I * [Devoe]

For Low Boiling Point Naphthas (LBPNs):

Acute toxicity:

LBPNs generally have low acute toxicity by the oral (median lethal dose [LD50] in rats > 2000 mg/kg-bw), inhalation (LD50 in rats > 5000 mg/m3) and dermal (LD50 in rabbits > 2000 mg/kg-bw) routes of exposure

Most LBPNs are mild to moderate eye and skin irritants in rabbits, with the exception of heavy catalytic cracked and heavy catalytic reformed naphthas, which have higher primary skin irritation indices

Sensitisation:

LBPNs do not appear to be skin sensitizers, but a poor response in the positive control was also noted in these studies Repeat dose toxicity:

The lowest-observed-adverse-effect concentration (LOAEC) and lowest-observed-adverse-effect level (LOAEL) values identified following short-term (2-89 days) and subchronic (greater than 90 days) exposure to the LBPN substances. These values were determined for a variety of endpoints after considering the toxicity data for all LBPNs in the group. Most of the studies were carried out by the inhalation route of exposure. Renal effects, including increased kidney weight, renal lesions (renal tubule dilation, necrosis) and hyaline droplet formation, observed in male rats exposed orally or by inhalation to most LBPNs, were considered species- and sex-specific These effects were determined to be due to a mechanism of action not relevant to humans -specifically, the interaction between hydrocarbon metabolites and alpha-2-microglobulin, an enzyme not produced in substantial amounts in female rats, mice and other species, including humans. The resulting nephrotoxicity and subsequent carcinogenesis in male rats were therefore not considered in deriving LOAEC/LOAEL values.

Only a limited number of studies of short-term and subchronic duration were identified for site-restricted LBPNs. The lowest LOAEC identified in these studies, via the inhalation route, is 5475 mg/m3, based on a concentration-related increase in liver weight in both male and female rats following a 13-week exposure to light catalytic cracked naphtha. Shorter exposures of rats to this test substance resulted in nasal irritation at 9041 mg/m3

No systemic toxicity was reported following dermal exposure to light catalytic cracked naphtha, but skin irritation and accompanying histopathological changes were increased, in a dose-dependent manner, at doses as low as 30 mg/kg-bw per day when applied 5 days per week

No non-cancer chronic toxicity studies (= 1 year) were identified for site-restricted LBPNs and very few non-cancer chronic toxicity studies were identified for other LBPNs. An LOAEC of 200 mg/m3 was noted in a chronic inhalation study that exposed mice and rats to unleaded gasoline (containing 2% benzene). This inhalation LOAEC was based on ocular discharge and ocular irritation in rats. At the higher concentration of 6170 mg/m3, increased kidney weight was observed in male and female rats (increased kidney weight was also observed in males only at 870 mg/m3). Furthermore, decreased body weight in male and female mice was also observed at 6170 mg/m3

A LOAEL of 714 mg/kg-bw was identified for dermal exposure based on local skin effects (inflammatory and degenerative skin changes) in mice following application of naphtha for 105 weeks. No systemic toxicity was reported.

Although few genotoxicity studies were identified for the site-restricted LBPNs, the genotoxicity of several other LBPN substances has been evaluated using a variety of in vivo and in vitro assays. While in vivo genotoxicity assays were negative overall, the in vitro tests exhibited mixed

For in vivo genotoxicity tests, LBPNs exhibited negative results for chromosomal aberrations and micronuclei induction, but exhibited positive results in one sister chromatid exchange assay although this result was not considered definitive for clastogenic activity as no genetic material was unbalanced or lost. Mixtures that were tested, which included a number of light naphthas, displayed mixed results (i.e., both positive and negative for the same assay) for chromosomal aberrations and negative results for the dominant lethal mutation assay. Unleaded gasoline (containing 2% benzene) was tested for its ability to induce unscheduled deoxyribonucleic acid (DNA) synthesis (UDS) and replicative DNA synthesis (RDS) in rodent hepatocytes and kidney cells. UDS and RDS were induced in mouse hepatocytes via oral exposure and RDS was induced in rat kidney cells via oral and inhalation exposure. Unleaded gasoline (benzene content not stated) exhibited negative results for chromosomal aberrations and the dominant lethal mutation assay and mixed results for atypical cell foci in rodent renal and hepatic cells. For in vitro genotoxicity studies, LBPNs were negative for six out of seven Ames tests, and were also negative for UDS and for forward mutations LBPNs exhibited mixed or equivocal results for the mouse lymphoma and sister chromatid exchange assays, as well as for cell transformation and positive results for one bacterial DNA repair assay. Mixtures that were tested, which included a number of light naphthas, displayed negative results for the Ames and mouse lymphoma assays Gasoline exhibited negative results for the Ames test battery, the sister chromatid exchange assay and for one mutagenicity assay . Mixed results were observed for UDS and the mouse lymphoma assay.

While the majority of in vivo genotoxicity results for LBPN substances are negative, the potential for genotoxicity of LBPNs as a group cannot be discounted based on the mixed in vitro genotoxicity results

Although a number of epidemiological studies have reported increases in the incidence of a variety of cancers, the majority of these studies are considered to contain incomplete or inadequate information. Limited data, however, are available for skin cancer and leukemia incidence, as well as mortality among petroleum refinery workers. It was concluded that there is limited evidence supporting the view that working in petroleum refineries entails a carcinogenic risk (Group 2A carcinogen). IARC (1989a) also classified gasoline as a Group 2B carcinogen; it considered the evidence for carcinogenicity in humans from gasoline to be inadequate and noted that published epidemiological studies had several limitations, including a lack of exposure data and the fact that it was not possible to separate the effects of combustion products from those of gasoline itself.

NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT Chemwatch: 5240-10 Page 12 of 21 Issue Date: 19/01/2023 Version No: 5.1 Print Date: 19/01/2023

Fromm Packaging Metal Marking Texpen/Dalo - All Colours

Similar conclusions were drawn from other reviews of epidemiological studies for gasoline (US EPA 1987a, 1987b). Thus, the evidence gathered

from these epidemiological studies is considered to be inadequate to conclude on the effect s of human exposure to LBPN substances.

No inhalation studies assessing the carcinogenicity of the site-restricted LBPNs were identified. Only unleaded gasoline has been examined for its carcinogenic potential, in several inhalation studies. In one study, rats and mice were exposed to 0, 200, 870 or 6170 mg/m3 of a 2% benzene formulation of the test substance, via inhalation, for approximately 2 years. A statistically significant increase in hepatocellular adenomas and carcinomas, as well as a non-statistical increase in renal tumours, were observed at the highest dose in female mice. A dose-dependent increase in the incidence of primary renal neoplasms was also detected in male rats, but this was not considered to be relevant to humans, as discussed previously. Carcinogenicity was also assessed for unleaded gasoline, via inhalation, as part of initiation/promotion studies. In these studies, unleaded gasoline did not appear to initiate tumour formation, but did show renal cell and hepatic tumour promotion ability, when rats and mice were exposed, via inhalation, for durations ranging from 13 weeks to approximately 1 year using an initiation/promotion protocol However, further examination of data relevant to the composition of unleaded gasoline demonstrated that this is a highly-regulated substance; it is expected to contain a lower percentage of benzene and has a discrete component profile when compared to other substances in the LBPN group. Both the European Commission and the International Agency for Research on Cancer (IARC) have classified LBPN substances as carcinogenic. All of these substances were classified by the European Commission (2008) as Category 2 (R45: may cause cancer) (benzene content = 0.1% by weight). IARC has classified gasoline, an LBPN, as a Group 2B carcinogen (possibly carcinogenic to humans) and "occupational exposures in petroleum refining" as Group 2A carcinogens (probably carcinogenic to humans).

Several studies were conducted on experimental animals to investigate the dermal carcinogenicity of LBPNs. The majority of these studies were conducted through exposure of mice to doses ranging from 694-1351 mg/kg-bw, for durations ranging from 1 year to the animals lifetime or until a tumour persisted for 2 weeks. Given the route of exposure, the studies specifically examined the formation of skin tumours. Results for carcinogenicity via dermal exposure are mixed. Both malignant and benign skin tumours were induced with heavy catalytic cracked naphtha, light catalytic cracked naphtha, light

straight-run naphtha and naphtha Significant increases in squamous cell carcinomas were also observed when mice were dermally treated with Stoddard solvent, but the latter was administered as a mixture (90% test substance), and the details of the study were not available. In contrast, insignificant increases in tumour formation or no tumours were observed when light alkylate naphtha, heavy catalytic reformed naphtha, sweetened naphtha, light catalytically cracked naphtha

or unleaded gasoline was dermally applied to mice. Negative results for skin tumours were also observed in male mice dermally exposed to sweetened naphtha using an initiation/promotion protocol.

Reproductive/ Developmental toxicity:

No reproductive or developmental toxicity was observed for the majority of LBPN substances evaluated. Most of these studies were carried out by inhalation exposure in rodents.

NOAEC values for reproductive toxicity following inhalation exposure ranged from 1701 mg/m3 (CAS RN 8052-41-3) to 27 687 mg/m3 (CAS RN 64741-63-5) for the LBPNs group evaluated, and from 7690 mg/m3 to 27 059 mg/m3 for the site-restricted light catalytic cracked and full-range catalytic reformed naphthas. However, a decreased number of pups per litter and higher frequency of post-implantation loss were observed following inhalation exposure of female rats to hydrotreated heavy naphtha (CAS RN 64742-48-9) at a concentration of 4679 mg/m3, 6 hours per day, from gestational days 7-20. For dermal exposures, NOAEL values of 714 mg/kg-bw (CAS RN 8030-30-6) and 1000 mg/kg-bw per day (CAS RN 68513-02-0) were noted. For oral exposures, no adverse effects on reproductive parameters were reported when rats were given site-restricted light catalytic cracked naphtha at 2000 mg/kg on gestational day 13 .

For most LBPNs, no treatment-related developmental effects were observed by the different routes of exposure However, developmental toxicity was observed for a few naphthas. Decreased foetal body weight and an increased incidence of ossification variations were observed when rat dams were exposed to light aromatized solvent naphtha, by gavage, at 1250 mg/kg-bw per day. In addition, pregnant rats exposed by inhalation to hydrotreated heavy naphtha at 4679 mg/m3 delivered pups with higher birth weights. Cognitive and memory impairments were also observed in the offspring.

Low Boiling Point Naphthas [Site-Restricted]

Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length, with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent that iso- or cyclo-paraffins.

The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption, is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver. For C9 aromatics (typically trimethylbenzenes - TMBs)

Acute Toxicity

Acute toxicity studies (oral, dermal and inhalation routes of exposure) have been conducted in rats using various solvent products containing predominantly mixed C9 aromatic hydrocarbons (CAS RN 64742-95-6). Inhalation LC50 s range from 6,000 to 10,000 mg/m 3 for C9 aromatic naphtha and 18,000 to 24,000 mg/m3 for 1,2,4 and 1,3,5-TMB, respectively. A rat oral LD50 reported for 1,2,4-TMB is 5 grams/kg bw and a rat dermal LD50 for the C9 aromatic naphtha is >4 ml/kg bw. These data indicate that C9 aromatic solvents show that LD50/LC50 values are greater than limit doses for acute toxicity studies established under OECD test guidelines

Irritation and Sensitization

Several irritation studies, including skin, eye, and lung/respiratory system, have been conducted on members of the category. The results indicate that C9 aromatic hydrocarbon solvents are mildly to moderately irritating to the skin, minimally irritating to the eye, and have the potential to irritate the respiratory tract and cause depression of respiratory rates in mice. Respiratory irritation is a key endpoint in the current occupational exposure limits established for C9 aromatic hydrocarbon solvents and trimethylbenzenes. No evidence of skin sensitization was identified. Repeated Dose Toxicity

Inhalation: The results from a subchronic (3 month) neurotoxicity study and a one-year chronic study (6 hr/day, 5 days/week) indicate that effects from inhalation exposure to C9 Aromatic Hydrocarbon Solvents on systemic toxicity are slight. A battery of neurotoxicity and neurobehavioral endpoints were evaluated in the 3-month inhalation study on C9 aromatic naphtha tested at concentrations of 0, 101, 452, or 1320 ppm (0, 500, 2,220, or 6,500 mg/m3). In this study, other than a transient weight reduction in the high exposure group (not statistically significant at termination of exposures), no effects were reported on neuropathology or neuro/behavioral parameters. The NOAEL for systemic and/or neurotoxicity was 6,500 mg/m3, the highest concentration tested. In an inhalation study of a commercial blend, rats were exposed to C9 aromatic naphtha concentrations of 0, 96, 198, or 373 ppm (0, 470, 970, 1830 mg/m3) for 6 hr/day, 5 days/week, for 12 months. Liver and kidney weights were increased in the high exposure group but no accompanying histopathology was observed in these organs.

The NOAEL was considered to be the high exposure level of 373 ppm, or 1830 mg/m3. In two subchronic rat inhalation studies, both of three months duration, rats were exposed to the individual TMB isomers (1,2,4-and 1,3,5-) to nominal concentrations of 0, 25, 100, or 250 ppm (0, 123, 492, or 1230 mg/m3). Respiratory irritation was observed at 492 (100 ppm) and 1230 mg/m3 (250 ppm) and no systemic toxicity was observed in either study. For both pure isomers, the NOELs are 25 ppm or 123 mg/m3 for respiratory irritation and 250 ppm or 1230 mg/m3 for systemic effects

Oral: The C9 aromatic naphtha has not been tested via the oral route of exposure. Individual TMB isomers have been evaluated in a series of repeated-dose oral studies ranging from 14 days to 3 months over a wide range of doses. The effects observed in these studies included increased liver and kidney weights, changes in blood chemistry, increased salivation, and decreased weight gain at higher doses. Organ weight changes appeared to be adaptive as they were not accompanied by histopathological effects. Blood changes appeared sporadic and without pattern. One study reported hyaline droplet nephropathy in male rats at the highest dose (1000 mg/kg bw-day), an effect that is often associated with alpha-2mu-globulin-induced nephropathy and not considered relevant to humans. The doses at which effects were detected were 100 mg/kg-bw day or above (an exception was the pilot 14 day oral study - LOAEL 150 mg/kg bw-day - but the follow up three month study had a

Chemwatch: 5240-10 Page 13 of 21 Issue Date: 19/01/2023 Version No: 5.1

Fromm Packaging Metal Marking Texpen/Dalo - All Colours

Print Date: 19/01/2023

LOAEL of 600 mg/kg/bw-day with a NOAEL of 200 mg/kg bw-day). Since effects generally were not severe and could be considered adaptive or spurious, oral exposure does not appear to pose a high toxicity hazard for pure trimethylbenzene isomers. Mutagenicity

In vitro genotoxicity testing of a variety of C9 aromatics has been conducted in both bacterial and mammalian cells. In vitro point mutation tests were conducted with Salmonella typhimurium and Escherichia coli bacterial strains, as well as with cultured mammalian cells such as the Chinese hamster cell ovary cells (HGPRT assay) with and without metabolic activation. In addition, several types of in vitro chromosomal aberration tests have been performed (chromosome aberration frequency in Chinese hamster ovary and lung cells, sister chromatid exchange in CHO cells). Results were negative both with and without metabolic activation for all category members. For the supporting chemical 1,2,3-TMB, a single in vitro chromosome aberration test was weakly positive. In in vivo bone marrow cytogenetics test, rats were exposed to C9 aromatic naphtha at concentrations of 0, 153, 471, or 1540 ppm (0, 750, 2,310, or 7,560 mg/m3) 6 hr/day, for 5 days. No evidence of in vivo somatic cell genotoxicity was detected. Based on the cumulative results of these assays, genetic toxicity is unlikely for substances in the C9 Aromatic Hydrocarbon Solvents Category

Reproductive and Developmental Toxicity

Results from the three-generation reproduction inhalation study in rats indicate limited effects from C9 aromatic naphtha. In each of three generations (F0, F1 and F2), rats were exposed to High Flash Aromatic Naphtha (CAS RN 64742-95-6) via whole body inhalation at target concentrations of 0, 100, 500, or 1500 ppm (actual mean concentrations throughout the full study period were 0, 103, 495, or 1480 ppm, equivalent to 0, 505, 2430, or 7265 mg/m3, respectively). In each generation, both sexes were exposed for 10 weeks prior to and two weeks during mating for 6 hrs/day, 5 days/wks. Female rats in the F0, F1, and F2 generation were then exposed during gestation days 0-20 and lactation days 5-21 for 6 hrs/day, 7 days/wk. The age at exposure initiation differed among generations; F0 rats were exposed starting at 9 weeks of age, F1 exposure began at 5-7 weeks, and F2 exposure began at postnatal day (PND) 22. In the F0 and F1 parental generations, 30 rats/sex /group were exposed and mated. However, in the F2 generation, 40/sex/group were initially exposed due to concerns for toxicity, and 30/sex /group were randomly selected for mating, except that all survivors were used at 1480 ppm. F3 litters were not exposed directly and were sacrificed on lactation day 21.

Systemic Effects on Parental Generations:

The F0 males showed statistically and biologically significantly decreased mean body weight by ~15% at 1480 ppm when compared with controls. Seven females died or were sacrificed in extremis at 1480 ppm. The F0 female rats in the 495 ppm exposed group had a 13% decrease in body weight gain when adjusted for initial body weight when compared to controls. The F1 parents at 1480 ppm had statistically significantly decreased mean body weights (by ~13% (females) and 22% (males)), and locomotor activity. F1 parents at 1480 ppm had increased ataxia and mortality (six females). Most F2 parents (70/80) exposed to 1480 ppm died within the first week. The remaining animals survived throughout the rest of the exposure period. At week 4 and continuing through the study, F2 parents at 1480 ppm had statistically significant mean body weights much lower than controls (~33% for males; ~28% for females); body weights at 495 ppm were also reduced significantly (by 13% in males and 15% in females). The male rats in the 495 ppm exposed group had a 12% decrease in body weight gain when adjusted for initial body weight when compared to controls. Based on reduced body weight observed, the overall systemic toxicity LOAEC is 495 ppm (2430 mg/m3). Reproductive Toxicity-Effects on Parental Generations: There were no pathological changes noted in the reproductive organs of any animal of the F0, F1, or F2 generation. No effects were reported on sperm morphology, gestational period, number of implantation sites, or post-implantation loss in any generation. Also, there were no statistically or biologically significant differences in any of the reproductive parameters, including: number of mated females, copulatory index, copulatory interval, number of females delivering a litter, number of females delivering a live litter, or male fertility in the F0 or in the F2 generation. Male fertility was statistically significantly reduced at 1480 ppm in the F1 rats. However, male fertility was not affected in the F0 or in the F2 generations; therefore, the biological significance of this change is unknown and may or may not be attributed to the test substance. No reproductive effects were observed in the F0 or F1 dams exposed to 1480 ppm (7265 mg/m3). Due to excessive mortality at the highest concentration (1480 ppm, only six dams available) in the F2 generation,, a complete evaluation is precluded. However, no clear signs of reproductive toxicity were observed in the F2 generation. Therefore, the reproductive NOAEC is considered 495 ppm (2430 mg/m3), which excludes analysis of the highest concentration due to excessive mortality.

Developmental Toxicity - Effects on Pups: Because of significant maternal toxicity (including mortality) in dams in all generations at the highest concentration (1480 ppm), effects in offspring at 1480 ppm are not reported here. No significant effects were observed in the F1 and F2 generation offspring at 103 or 495 ppm. However, in F3 offspring, body weights and body weight gain were reduced by ~ 10-11% compare controls at 495 ppm for approximately a week (PND 14 through 21). Maternal body weight was also depressed by ~ 12% throughout the gestational period compared with controls. The overall developmental LOAEC from this study is 495 ppm (2430 mg/m3) based on the body weights reductions observed in the F3 offspring.

Conclusion: No effects on reproductive parameters were observed at any exposure concentration, although a confident assessment of the group exposed at the highest concentration was not possible. A potential developmental effect (reduction in mean pup weight and weight gain) was observed at a concentration that was also associated with maternal toxicity.

1.2.4-TRIMETHYL BENZENE

CHEMWATCH 2325 1,3,5-trimethylbenzene

CARBON BLACK

SILICA AMORPHOUS

Inhalation (rat) TCLo: 50 mg/m3/6h/90D-I Nil reported

Reports indicate high/prolonged exposures to amorphous silicas induced lung fibrosis in experimental animals; in some experiments these effects were reversible. [PATTYS]

For silica amorphous:

Derived No Adverse Effects Level (NOAEL) in the range of 1000 mg/kg/d.

In humans, synthetic amorphous silica (SAS) is essentially non-toxic by mouth, skin or eyes, and by inhalation. Epidemiology studies show little evidence of adverse health effects due to SAS. Repeated exposure (without personal protection) may cause mechanical irritation of the eye and drying/cracking of the skin.

When experimental animals inhale synthetic amorphous silica (SAS) dust, it dissolves in the lung fluid and is rapidly eliminated. If swallowed, the vast majority of SAS is excreted in the faeces and there is little accumulation in the body. Following absorption across the gut, SAS is eliminated via urine without modification in animals and humans. SAS is not expected to be broken down (metabolised) in mammals.

After ingestion, there is limited accumulation of SAS in body tissues and rapid elimination occurs. Intestinal absorption has not been calculated, but appears to be insignificant in animals and humans. SASs injected subcutaneously are subjected to rapid dissolution and removal. There is no indication of metabolism of SAS in animals or humans based on chemical structure and available data. In contrast to crystalline silica, SAS is soluble in physiological media and the soluble chemical species that are formed are eliminated via the urinary tract without modification Both the mammalian and environmental toxicology of SASs are significantly influenced by the physical and chemical properties, particularly those of solubility and particle size. SAS has no acute intrinsic toxicity by inhalation. Adverse effects, including suffocation, that have been reported were caused by the presence of high numbers of respirable particles generated to meet the required test atmosphere. These results are not representative of exposure to commercial SASs and should not be used for human risk assessment. Though repeated exposure of the skin may

cause dryness and cracking, SAS is not a skin or eye irritant, and it is not a sensitiser. Repeated-dose and chronic toxicity studies confirm the absence of toxicity when SAS is swallowed or upon skin contact.

Long-term inhalation of SAS caused some adverse effects in animals (increases in lung inflammation, cell injury and lung collagen content), all of which subsided after exposure

Numerous repeated-dose, subchronic and chronic inhalation toxicity studies have been conducted with SAS in a number of species, at airborne concentrations ranging from 0.5 mg/m3 to 150 mg/m3. Lowest-observed adverse effect levels (LOAELs) were typically in the range of 1 to 50 mg/m3. When available, the no-observed adverse effect levels (NOAELs) were between 0.5 and 10 mg/m3. The difference in values may be explained by different particle size, and therefore the number of particles administered per unit dose. In general, as particle size decreases so does the NOAEL/LOAEL

Neither inhalation nor oral administration caused neoplasms (tumours). SAS is not mutagenic in vitro. No genotoxicity was detected in in vivo assays. SAS does not impair development of the foetus. Fertility was not specifically studied, but the reproductive organs in long-term studies were not affected.

For Synthetic Amorphous Silica (SAS)

Repeated dose toxicity

Oral (rat), 2 weeks to 6 months, no significant treatment-related adverse effects at doses of up to 8% silica in the diet.

Chemwatch: 5240-10 Page 14 of 21 Issue Date: 19/01/2023 Version No: 5.1

Fromm Packaging Metal Marking Texpen/Dalo - All Colours

Print Date: 19/01/2023

Inhalation (rat), 13 weeks, Lowest Observed Effect Level (LOEL) = 1.3 mg/m3 based on mild reversible effects in the lungs. Inhalation (rat), 90 days, LOEL = 1 mg/m3 based on reversible effects in the lungs and effects in the nasal cavity. For silane treated synthetic amorphous silica:

Repeated dose toxicity: oral (rat), 28-d, diet, no significant treatment-related adverse effects at the doses tested.

There is no evidence of cancer or other long-term respiratory health effects (for example, silicosis) in workers employed in the manufacture of SAS. Respiratory symptoms in SAS workers have been shown to correlate with smoking but not with SAS exposure, while serial pulmonary function values and chest radiographs are not adversely affected by long-term exposure to SAS.

1.3.5-TRIMETHYL BENZENE

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

CHEMWATCH 12171 1,2,4-trimethylbenzene

XYLENE

Reproductive effector in rats

Liver changes, utheral tract, effects on fertility, foetotoxicity, specific developmental abnormalities (musculoskeletal system) recorded. Ethylbenzene is readily absorbed following inhalation, oral, and dermal exposures, distributed throughout the body, and excreted primarily through urine. There are two different metabolic pathways for ethylbenzene with the primary pathway being the alpha-oxidation of ethylbenzene to 1-phenylethanol, mostly as the R-enantiomer. The pattern of urinary metabolite excretion varies with different mammalian species. In humans, ethylbenzene is excreted in the urine as mandelic acid and phenylgloxylic acids; whereas rats and rabbits excrete hippuric acid and phenaceturic acid as the main metabolism. Ethylbenzene can induce liver enzymes and hence its own metabolism as well as the metabolism of other

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

ETHYLBENZENE

Hearing loss has been reported in rats (but not guinea pigs) exposed to relatively high exposures (400 ppm and greater) of ethylbenzene In chronic toxicity/carcinogenicity studies, both rats and mice were exposed via inhalation to 0, 75, 250 or 750 ppm for 104 weeks. In rats, the kidney was the target organ of toxicity, with renal tubular hyperplasia noted in both males and females at the 750 ppm level only. In mice, the liver and lung were the principal target organs of toxicity. In male mice at 750 ppm, lung toxicity was described as alveolar epithelial metaplasia, and liver toxicity was described as hepatocellular syncitial alteration, hypertrophy and mild necrosis; this was accompanied by increased follicular cell hyperplasia in the thyroid. As a result the NOAEL in male mice was determined to be 250 ppm. In female mice, the 750 ppm dose group had an increased incidence of eosinophilic foci in the liver (44% vs 10% in the controls) and an increased incidence in follicular cell hyperplasia in the thyroid gland.

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

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

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

KAOLIN & TITANIUM DIOXIDE & CARBON BLACK & **ALUMINIUM HYDROXIDE**

No significant acute toxicological data identified in literature search.

TITANIUM DIOXIDE & 1,2,4-TRIMETHYL BENZENE & 1,3,5-TRIMETHYL BENZENE

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.

TITANIUM DIOXIDE & ETHYLBENZENE

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.

TITANIUM DIOXIDE & **CARBON BLACK & ETHYLBENZENE**

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

For trimethylbenzenes:

Absorption of 1,2,4-trimethylbenzene occurs after oral, inhalation, or dermal exposure. Occupationally, inhalation and dermal exposures are the most important routes of absorption although systemic intoxication from dermal absorption is not likely to occur due to the dermal irritation caused by the chemical prompting quick removal. Following oral administration of the chemical to rats, 62.6% of the dose was recovered as urinary metabolites indicating substantial absorption . 1,2,4-Trimethylbenzene is lipophilic and may accumulate in fat and fatty tissues. In the blood stream, approximately 85% of the chemical is bound to red blood cells Metabolism occurs by side-chain oxidation to form alcohols and carboxylic acids which are then conjugated with glucuronic acid, glycine, or sulfates for urinary excretion. After a single oral dose to rats of 1200 mg/kg, urinary metabolites consisted of approximately 43.2% glycine, 6.6% glucuronic, and 12.9% sulfuric acid conjugates . The two principle $metabolites \ excreted \ by \ rabbits \ after \ oral \ administration \ of \ 438 \ mg/kg/day \ for \ 5 \ days \ were \ 2,4-dimethylbenzoic \ acid \ and \ 3,4-dimethylhippuric \ acid \$

NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT & 1,2,4-TRIMETHYL BENZENE & 1,3,5-TRIMETHYL BENZENE

The major routes of excretion of 1,2,4-trimethyl- benzene are exhalation of parent compound and elimination of urinary metabolites. Half-times for urinary metabolites were reported as 9.5 hours for glycine, 22.9 hours for glucuronide, and 37.6 hours for sulfuric acid conjugates. Acute Toxicity Direct contact with liquid 1,2,4-trimethylbenzene is irritating to the skin and breathing the vapor is irritating to the respiratory tract causing pneumonitis. Breathing high concentrations of the chemical vapor causes headache, fatigue, and drowsiness. In humans liquid 1,2,4trimethylbenzene is irritating to the skin and inhalation of vapor causes chemical pneumonitis . High concentrations of vapor (5000-9000 ppm) cause headache, fatigue, and drowsiness. The concentration of 5000 ppm is roughly equivalent to a total of 221 mg/kg assuming a 30 minute exposure period (see end note 1). 2. Animals - Mice exposed to 8130-9140 ppm 1,2,4-trimethylbenzene (no duration given) had loss of righting response and loss of reflexes Direct dermal contact with the chemical (no species given) causes vasodilation, erythema, and irritation (U.S. EPA). Seven of 10 rats died after an oral dose of 2.5 mL of a mixture of trimethylbenzenes in olive oil (average dose approximately 4.4 g/kg). Rats and mice were exposed by inhalation to a coal tar distillate containing about 70% 1,3,5- and 1,2,4-trimethylbenzene; no pathological changes were noted in either species after exposure to 1800-2000 ppm for up to 48 continuous hours, or in rats after 14 exposures of 8 hours each at the same exposure levels. No effects were reported for rats exposed to a mixture of trimethyl- benzenes at 1700 ppm for 10 to 21 days Neurotoxicity 1,2,4-Trimethylbenzene depresses the central nervous system. Exposure to solvent mixtures containing the chemical causes headache, fatique, nervousness, and drowsiness, Occupationally, workers exposed to a solvent containing 50% 1,2,4-trimethylbenzene had

nervousness, headaches, drowsiness, and vertigo (U.S. EPA). Headache, fatigue, and drowsiness were reported for workers exposed (no dose given) to paint thinner containing 80% 1,2,4- and 1,3,5-trimethylbenzenes

Results of the developmental toxicity study indicate that the C9 fraction caused adverse neurological effects at the highest dose (1500 ppm) tested.

Subchronic/Chronic Toxicity Long-term exposure to solvents containing 1.2.4-trimethylbenzene may cause nervousness, tension, and bronchitis. Painters that worked for several years with a solvent containing 50% 1,2,4- and 30% 1,3,5-trimethylbenzene showed nervousness, Chemwatch: 5240-10 Page 15 of 21

Issue Date: 19/01/2023 Version No: 5.1 Print Date: 19/01/2023 Fromm Packaging Metal Marking Texpen/Dalo - All Colours

tension and anxiety, asthmatic bronchitis, anemia, and alterations in blood clotting; haematological effects may have been due to trace amounts of benzene

Rats given 1,2,4-trimethylbenzene orally at doses of 0.5 or 2.0 g/kg/day, 5 days/week for 4 weeks. All rats exposed to the high dose died and 1 rat in the low dose died (no times given); no other effects were reported. Rats exposed by inhalation to 1700 ppm of a trimethylbenzene isomeric mixture for 4 months had decreased weight gain, lymphopenia and neutrophilia

Genotoxicity: Results of mutagenicity testing, indicate that the C9 fraction does not induce gene mutations in prokaryotes (Salmonella tymphimurium/mammalian microsome assay); or in mammalian cells in culture (in Chinese hamster ovary cells with and without activation). The C9 fraction does not does not induce chromosome mutations in Chinese hamster ovary cells with and without activation; does not induce chromosome aberrations in the bone marrow of Sprague-Dawley rats exposed by inhalation (6 hours/day for 5 days); and does not induce sister chromatid exchange in Chinese hamster ovary cells with and without activation.

Developmental/Reproductive Toxicity: A three-generation reproductive study on the C9 fraction was conducted CD rats (30/sex/group) were exposed by inhalation to the C9 fraction at concentrations of 0, 100, 500, or 1500 ppm (0, 100, 500, or 1500 mg/kg/day) for 6 hours/day, 5 days/week. There was evidence of parental and reproductive toxicity at all dose levels. Indicators of parental toxicity included reduced body weights, increased salivation, hunched posture, aggressive behavior, and death. Indicators of adverse reproductive system effects included reduced litter size and reduced pup body weight. The LOEL was 100 ppm; a no-observed-effect level was not established Developmental toxicity, including possible develop- mental neurotoxicity, was evident in rats in a 3-generation reproductive study

No effects on fecundity or fertility occurred in rats treated dermally with up to 0.3 mL/rat/day of a mixture of trimethyl- benzenes, 4-6 hours/day, 5 days/week over one generation

NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT & STODDARD SOLVENT

For petroleum: This product contains benzene, which can cause acute myeloid leukaemia, and n-hexane, which can be metabolized to compounds which are toxic to the nervous system. This product contains toluene, and animal studies suggest high concentrations of toluene lead to hearing loss. This product contains ethyl benzene and naphthalene, from which animal testing shows evidence of tumour formation Cancer-causing potential: Animal testing shows inhaling petroleum causes tumours of the liver and kidney; these are however not considered to be relevant in humans.

Mutation-causing potential: Most studies involving gasoline have returned negative results regarding the potential to cause mutations, including all recent studies in living human subjects (such as in petrol service station attendants).

Reproductive toxicity: Animal studies show that high concentrations of toluene (>0.1%) can cause developmental effects such as lower birth weight and developmental toxicity to the nervous system of the foetus. Other studies show no adverse effects on the foetus. Human effects: Prolonged or repeated contact may cause defatting of the skin which can lead to skin inflammation and may make the skin more susceptible to irritation and penetration by other materials.

Animal testing shows that exposure to gasoline over a lifetime can cause kidney cancer, but the relevance in humans is questionable.

1,2,4-TRIMETHYL BENZENE & 1,3,5-TRIMETHYL BENZENE

Other Toxicity data is available for CHEMWATCH 12172 1,2,3-trimethylbenzene

SILICA AMORPHOUS & **XYLENE** The substance is classified by IARC as Group 3: NOT classifiable as to its carcinogenicity to humans.

Evidence of carcinogenicity may be inadequate or limited in animal testing.

1,3,5-TRIMETHYL BENZENE & **XYLENE**

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.

XYLENE & ETHYLBENZENE

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

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

Leaend:

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

- Data available to make classification

SECTION 12 Ecological information

Toxicity

Fromm Packaging Metal Marking Texpen/Dalo - All Colours	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
	Endpoint	Test Duration (hr)	Species	Value	Source
kaolin	Not Available	Not Available	Not Available	Not Available	Not Available
	Endpoint	Test Duration (hr)	Species	Value	Source
	BCF	1008h	Fish	<1.1-9.6	7
	EC50	72h	Algae or other aquatic plants	3.75-7.58mg/l	4
titanium dioxide	EC50	48h	Crustacea	1.9mg/l	2
	NOEC(ECx)	504h	Crustacea	0.02mg/l	4
	LC50	96h	Fish	1.85-3.06mg/l	4
	EC50	96h	Algae or other aquatic plants	179.05mg/l	2
naphtha petroleum, light aromatic solvent	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	96h	Algae or other aquatic plants	64mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	1mg/l	1

Page 16 of 21

Fromm Packaging Metal Marking Texpen/Dalo - All Colours

Issue Date: 19/01/2023 Print Date: 19/01/2023

	EC50	72h	Algae or other aquatic plants	3	19mg/l	1
	EC50	48h	Crustacea		6.14mg/l	1
	Endpoint	Test Duration (hr)	Species		Value	Sou
	BCF	1344h	Fish		31-207	7
	EC50(ECx)	96h	Algae or other aquatic plants		2.356mg/l	2
1,2,4-trimethyl benzene	EC50	48h	Crustacea		ca.6.14mg/l	1
	LC50	96h	Fish		3.41mg/l	2
	EC50	96h	Algae or other aquatic plants		2.356mg/l	2
	Endpoint	Test Duration (hr)	Species	Val	lue	Sou
	EC50	72h	Algae or other aquatic plants	>0.	2mg/l	2
carbon black	EC50	48h	Crustacea	33.	076-41.968mg/l	4
	NOEC(ECx)	24h	Crustacea	320	00mg/l	1
	LC50	96h	Fish		00mg/l	2
	Endpoint	Test Duration (hr)	Species		Value	Sou
	EC0(ECx)	24h	Crustacea		>=10000mg/l	1
	EC50	72h	Algae or other aquatic plants		14.1mg/l	2
silica amorphous	EC50	48h	Crustacea		>86mg/l	2
	LC50	96h	Fish		1033.016mg/l	2
	EC50	96h	Algae or other aquatic plants		217.576mg/l	2
	For to all of	T (D			W.L.	
	Endpoint	Test Duration (hr)	Species		Value	Sou
	BCF	1680h	Fish		23-342	7
1,3,5-trimethyl benzene	EC50	48h	Crustacea		13mg/L	5
	NOEC(ECx)	384h	Crustacea		0.257mg/l	2
	LC50	96h	Fish		5.216mg/l	2
	EC50	96h	Algae or other aquatic plants		3.084mg/l	2
	Endpoint	Test Duration (hr)	Species		Value	Sou
	NOEC(ECx)	3072h	Fish		1mg/l	1
Stoddard Solvent	NOEC(ECx)	720h	Crustacea		0.024mg/l	2
	LC50	96h	Fish		0.14mg/l	2
	EC50	96h	Algae or other aquatic plants		0.277mg/l	2
	Endpoint	Test Duration (hr)	Species		Value	Sou
	EC50	48h	Crustacea		>0.065mg/l	4
aluminium hydroxide	NOEC(ECx)	72h	Algae or other aquatic plants		>100mg/l	1
	LC50	96h	Fish		0.57mg/l	2
	EC50	96h	Algae or other aquatic plants		0.46mg/l	2
	Endpoint	Test Duration (hr)	Species		Value	Sou
	EC50	72h	Algae or other aquatic plants	S	4.6mg/l	2
xylene	EC50	48h	Crustacea		1.8mg/l	2
	NOEC(ECx)	73h	Algae or other aquatic plants	3	0.44mg/l	2
	LC50	96h	Fish		2.6mg/l	2
	Endpoint	Test Duration (hr)	Species	V	'alue	Sou
	EC50	72h	Algae or other aquatic plants	4	.6mg/l	1
	EC50	48h	Crustacea	1	.37-4.4mg/l	4
ethylbenzene	NOEC(ECx)	720h	Fish	0	.381mg/L	4
	LC50	96h	Fish	3	.381-4.075mg/L	4
	EC50	96h	Algae or other aquatic plants	3	.6mg/l	2
Legend:			CHA Registered Substances - Ecotoxicological C Aquatic Hazard Assessment Data 6. NITE (Ja			

Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

DO NOT discharge into sewer or waterways

Chemwatch: **5240-10**Version No: **5.1**

Page 17 of 21

Fromm Packaging Metal Marking Texpen/Dalo - All Colours

Issue Date: **19/01/2023**Print Date: **19/01/2023**

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
titanium dioxide	HIGH	HIGH
1,2,4-trimethyl benzene	LOW (Half-life = 56 days)	LOW (Half-life = 0.67 days)
silica amorphous	LOW	LOW
1,3,5-trimethyl benzene	HIGH	HIGH
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
ethylbenzene	HIGH (Half-life = 228 days)	LOW (Half-life = 3.57 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
titanium dioxide	LOW (BCF = 10)
1,2,4-trimethyl benzene	LOW (BCF = 275)
silica amorphous	LOW (LogKOW = 0.5294)
1,3,5-trimethyl benzene	LOW (BCF = 342)
Stoddard Solvent	LOW (BCF = 159)
xylene	MEDIUM (BCF = 740)
ethylbenzene	LOW (BCF = 79.43)

Mobility in soil

Ingredient	Mobility
titanium dioxide	LOW (KOC = 23.74)
1,2,4-trimethyl benzene	LOW (KOC = 717.6)
silica amorphous	LOW (KOC = 23.74)
1,3,5-trimethyl benzene	LOW (KOC = 703)
ethylbenzene	LOW (KOC = 517.8)

SECTION 13 Disposal considerations

Waste treatment methods

Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.

A Hierarchy of Controls seems to be common - the user should investigate:

- ► Reduction
- ► Reuse
- ► Recycling
- Disposal (if all else fails)

Product / Packaging disposal

This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.

- ▶ 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.
- $\mbox{\ensuremath{\,^{\blacktriangleright}}}$ Where in doubt contact the responsible authority.

SECTION 14 Transport information

Labels Required



Marine Pollutant



HAZCHEM

•3Y

Land transport (ADG)

UN number
UN proper shipping name

1263

PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)

Chemwatch: **5240-10** Page **18** of **21**

Version No: **5.1**

Fromm Packaging Metal Marking Texpen/Dalo - All Colours

Issue Date: 19/01/2023 Print Date: 19/01/2023

Transport hazard class(es)	Class 3 Subrisk Not Applicable		
Packing group	III		
Environmental hazard	Environmentally hazardous		
Special precautions for user	Special provisions 163 223 367 Limited quantity 5 L		

Air transport (ICAO-IATA / DGR)

UN number	1263			
UN proper shipping name	Paint related material (in liquid filler and liquid lace		ounds); Paint (including	paint, lacquer, enamel, stain, shellac, varnish, polish,
	ICAO/IATA Class	3		
Transport hazard class(es)	ICAO / IATA Subrisk	Not Applicable		
	ERG Code	3L		
Packing group	III			
Environmental hazard	Environmentally hazardo	ous		
	Special provisions		A3 A72 A192	
	Cargo Only Packing Instructions		366	
	Cargo Only Maximum Qty / Pack		220 L	
Special precautions for user	Passenger and Cargo Packing Instructions		355	
	Passenger and Cargo Maximum Qty / Pack		60 L	
	Passenger and Cargo Limited Quantity Packing Instructions		Y344	
	Passenger and Cargo	Limited Maximum Qty / Pack	10 L	

Sea transport (IMDG-Code / GGVSee)

UN number	1263
UN proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)
Transport hazard class(es)	IMDG Class 3 IMDG Subrisk Not Applicable
Packing group	III
Environmental hazard	Marine Pollutant
Special precautions for user	EMS Number F-E, S-E Special provisions 163 223 367 955 Limited Quantities 5 L

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

Not Applicable

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

Product name	Group
kaolin	Not Available
titanium dioxide	Not Available
naphtha petroleum, light aromatic solvent	Not Available
1,2,4-trimethyl benzene	Not Available
carbon black	Not Available
silica amorphous	Not Available
1,3,5-trimethyl benzene	Not Available
Stoddard Solvent	Not Available
aluminium hydroxide	Not Available
xylene	Not Available
ethylbenzene	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
kaolin	Not Available

Issue Date: **19/01/2023**Print Date: **19/01/2023**

Product name	Ship Type
titanium dioxide	Not Available
naphtha petroleum, light aromatic solvent	Not Available
1,2,4-trimethyl benzene	Not Available
carbon black	Not Available
silica amorphous	Not Available
1,3,5-trimethyl benzene	Not Available
Stoddard Solvent	Not Available
aluminium hydroxide	Not Available
xylene	Not Available
ethylbenzene	Not Available

SECTION 15 Regulatory information

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

kaolin is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List

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

naphtha petroleum, light aromatic solvent is found on the following regulatory lists

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

1,2,4-trimethyl benzene 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

carbon black is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List

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

silica amorphous is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring

Australian Inventory of Industrial Chemicals (AIIC)

1,3,5-trimethyl benzene 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

Stoddard Solvent 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

aluminium hydroxide is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

xylene 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

ethylbenzene is found on the following regulatory lists

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

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

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

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

Australian Inventory of Industrial Chemicals (AIIC)

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)

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

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

Australian Inventory of Industrial Chemicals (AIIC)

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 1: Carcinogenic to humans

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

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

Australian Inventory of Industrial Chemicals (AIIC)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

Version No: **5.1**

Fromm Packaging Metal Marking Texpen/Dalo - All Colours

Issue Date: 19/01/2023
Print Date: 19/01/2023

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) Schedule 5
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

National Inventory Status

National Inventory	Yes Yes		
Australia - AIIC / Australia Non-Industrial Use			
Canada - DSL	Yes		
Canada - NDSL	No (kaolin; naphtha petroleum, light aromatic solvent; 1,2,4-trimethyl benzene; carbon black; 1,3,5-trimethyl benzene; Stoddard Solvent; aluminium hydroxide; xylene; ethylbenzene)		
China - IECSC	Yes		
Europe - EINEC / ELINCS / NLP	Yes		
Japan - ENCS	No (kaolin)		
Korea - KECI	Yes		
New Zealand - NZIoC	Yes		
Philippines - PICCS	Yes		
USA - TSCA	Yes		
Taiwan - TCSI	Yes		
Mexico - INSQ	Yes		
Vietnam - NCI	Yes		
Russia - FBEPH	Yes		
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.		

SECTION 16 Other information

Revision Date	19/01/2023
Initial Date	24/01/2017

SDS Version Summary

Version	Date of Update	Sections Updated
4.1	23/12/2022	Classification review due to GHS Revision change.
5.1	19/01/2023	Acute Health (eye), Acute Health (inhaled), Acute Health (swallowed), Advice to Doctor, Appearance, Chronic Health, Disposal, Environmental, Ingredients, Use

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 $_{\circ}$

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

BEI: Biological Exposure Index

AIIC: Australian Inventory of Industrial Chemicals

DSL: Domestic Substances List

NDSL: Non-Domestic Substances List

IECSC: Inventory of Existing Chemical Substance in China

EINECS: European INventory of Existing Commercial chemical Substances

ELINCS: European List of Notified Chemical Substances

NLP: No-Longer Polymers

ENCS: Existing and New Chemical Substances Inventory

KECI: Korea Existing Chemicals Inventory

NZIoC: New Zealand Inventory of Chemicals

PICCS: Philippine Inventory of Chemicals and Chemical Substances

TSCA: Toxic Substances Control Act

Chemwatch: 5240-10 Page 21 of 21 Issue Date: 19/01/2023 Version No: 5.1

Fromm Packaging Metal Marking Texpen/Dalo - All Colours

Print Date: 19/01/2023

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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