

Titan Pelargonic 525 (Titan Pelargonic 525 Herbicide)

Titan Ag

Chemwatch: 7642-640

Version No: 3.1

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

Chemwatch Hazard Alert Code: 3

Initial Date: 24/09/2025

Revision Date: 05/05/2026

Print Date: 03/06/2026

L.GHS.AUS.EN.E

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

Product Identifier

Product name	Titan Pelargonic 525 (Titan Pelargonic 525 Herbicide)
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	CORROSIVE LIQUID, N.O.S. (contains pelargonic acid)
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	Agricultural herbicide.
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Details of the manufacturer or importer of the safety data sheet

Registered company name	Titan Ag
Address	Princes Street Marina, Suite 15/16 Princes Street Newport NSW 2106 Australia
Telephone	+61 2 9999 6655
Fax	+61 2 9999 0483
Website	https://www.titanag.com.au/
Email	titanorders@titanag.com.au

Emergency telephone number

Association / Organisation	Titan Ag
Emergency telephone number(s)	1800 033 111 (24 hours)
Other emergency telephone number(s)	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

COMBUSTIBLE LIQUID, regulated for storage purposes only

Poisons Schedule	S5
Classification ^[1]	Flammable Liquids Category 4, Corrosive to Metals Category 1, Skin Corrosion/Irritation Category 1B, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 1, Reproductive Toxicity Category 2, 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	Danger

Hazard statement(s)

H227	Combustible liquid.
H290	May be corrosive to metals.
H314	Causes severe skin burns and eye damage.
H317	May cause an allergic skin reaction.
H361fd	Suspected of damaging fertility. Suspected of damaging the unborn child.
H411	Toxic to aquatic life with long lasting effects.
AUH001	Explosive when dry.
AUH019	May form explosive peroxides.

Precautionary statement(s) Prevention

P202	Do not handle until all safety precautions have been read and understood.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P260	Do not breathe mist/vapours/spray.
P264	Wash all exposed external body areas thoroughly after handling.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P234	Keep only in original packaging.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P370+P378	In case of fire: Use alcohol resistant foam or fine spray/water fog to extinguish.
P302+P352	IF ON SKIN: Wash with plenty of water.
P363	Wash contaminated clothing before reuse.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P390	Absorb spillage to prevent material damage.
P391	Collect spillage.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

P403	Store in a well-ventilated place.
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.
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No further product hazard information.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
112-05-0	55-60	pelargonic acid
9016-45-9	1-10	nonylphenol ethoxylates
5989-27-5	<5	d-limonene
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▶ Immediately hold eyelids apart and flush the eye continuously with 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. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin or hair contact occurs:

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	▶ Immediately flush body and clothes with large amounts of water, using safety shower if available. ▶ Quickly remove all contaminated clothing, including footwear. ▶ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▶ Transport to hospital, or doctor.
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.
Ingestion	▶ For advice, contact a Poisons Information Centre or a doctor at once. ▶ Urgent hospital treatment is likely to be needed. ▶ 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. ▶ Transport to hospital or doctor without delay.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.
For acute or short term repeated exposures to strong acids:

- ▶ Airway problems may arise from laryngeal edema and inhalation exposure. Treat with 100% oxygen initially.
- ▶ Respiratory distress may require cricothyroidotomy if endotracheal intubation is contraindicated by excessive swelling
- ▶ Intravenous lines should be established immediately in all cases where there is evidence of circulatory compromise.
- ▶ Strong acids produce a coagulation necrosis characterised by formation of a coagulum (eschar) as a result of the dessicating action of the acid on proteins in specific tissues.

INGESTION:

- ▶ Immediate dilution (milk or water) within 30 minutes post ingestion is recommended.
- ▶ **DO NOT attempt to neutralise the acid since exothermic reaction may extend the corrosive injury.**
- ▶ Be careful to avoid further vomit since re-exposure of the mucosa to the acid is harmful. Limit fluids to one or two glasses in an adult.
- ▶ Charcoal has no place in acid management.
- ▶ Some authors suggest the use of lavage within 1 hour of ingestion.

SKIN:

- ▶ Skin lesions require copious saline irrigation. Treat chemical burns as thermal burns with non-adherent gauze and wrapping.
- ▶ Deep second-degree burns may benefit from topical silver sulfadiazine.

EYE:

- ▶ Eye injuries require retraction of the eyelids to ensure thorough irrigation of the conjunctival cul-de-sacs. Irrigation should last at least 20-30 minutes. **DO NOT use neutralising agents or any other additives.** Several litres of saline are required.
- ▶ Cycloplegic drops, (1% cyclopentolate for short-term use or 5% homatropine for longer term use) antibiotic drops, vasoconstrictive agents or artificial tears may be indicated dependent on the severity of the injury.
- ▶ Steroid eye drops should only be administered with the approval of a consulting ophthalmologist).

[Ellenhorn and Barceloux: Medical Toxicology]

SECTION 5 Firefighting measures

Extinguishing media

- ▶ Foam.
- ▶ 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
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Advice for firefighters

Fire Fighting	▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear full body protective clothing with breathing apparatus. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ 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	▶ Combustible. ▶ Slight fire hazard when exposed to heat or flame. ▶ Acids may react with metals to produce hydrogen, a highly flammable and explosive gas. ▶ Heating may cause expansion or decomposition leading to violent rupture of containers. ▶ May emit acrid smoke and corrosive fumes. Combustion products include: ▶ carbon dioxide (CO2) ▶ other pyrolysis products typical of burning organic material.
HAZCHEM	2X

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	Environmental hazard - contain spillage. Slippery when spilt. ▶ 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 spill with sand, earth, inert material or vermiculite. ▶ Wipe up. ▶ Place in a suitable, labelled container for waste disposal.																																																																															
Major Spills	Environmental hazard - contain spillage. Chemical Class:acidic compounds, organic For release onto land: recommended sorbents listed in order of priority. <table><tr><th>SORBENT TYPE</th><th>RANK</th><th>APPLICATION</th><th>COLLECTION</th><th>LIMITATIONS</th></tr><tr><td colspan="5">LAND SPILL - SMALL</td></tr><tr><td>wood fiber - pillow</td><td>1</td><td>throw</td><td>pitchfork</td><td>R, P, DGC, RT</td></tr><tr><td>cross-linked polymer - particulate</td><td>1</td><td>shovel</td><td>shovel</td><td>R,W,SS</td></tr><tr><td>cross-linked polymer - pillow</td><td>1</td><td>throw</td><td>pitchfork</td><td>R, DGC, RT</td></tr><tr><td>sorbent clay - particulate</td><td>2</td><td>shovel</td><td>shovel</td><td>R, I, P</td></tr><tr><td>foamed glass - pillow</td><td>2</td><td>throw</td><td>pitchfork</td><td>R, P, DGC, RT</td></tr><tr><td>wood fiber - particulate</td><td>3</td><td>shovel</td><td>shovel</td><td>R, W, P, DGC</td></tr><tr><td colspan="5">LAND SPILL - MEDIUM</td></tr><tr><td>cross-linked polymer -particulate</td><td>1</td><td>blower</td><td>skiploader</td><td>R, W, SS</td></tr><tr><td>polypropylene - particulate</td><td>2</td><td>blower</td><td>skiploader</td><td>W, SS, DGC</td></tr><tr><td>sorbent clay - particulate</td><td>2</td><td>blower</td><td>skiploader</td><td>R, I, P</td></tr><tr><td>cross-linked polymer - pillow</td><td>3</td><td>throw</td><td>skiploader</td><td>R, DGC, RT</td></tr><tr><td>polypropylene - mat</td><td>3</td><td>throw</td><td>skiploader</td><td>W, SS, DGC</td></tr><tr><td>expanded mineral - particulate</td><td>3</td><td>blower</td><td>skiploader</td><td>R, I, W, P, DGC</td></tr></table> Legend DGC: Not effective where ground cover is dense R; Not reusable I: Not incinerable P: Effectiveness reduced when rainy RT:Not effective where terrain is rugged SS: Not for use within environmentally sensitive sites W: Effectiveness reduced when windy Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control; R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988 Slippery when spilt. Moderate hazard. ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ No smoking, naked lights or ignition sources. ▶ Increase ventilation. ▶ Stop leak if safe to do so. ▶ Contain spill with sand, earth or vermiculite. ▶ 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.					SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS	LAND SPILL - SMALL					wood fiber - pillow	1	throw	pitchfork	R, P, DGC, RT	cross-linked polymer - particulate	1	shovel	shovel	R,W,SS	cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT	sorbent clay - particulate	2	shovel	shovel	R, I, P	foamed glass - pillow	2	throw	pitchfork	R, P, DGC, RT	wood fiber - particulate	3	shovel	shovel	R, W, P, DGC	LAND SPILL - MEDIUM					cross-linked polymer -particulate	1	blower	skiploader	R, W, SS	polypropylene - particulate	2	blower	skiploader	W, SS, DGC	sorbent clay - particulate	2	blower	skiploader	R, I, P	cross-linked polymer - pillow	3	throw	skiploader	R, DGC, RT	polypropylene - mat	3	throw	skiploader	W, SS, DGC	expanded mineral - particulate	3	blower	skiploader	R, I, W, P, DGC
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Personal Protective Equipment advice is contained in Section 8 of the SDS.

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▶ DO NOT allow clothing wet with material to stay in contact with skin ▶ Avoid skin contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ Avoid smoking, naked lights or ignition sources. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ 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.
Other information	Consider storage under inert gas. ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ No smoking, naked lights or ignition sources. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers.

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- ▶ Protect containers against physical damage and check regularly for leaks.
- ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▶ DO NOT use aluminium or galvanised containers ▶ Lined metal can, lined metal pail/ can. ▶ Plastic pail. ▶ Polyliner drum. ▶ Packing as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks. ▶ Metal can or drum ▶ Packaging as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	d-Limonene: ▶ forms unstable peroxides in storage, unless inhibited; may polymerise ▶ reacts with strong oxidisers and may explode or combust ▶ is incompatible with strong acids, including acidic clays, peroxides, halogens, vinyl chloride and iodine pentafluoride ▶ flow or agitation may generate electrostatic charges due to low conductivity ▶ The various oxides of nitrogen and peroxyacids may be dangerously reactive in the presence of alkenes. BREThERICK L.: Handbook of Reactive Chemical Hazards ▶ Avoid reaction with strong Lewis or mineral acids. ▶ Reaction with halogens requires carefully controlled conditions. ▶ Free radical initiators should be avoided. ▶ Reacts with mild steel, galvanised steel / zinc producing hydrogen gas which may form an explosive mixture with air. ▶ Avoid strong bases. Terpenoids and terpenes, are generally unsaturated, are thermolabile, are often volatile and may be easily oxidised or hydrolysed depending on their respective structure. Terpenoids are subject to autoxidation. Autoxidation is any oxidation that occurs in open air or in presence of oxygen (and sometimes UV radiation) and forms peroxides and hydroperoxides. Though autoxidation has been particularly investigated in the field of fatty oils, it also plays a most crucial part for terpenoid deterioration. Although virtually all types of organic materials can undergo air oxidation, certain types are particularly prone to autoxidation, including unsaturated compounds that have allylic or benzylic hydrogen atoms (C6H5CH2-); these materials are converted to hydroperoxides by autoxidation. Promoted by heat, catalytic quantities of redox-reactive metals, and exposure to light, autoxidation may result in the formation of explosive peroxides which may become explosive upon concentration. As a rule, however, primary autoxidation products such as hydroperoxides eventually break down during advanced stages of oxidation depending on their individual stability. Thereby they give rise to a range of stable oxidised secondary products such as mono- to polyvalent alcohols, aldehydes, ketones, epoxides, peroxides, or acids as well as highly viscous, often oxygen-bearing polymers. Light, heat, or increasing acidity often promote this breakdown. Compounds rich in allylic hydrogen atoms (2HC=CHCH2-R), found in most terpenoids, make up the most probable targets for autoxidation. Several terpenoids (typically oxygen containing derivatives) are saturated and do not react in a similar fashion to their unsaturated congeners. Thermolabile terpenoids, especially mere terpenes and aldehydes, are susceptible to rearrangement processes at elevated temperatures. Terpenic conversion reactions, upon heating, have been reported both for isolated compounds as well as for essential oils.(which tend to be rich in mono-, and sesqui-terpenes. Mono-, bi-, or tricyclic mono- terpenoids (those containing two isoprene units, dienes) and sesquiterpenoids (with three isoprene units, trienes) of different chemical classes, such as hydrocarbons, ketones, alcohols, oxides, aldehydes, phenols, or esters, make up the major part in essential oils. Electron-donating groups and increasing alkyl substitution contribute to a stronger carbon-peroxide bond through a hyperconjugative effect, thus leading to more stable and subsequently built-up hydroperoxides. Some oxygen-bearing terpenoids such as menthol, eucalyptol (1,8-cineol), and menthone do not form hydroperoxides upon oxidation but are directly converted into ketones, acids, and aldehydes. None of these are unsaturated compounds. Due to their low volatility, diterpenes (with four isoprenes, tetraenes) are barely encountered in genuine essential oils obtained by distillation, while tri- and higher terpenoids such as sterols or carotenoids are only present in the nonvolatile fractions such as plant resins or gums and will remain in the residue Aging processes generally come along with a more or less pronounced quality loss In addition to the frequent development of unpleasant and often pungent flavours, shifting colors such as the formation of a yellow staining or changes in consistency up to resinification have been reported both upon degradation of single terpenoids as well as of essential oils. · The interaction of alkenes and alkynes with nitrogen oxides and oxygen may produce explosive addition products; these may form at very low temperatures and explode on heating to higher temperatures (the addition products from 1,3-butadiene and cyclopentadiene form rapidly at -150 C and ignite or explode on warming to -35 to -15 C). These derivatives ("pseudo- nitrosites") were formerly used to characterise terpene hydrocarbons. · Exposure to air must be kept to a minimum so as to limit the build-up of peroxides which will concentrate in bottoms if the product is distilled. The product must not be distilled to dryness if the peroxide concentration is substantially above 10 ppm (as active oxygen) since explosive decomposition may occur. Distillate must be immediately inhibited to prevent peroxide formation. The effectiveness of the antioxidant is limited once the peroxide levels exceed 10 ppm as active oxygen. Addition of more inhibitor at this point is generally ineffective. Prior to distillation it is recommended that the product should be washed with aqueous ferrous ammonium sulfate to destroy peroxides; the washed product should be immediately re-inhibited. · A range of exothermic decomposition energies for double bonds is given as 40-90 kJ/mol. The relationship between energy of decomposition and processing hazards has been the subject of discussion; it is suggested that values of energy released per unit of mass, rather than on a molar basis (J/g) be used in the assessment. For example, in "open vessel processes" (with man-hole size openings, in an industrial setting), substances with exothermic decomposition energies below 500 J/g are unlikely to present a danger, whilst those in "closed vessel processes" (opening is a safety valve or bursting disk) present some danger where the decomposition energy exceeds 150 J/g. BREThERICK: Handbook of Reactive Chemical Hazards, 4th Edition · The reaction of ozone with alkenes is believed to proceed via the formation of a vibrationally excited Primary Ozonide (POZ) which falls apart to give a vibrationally excited Criegee Intermediate (CI) The CI can decompose to give OH radicals, or be stabilised. This may be of relevance in atmospheric chemistry. · Violent explosions at low temperatures in ammonia synthesis gas units have been traced to the addition products of dienes and nitrogen dioxide ▶ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

MATERIAL DATA

Continued...

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Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure. General exhaust is adequate under normal operating conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant. <table border="1"> <thead> <tr> <th>Type of Contaminant:</th><th>Air Speed:</th></tr> </thead> <tbody> <tr> <td>solvent, vapours, degreasing etc., evaporating from tank (in still air)</td><td>0.25-0.5 m/s (50-100 f/min)</td></tr> <tr> <td>aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)</td><td>0.5-1 m/s (100-200 f/min.)</td></tr> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td><td>1-2.5 m/s (200-500 f/min)</td></tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td><td>2.5-10 m/s (500-2000 f/min.)</td></tr> </tbody> </table> Within each range the appropriate value depends on: <table border="1"> <thead> <tr> <th>Lower end of the range</th><th>Upper end of the range</th></tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td><td>1: Disturbing room air currents</td></tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only</td><td>2: Contaminants of high toxicity</td></tr> <tr> <td>3: Intermittent, low production.</td><td>3: High production, heavy use</td></tr> <tr> <td>4: Large hood or large air mass in motion</td><td>4: Small hood - local control only</td></tr> </tbody> </table> Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.	Type of Contaminant:	Air Speed:	solvent, vapours, degreasing etc., evaporating from tank (in still air)	0.25-0.5 m/s (50-100 f/min)	aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min)	grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)	Lower end of the range	Upper end of the range	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents	2: Contaminants of low toxicity or of nuisance value only	2: Contaminants of high toxicity	3: Intermittent, low production.	3: High production, heavy use	4: Large hood or large air mass in motion	4: Small hood - local control only
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4: Large hood or large air mass in motion	4: Small hood - local control only																				
Individual protection measures, such as personal protective equipment																					
Eye and face protection	▶ Chemical goggles. ▶ Full face shield may be required for supplementary but never for primary protection of eyes. ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]																				
Skin protection	See Hand protection below																				
Hands/feet protection	▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber ▶ When handling corrosive liquids, wear trousers or overalls outside of boots, to avoid spills entering boots. NOTE: ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. 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. 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																				

Continued...

	· 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. ▸ Neoprene rubber gloves
Body protection	See Other protection below
Other protection	▸ Overalls. ▸ P.V.C apron. ▸ Barrier cream. ▸ Skin cleansing cream. ▸ Eye wash unit.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the: **"Forsberg Clothing Performance Index"**.
The effect(s) of the following substance(s) are taken into account in the **computer-generated** selection:
Titan Pelargonic 525 (Titan Pelargonic 525 Herbicide)

Material	CPI
NITRILE	A
PVA	A
VITON	A

* 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.

Ansell Glove Selection

Glove — In order of recommendation
AlphaTec 02-100
AlphaTec® Solvex® 37-185
AlphaTec® 58-008
AlphaTec® 58-530B
AlphaTec® 58-530W
AlphaTec® 58-735
AlphaTec® 79-700
AlphaTec® 15-554
AlphaTec® 38-612
MICROFLEX® 93-260

The suggested gloves for use should be confirmed with the glove supplier.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Light yellow clear liquid with characteristic odour; emulsifies with water.		
Physical state	Liquid	Relative density (Water = 1)	0.9
Odour	Characteristic	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	>62	Taste	Not Available

Respiratory protection

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

Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator
up to 10	1000	AB-AUS / Class1 P2	-
up to 50	1000	-	AB-AUS / Class 1 P2
up to 50	5000	Airline *	-
up to 100	5000	-	AB-2 P2
up to 100	10000	-	AB-3 P2
100+			Airline**

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

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

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Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Combustible.	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Partly miscible	pH as a solution (1%)	4-5
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available
Heat of Combustion (kJ/g)	Not Available	Ignition Distance (cm)	Not Available
Flame Height (cm)	Not Available	Flame Duration (s)	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	Enclosed Space Ignition Deflagration Density (g/m3)	Not Available

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

a) Acute Toxicity	Based on available data, the classification criteria are not met.
b) Skin Irritation/Corrosion	There is sufficient evidence to classify this material as skin corrosive or irritating.
c) Serious Eye Damage/Irritation	There is sufficient evidence to classify this material as eye damaging or irritating
d) Respiratory or Skin sensitisation	There is sufficient evidence to classify this material as sensitising to skin or the respiratory system
e) Mutagenicity	Based on available data, the classification criteria are not met.
f) Carcinogenicity	Based on available data, the classification criteria are not met.
g) Reproductivity	There is sufficient evidence to classify this material as toxic to reproductivity
h) STOT - Single Exposure	Based on available data, the classification criteria are not met.
i) STOT - Repeated Exposure	Based on available data, the classification criteria are not met.
j) Aspiration Hazard	Based on available data, the classification criteria are not met.

Inhaled	The material is not thought to produce adverse health effects following inhalation (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting. Acidic corrosives produce respiratory tract irritation with coughing, choking and mucous membrane damage. Symptoms of exposure may include dizziness, headache, nausea and weakness. In more severe exposures, pulmonary oedema may be evident either immediately or after a latent period of 5-72 hours. Symptoms of pulmonary oedema include a tightness in the chest, dyspnoea, frothy sputum and cyanosis. Examination may reveal hypotension, a weak and rapid pulse and moist rates. Death, due to anoxia, may occur several hours after onset of the pulmonary oedema.
Ingestion	The material can produce chemical burns within the oral cavity and gastrointestinal tract following ingestion. Accidental ingestion of the material may be damaging to the health of the individual. Nonionic surfactants may produce localised irritation of the oral or gastrointestinal mucosa and induce vomiting and mild diarrhoea. Ingestion of low-molecular organic acid solutions may produce spontaneous haemorrhaging, intravascular coagulation, gastrointestinal damage and oesophageal and pyloric stricture. Ingestion of acidic corrosives may produce circumoral burns with a distinct discolouration of the mucous membranes of the mouth, throat and oesophagus. Immediate pain and difficulties in swallowing and speaking may also be evident. Oedema of the epiglottis may produce respiratory distress and possibly, asphyxia. Nausea, vomiting, diarrhoea and a pronounced thirst may occur. More severe exposures may produce a vomitus containing fresh or dark blood and large shreds of mucosa. Shock, with marked hypotension, weak and rapid pulse, shallow respiration and clammy skin may be symptomatic of the exposure. Circulatory collapse may, if left untreated, result in renal failure. Severe cases may show gastric and oesophageal perforation with peritonitis, fever and abdominal rigidity. Stricture of the oesophageal, gastric and pyloric sphincter may occur as within several weeks or may be delayed for years. Death may be rapid and often results from asphyxia, circulatory collapse or aspiration of even minute amounts. Delayed deaths may be due to peritonitis, severe nephritis or pneumonia. Coma and convulsions may be terminal.
Skin Contact	The material can produce chemical burns following direct contact with the skin. Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions. One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been established that there is a connection between the potential of surfactants to denature protein in vitro and their effect on the skin. Nonionic surfactants do not carry any net charge and, therefore, they can only form hydrophobic bonds with proteins. For this reason, proteins are not deactivated by nonionic surfactants, and proteins with poor solubility are not solubilized by nonionic surfactants Open cuts, abraded or irritated skin should not be exposed to this material Skin contact with acidic corrosives may result in pain and burns; these may be deep with distinct edges and may heal slowly with the formation of scar tissue. 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.

Titan Pelargonic 525 (Titan Pelargonic 525 Herbicide)

Eye	The material can produce chemical burns to the eye following direct contact. Vapours or mists may be extremely irritating. When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation. Irritation of the eyes may produce a heavy secretion of tears (lachrymation). Some nonionic surfactants may produce a localised anaesthetic effect on the cornea; this may effectively eliminate the warning discomfort produced by other substances and lead to corneal injury. Irritant effects range from minimal to severe dependent on the nature of the surfactant, its concentration and the duration of contact. Pain and corneal damage represent the most severe manifestation of irritation. Direct eye contact with acid corrosives may produce pain, lachrymation, photophobia and burns. Mild burns of the epithelia generally recover rapidly and completely. Severe burns produce long-lasting and possible irreversible damage. The appearance of the burn may not be apparent for several weeks after the initial contact. The cornea may ultimately become deeply vascularised and opaque resulting in blindness. Dilute solutions of low-molecular organic acids cause conjunctival hyperaemia, prompt pain and corneal injury.
Chronic	Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances that can cause occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers. Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. Harmful: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests. Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects. Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects. On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Repeated or prolonged exposure to acids may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis. The impact of inhaled acidic agents on the respiratory tract depends upon a number of interrelated factors. These include physicochemical characteristics, e.g., gas versus aerosol; particle size (small particles can penetrate deeper into the lung); water solubility (more soluble agents are more likely to be removed in the nose and mouth). Given the general lack of information on the particle size of aerosols involved in occupational exposures to acids, it is difficult to identify their principal deposition site within the respiratory tract. Acid mists containing particles with a diameter of up to a few micrometers will be deposited in both the upper and lower airways. They are irritating to mucous epithelia, they cause dental erosion, and they produce acute effects in the lungs (symptoms and changes in pulmonary function). Asthmatics appear to be at particular risk for pulmonary effects. Prolonged or repeated skin contact may cause degreasing with drying, cracking and dermatitis following. A number of common flavor and fragrance chemicals can form peroxides surprisingly fast in air. Antioxidants can in most cases minimize the oxidation. Fragrance terpenes are easily oxidized in air. Non-oxidised forms are very weak sensitizers; however, after oxidation, the hydroperoxides are strong sensitizers which may cause allergic reactions. Autooxidation of fragrance terpenes contributes greatly to fragrance allergy. There is the need to test for compounds the patients are actually exposed to, not only the ingredients originally applied in commercial formulations. Hydroperoxides of d-limonene are potent contact allergens when studied in guinea pigs. They may result when d-limonene is unstabilised against oxidation, or upon prolonged standing at room temperature and/ or upon exposure to light, or when stabiliser levels diminish. The two major hydroperoxides in auto-oxidised d-limonene, are cis- and trans- limonene-2-hydroperoxide (2-hydroperoxy-p-mentha-6,8-diene). In photo-oxidised d-limonene, they represent a minor fraction. Hydroperoxides may bind to proteins of the skin to make antigens either via a radical mechanism or after reactions to give epoxides. The cross-reactivity between the epoxide limonene-1,2-oxide, a potent contact allergen, and the hydroperoxides is NOT significant, indicating different mechanisms of sensitisation. d-Limonene was considered to be weakly carcinogenic for the mouse fore-stomach epithelium, but not tumour producing. In 13-week and 2-year gavage-studies, male rats showed a range of compound-related kidney lesions including exacerbation of age-related nephropathy, mineralisation in the renal medulla, hyperplasia of the transitional epithelium overlying the renal papilla and proliferation of the renal tubular epithelium. Neoplasms were believed to be caused by progression to tubular cell hyperplasia to tubular cell adenomas and, with increasing size, to adenocarcinomas or carcinomas. The similarity of the nephrotoxicity caused by trichloroethylene and N-(4'-fluoro-4-biphenyl)acetamide, tris(2,3-dibromopropyl)phosphate in rats and the species specific nature of the response suggests that degeneration and necrosis of convoluted tubules may be associated with the accumulation of alpha-2u-globin (a2u-G). Since a2u-G is a species and gender-specific protein that is causal for both the cytotoxic and carcinogenic response in male rats, extrapolation of d-limonene carcinogenicity data from rat studies to other species (including humans) is probably not warranted. Humans do not synthesise a2u-G; they do however produce other related low molecular weight proteins capable of binding chemicals that cause a2u-G nephropathy in rats but this does not necessarily connote human risk. The Risk Assessment Forum of the USA EPA concluded; - Male renal rat tumours arising as a result of a process involving a2u-G accumulation do not contribute to the qualitative weight-of-evidence that the chemical poses a human carcinogenic hazard. Such tumours are included in dose-response extrapolations for the estimation of human carcinogenic risk. - If the chemical induces a2u-G accumulation in male rats, the associated nephropathy is not to be used as an end-point for determining non-carcinogenic hazard. Peroxidisable terpenes and terpenoids should only be used when the level of peroxides is kept to the lowest practicable level, for instance by adding antioxidants at the time of production. Such products should have a peroxide value of less than 10 millimoles peroxide per liter. This requirement is based on the published literature mentioning sensitising properties when containing peroxides. Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.

Titan Pelargonic 525 (Titan Pelargonic 525 Herbicide)	TOXICITY	IRRITATION
	Not Available	Not Available

pelargonic acid	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (Human - woman): 80%/47H - Mild
	Inhalation (Rat) LC50: >0.46<3.8 mg/l4h ^[1]	Eye (Rodent - rabbit): 100uL - Mild
	Oral (Rat) LD50: >2000 mg/kg ^[1]	Eye (Rodent - rabbit): 91mg - Severe
		Eye: adverse effect observed (irritating) ^[1]
		Skin (Human - man): 20%/48H - Mild
		Skin (Human - man): 60%/24H
		Skin (Human): 10%/47H - Mild
		Skin (Human): 20%/24H - Moderate
		Skin (Human): 80%/48H - Moderate
		Skin (Rodent - guinea pig): 100% - Severe
		Skin (Rodent - rabbit): 500mg/24H - Moderate
nonylphenol ethoxylates	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 2943.2 mg/kg ^[2]	Eye (Rodent - guinea pig): 20mg - Severe
	Oral (Rat) LD50: 1310 mg/kg ^[2]	Eye (Rodent - mouse): 20mg - Severe
		Eye (Rodent - rabbit): 100mg - Severe
		Eye (Rodent - rabbit): 15mg - Severe
		Eye (Rodent - rabbit): 20mg - Severe
		Eye (Rodent - rabbit): 5mg - Severe
		Eye (Rodent - rabbit): 5mg - Severe
		Eye (Rodent - rabbit): 5mg - Severe
		Eye (Rodent - rat): 20mg
		Skin (Human): 15mg/3D - Mild
		Skin (Rodent - rabbit): 500mg - Mild
d-limonene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >2000 mg/kg ^[1]	Skin (Rodent - guinea pig): 4pph/24H
		Skin (Rodent - mouse): 700mg/7D - Severe
		Skin (Rodent - rabbit): 10%/24H - Mild
		Skin (Rodent - rabbit): 500mg/24H - Moderate
		Skin (Rodent - rat): 100%/1H
		Skin: no adverse effect observed (not irritating) ^[1]

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

PELARGONIC ACID	for acid mists, aerosols, vapours Data from assays for genotoxic activity in vitro suggest that eukaryotic cells are susceptible to genetic damage when the pH falls to about 6.5. Cells from the respiratory tract have not been examined in this respect. Mucous secretion may protect the cells of the airways from direct exposure to inhaled acidic mists, just as mucous plays an important role in protecting the gastric epithelium from its auto-secreted hydrochloric acid. In considering whether pH itself induces genotoxic events in vivo in the respiratory system, comparison should be made with the human stomach, in which gastric juice may be at pH 1-2 under fasting or nocturnal conditions, and with the human urinary bladder, in which the pH of urine can range from <5 to > 7 and normally averages 6.2. Furthermore, exposures to low pH in vivo differ from exposures <i>in vitro</i> in that, <i>in vivo</i>, only a portion of the cell surface is subjected to the adverse conditions, so that perturbation of intracellular homeostasis may be maintained more readily than in vitro. The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration. Pelargonic acid and its Esters - Cosmetic Ingredient Review: CIR Expert Panel Meeting. June 2010 For pelargonic acid esters (branched and straight-chain): The CIR Expert Panel concluded that these esters are safe as cosmetic ingredients in the present practices of use and concentration. Pelargonic acid and nonanoate esters are cosmetic ingredients that function as skin conditioning agents in cosmetics. Pelargonic acid, a fatty acid, is absorbed and then metabolized by beta-oxidation in the body. The percutaneous absorption of isononyl alcohol was reported in an acute dermal toxicity study. Iso-fatty acids and straight-chain fatty acids both are metabolized at the beta carbon to yield two-carbon fractions by mitochondrial and microsomal fractions of rat liver homogenate. The CIR Expert Panel recognizes that the pelargonic acid branched esters included in a safety assessment are not pure substances, but are always mixtures. Thus, the international nomenclature cosmetic ingredient (INCI) name for each pelargonic acid branched ester actually
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Titan Pelargonic 525 (Titan Pelargonic 525 Herbicide)

refers to a mixture with general properties that, in many instances, cannot be fully characterized. Regardless, the material, as supplied to the industry for use in cosmetics, is the same material that is used in safety testing.

The Expert Panel noted the availability of acute inhalation toxicity data on pelargonic acid, isononanoic acid, and isononyl alcohol, but not on any other of the ingredients that are being reviewed in this safety assessment. However, in the absence of these data, the Panel determined that the ingredients included in this review can be used safely in hair sprays, because the product particle size is not respirable. The Panel reasoned that the particle size of aerosol hair sprays (around 38 µm) and pump hair sprays (>80 µm) is large compared to respirable particle sizes (≤ 10 µm). Because animal sources of cetearyl isononanoate, cetyl isononanoate, cholesteryl nonanoate, dihydrocholesteryl nonanoate, and isostearyl isononanoate have been reported, the Panel was also concerned with the dangers inherent in using animal-derived ingredients, namely the transmission of infectious agents.

The Expert Panel discussed that this group of ingredients contains both iso (branched) fatty acid esters and straight-chain fatty acid esters. While the metabolic fate of branched esters, alcohols, and fatty acids is still unknown, limited data on metabolism of iso forms versus straight-chain forms in rat liver homogenates indicated that both are oxidized in a similar fashion, but that the iso form additionally may be oxidized at an additional hydrolysis site.

Panel deliberations on the safety of pelargonic acid esters focused on current ingredient use concentration data from industry in relation to human RIPT data on cosmetic products containing these ingredients. Current ingredient use concentrations ranged from 0.01% (cholesteryl nonanoate) to 74% (ethylhexyl isononanoate), and ethylhexyl isononanoate was a mild skin irritant, but a nonsensitizer, when tested undiluted. Similarly, cetearyl nonanoate and neopentyl glycol diisononanoate, both undiluted, did not induce sensitization in human RIPTs. Other data indicate that the highest ester concentration evaluated in a human RIPT was 51.35% isodecyl isononanoate in a makeup product (nonirritant and nonsensitizer), which approaches the maximum use concentration of 59% for this ingredient. Based on negative results for sensitization at high ingredient test concentrations, the Expert Panel reasoned that is not likely that the pelargonic acid esters reviewed in this safety assessment would induce sensitization in the present practices of use https://www.cir-safety.org/sites/default/files/115_final_pelarg_rev2.pdf

For aliphatic fatty acids (and salts)

Acute oral (gavage) toxicity:

The acute oral LD50 values in rats for both were greater than >2000 mg/kg bw. Clinical signs were generally associated with poor condition following administration of high doses (salivation, diarrhoea, staining, piloerection and lethargy). There were no adverse effects on body weight in any study. In some studies, excess test substance and/or irritation in the gastrointestinal tract was observed at necropsy.

Skin and eye irritation potential, with a few stated exceptions, is chain length dependent and decreases with increasing chain length.

According to several OECD test regimes the animal skin irritation studies indicate that the C6-10 aliphatic acids are severely irritating or corrosive, while the C12 aliphatic acid is irritating, and the C14-22 aliphatic acids generally are not irritating or mildly irritating.

Human skin irritation studies using more realistic exposures (30-minute, 1-hour or 24-hours) indicate that the aliphatic acids have sufficient, good or very good skin compatibility.

Animal eye irritation studies indicate that among the aliphatic acids, the C8-12 aliphatic acids are irritating to the eye while the C14-22 aliphatic acids are not irritating.

Eye irritation potential of the ammonium salts does not follow chain length dependence; the C18 ammonium salts are corrosive to the eyes.

Dermal absorption:

The in vitro penetration of C10, C12, C14, C16 and C18 fatty acids (as sodium salt solutions) through rat skin decreases with increasing chain length. At 86.73 µg C16/cm² and 91.84 µg C18/cm², about 0.23% and less than 0.1% of the C16 and C18 soap solutions is absorbed after 24 h exposure, respectively.

Sensitisation:

No sensitisation data were located.

Repeat dose toxicity:

Repeated dose oral (gavage or diet) exposure to aliphatic acids did not result in systemic toxicity with NOAELs greater than the limit dose of 1000 mg/kg bw.

Mutagenicity

Aliphatic acids do not appear to be mutagenic or clastogenic in vitro or in vivo

Carcinogenicity

No data were located for carcinogenicity of aliphatic fatty acids.

Reproductive toxicity

No effects on fertility or on reproductive organs, or developmental effects were observed in studies on aliphatic acids and the NOAELs correspond to the maximum dose tested. The weight of evidence supports the lack of reproductive and developmental toxicity potential of the aliphatic acids category.

Given the large number of substances in this category, their closely related chemical structure, expected trends in physical chemical properties, and similarity of toxicokinetic properties, both mammalian and aquatic endpoints were filled using read-across to the closest structural analogue, and selecting the most conservative supporting substance effect level.

Structure-activity relationships are not evident for the mammalian toxicity endpoints. That is, the low mammalian toxicity of this category of substances limits the ability to discern structural effects on biological activity. Regardless, the closest structural analogue with the most conservative effect value was selected for read across. Irritation is observed for chain lengths up to a cut-off at or near 12 carbons).

Metabolism:

The aliphatic acids share a common degradation pathway in which they are metabolized to acetyl-CoA or other key metabolites in all living systems. Common biological pathways result in structurally similar breakdown products, and are, together with the physico-chemical properties, responsible for similar environmental behavior and essentially identical hazard profiles with regard to human health.

Differences in metabolism or biodegradability of even and odd numbered carbon chain compounds or saturated/unsaturated compounds are not expected; even- and odd-numbered carbon chain compounds, and the saturated and unsaturated compounds are naturally occurring and are expected to be metabolized and biodegraded in the same manner.

The acid and alkali salt forms of the homologous aliphatic acid are expected to have many similar physicochemical and toxicological properties when they become bioavailable; therefore, data read across is used for those instances where data are available for the acid form but not the salt, and vice versa. In the gastrointestinal tract, acids and bases are absorbed in the undissociated (non-ionised) form by simple diffusion or by facilitated diffusion. It is expected that both the acids and the salts will be present in (or converted to) the acid form in the stomach. This means that for both aliphatic acid or aliphatic acid salt, the same compounds eventually enter the small intestine, where equilibrium, as a result of increased pH, will shift towards dissociation (ionised form).

Hence, the situation will be similar for compounds originating from acids and therefore no differences in uptake are anticipated

Note that the saturation or unsaturation level is not a factor in the toxicity of these substances and is not a critical component of the read across process.

Toxicokinetics:

The turnover of the [14C] surfactants in the rat showed that there was no significant difference in the rate or route of excretion of 14C given by intraperitoneal or subcutaneous administration. The main route of excretion was as 14CO₂ in the expired air at 6 h after administration. The remaining material was incorporated in the body. Longer fatty acid chains are more readily incorporated than shorter chains. At ca. 1.55 and 1.64 mg/kg bw, 71% of the C16:0 and 56% of the C18:0 was incorporated and 21% and 38% was excreted as 14CO₂, respectively.

Glycidyl fatty acid esters (GEs), one of the main contaminants in processed oils, are mainly formed during the deodorisation step in the refining process of edible oils and therefore occur in almost all refined edible oils. GEs are potential carcinogens, due to the fact that they readily hydrolyze into the free form glycidol in the gastrointestinal tract, which has been found to induce tumours in various rat tissues.

Therefore, significant effort has been devoted to inhibit and eliminate the formation of GEs

GEs contain a common terminal epoxide group but exhibit different fatty acid compositions. This class of compounds has been reported in edible oils after overestimation of 3-monochloropropane-1,2-diol (3-MCPD) fatty acid esters analysed by an indirect method, 3-MCPD esters have been studied as food processing contaminants and are found in various food types and food ingredients, particularly in refined edible oils. 3-Monochloropropane-1,2-diol (3-MCPD) and 2-monochloropropane-1,3-diol (2-MCPD) are chlorinated derivatives of glycerol (1,2,3-propanetriol). 3- and 2-MCPD and their fatty acid esters are among non-volatile chloropropanols. Glycidol is associated with the formation and decomposition of 3- and 2-MCPD. It forms monoesters with fatty acids (GE) during the refining of vegetable oils. Chloropropanols are formed in HVP during the hydrochloric acid-mediated hydrolysis step of the manufacturing process. In food production, chloropropanols form from the reaction of endogenous or added chloride with glycerol or acylglycerol.

Although harmful effects on humans and animals have not been demonstrated, the corresponding hydrolysates, 3-MCPD and glycidol, have

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been identified as rodent genotoxic carcinogens, ultimately resulting in the formation of kidney tumours (3-MCPD) and tumours at other tissue sites (glycidol). Therefore, 3-MCPD and glycidol have been categorised as "possible human carcinogens (group 2B) and "probably carcinogenic to humans (group 2A), respectively, by the International Agency for Research on Cancer (IARC). Diacylglyceride (DAG) based oils produced by one company were banned from the global market due to "high levels" of GE. Several reports have also suggested that a bidirectional transformation process may occur not only between glycidol and 3-MCPD but also their esterified forms in the presence of chloride ions. The transformation rate of glycidol to 3-MCPD was higher than that of 3-MCPD to glycidol under acidic conditions in the presence of chloride ion. Precursors of GEs in refined oils have been identified as partial acylglycerols, that is, DAGs and monoacylglycerides (MAGs); however, whether they also originate from triacylglycerides (TAGs) is still a topic of controversial debates. Several authors noted that pure TAGs were stable during heat treatment (such as 235 deg C) for 3 h and were therefore not involved in the formation of GEs. However, experimental results have shown that small amounts of GEs are present in a heat-treated oil model consisting of almost 100% TAGs. The formation of GEs from TAGs can be attributed to the pyrolysis of TAGs to DAGs and MAGs. In contrast, 3-MCPD esters in refined oils can be obtained from TAG. Presently, the mechanism for the formation of GE intermediates and the relationship between GEs and 3-MCPD esters are still unknown.

**NONYLPHENOL
ETHOXYLATES**

Oral (rat) TDLo: 150 mg/kg/3D-I Skin (rabbit): 500 mg mild

For nonylphenol and its compounds:

Alkylphenols like nonylphenol and bisphenol A have estrogenic effects in the body. They are known as xenoestrogens. Estrogenic substances and other endocrine disruptors are compounds that have hormone-like effects in both wildlife and humans. Xenoestrogens usually function by binding to estrogen receptors and acting competitively against natural estrogens. Nonylphenol has been found to act as an agonist of GPER (G protein-coupled estrogen receptor). Nonylphenol has been shown to mimic the natural hormone 17beta-estradiol, and it competes with the endogenous hormone for binding with the estrogen receptors ERalpha and ERbeta.

Effects in pregnant women.

Subcutaneous injections of nonylphenol in late pregnancy causes the expression of certain placental and uterine proteins, namely CaBP-9k, which suggest it can be transferred through the placenta to the fetus. It has also been shown to have a higher potency on the first trimester placenta than the endogenous estrogen 17beta-estradiol. In addition, early prenatal exposure to low doses of nonylphenol cause an increase in apoptosis (programmed cell death) in placental cells. These "low doses" ranged from 10-13-10-9 M, which is lower than what is generally found in the environment.

Nonylphenol has also been shown to affect cytokine signaling molecule secretions in the human placenta. In vitro cell cultures of human placenta during the first trimester were treated with nonylphenol, which increase the secretion of cytokines including interferon gamma, interleukin 4, and interleukin 10, and reduced the secretion of tumor necrosis factor alpha. This unbalanced cytokine profile at this part of pregnancy has been documented to result in implantation failure, pregnancy loss, and other complications.

Effects on metabolism

Nonylphenol has been shown to act as an obesity enhancing chemical or obesogen, though it has paradoxically been shown to have anti-obesity properties. Growing embryos and newborns are particularly vulnerable when exposed to nonylphenol because low-doses can disrupt sensitive processes that occur during these important developmental periods. Prenatal and perinatal exposure to nonylphenol has been linked with developmental abnormalities in adipose tissue and therefore in metabolic hormone synthesis and release. Specifically, by acting as an estrogen mimic, nonylphenol has generally been shown to interfere with hypothalamic appetite control. The hypothalamus responds to the hormone leptin, which signals the feeling of fullness after eating, and nonylphenol has been shown to both increase and decrease eating behavior by interfering with leptin signaling in the midbrain. Nonylphenol has been shown mimic the action of leptin on neuropeptide Y and anorectic POMC neurons, which has an anti-obesity effect by decreasing eating behavior. This was seen when estrogen or estrogen mimics were injected into the ventromedial hypothalamus. On the other hand, nonylphenol has been shown to increase food intake and have obesity enhancing properties by lowering the expression of these anorexigenic neurons in the brain. Additionally, nonylphenol affects the expression of ghrelin: an enzyme produced by the stomach that stimulates appetite. Ghrelin expression is positively regulated by estrogen signaling in the stomach, and it is also important in guiding the differentiation of stem cells into adipocytes (fat cells). Thus, acting as an estrogen mimic, prenatal and perinatal exposure to nonylphenol has been shown to increase appetite and encourage the body to store fat later in life. Finally, long-term exposure to nonylphenol has been shown to affect insulin signaling in the liver of adult male rats.

Cancer

Nonylphenol exposure has also been associated with breast cancer. It has been shown to promote the proliferation of breast cancer cells, due to its agonistic activity on ERalpha (estrogen receptor alpha) in estrogen-dependent and estrogen-independent breast cancer cells.

Some argue that nonylphenol's suggested estrogenic effect coupled with its widespread human exposure could potentially influence hormone-dependent breast cancer disease

Polyethers (such as ethoxylated surfactants and polyethylene glycols) are highly susceptible to being oxidized in the air. They then form complex mixtures of oxidation products.

Animal testing reveals that whole the pure, non-oxidised surfactant is non-sensitizing, many of the oxidation products are sensitizers. The oxidation products also cause irritation.

Human beings have regular contact with alcohol ethoxylates through a variety of industrial and consumer products such as soaps, detergents, and other cleaning products. Exposure to these chemicals can occur through ingestion, inhalation, or contact with the skin or eyes. Studies of acute toxicity show that volumes well above a reasonable intake level would have to occur to produce any toxic response. Moreover, no fatal case of poisoning with alcohol ethoxylates has ever been reported. Multiple studies investigating the acute toxicity of alcohol ethoxylates have shown that the use of these compounds is of low concern in terms of oral and dermal toxicity.

Clinical animal studies indicate these chemicals may produce gastrointestinal irritation such as ulcerations of the stomach, pilo-erection, diarrhea, and lethargy. Similarly, slight to severe irritation of the skin or eye was generated when undiluted alcohol ethoxylates were applied to the skin and eyes of rabbits and rats. The chemical shows no indication of being a genotoxin, carcinogen, or mutagen (HERA 2007). No information was available on levels at which these effects might occur, though toxicity is thought to be substantially lower than that of nonylphenol ethoxylates.

Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.

Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture.

On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autooxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose allergic contact dermatitis (ACD) to these compounds by patch testing

Overall, alcohol alkoxyates (AAs) are not expected to be systemically toxic, although some short chain ethylene glycol ethers, e.g. methyl and ethyl homologues are of concern for a range of adverse health effects. They include skin and eye irritation, liver and kidney damage, bone marrow and central nervous system (CNS) depression, testicular atrophy, developmental toxicity, and immunotoxicity. For higher propyl and butyl homologues, the toxicity involves haemolysis (anaemia) with secondary effects relating to haemosiderin accumulation in the spleen, liver and kidney, and compensatory haematopoiesis in the bone marrow. Systemic toxicity was shown to decrease with increasing alkyl chain lengths and/or alkoxylation degrees (ECETOC, 2005; US EPA, 2010). The chemicals ethylene glycol hexyl ether (with a longer alkyl chain length, CAS No. 112-25-4) and diethylene glycol butyl ether (with a higher ethoxylation degree, CAS No. 112-34-5) have no evidence of systemic effects including haemolysis.

Commercially available AAs are mixtures of homologues of varying carbon chain lengths and it is possible that some of the chemicals with an average alkyl chain length $C \geq 6$ may also contain shorter alkyl chains $C < 6$. It is not practical to quantify the proportion of shorter $C < 6$ chain lengths present in such chemicals, or these shorter chain lengths may not be present at all. The available data suggest a lack of systemic toxicity for the AE chemicals with potential short alkyl chain presence (NICNASa); therefore, the toxicity of the chemicals in this assessment is unlikely to be significantly affected by the presence of shorter chain alkyl groups.

Alcohol ethoxylates are according to CESIO (2000) classified as Irritant or Harmful depending on the number of EO-units:

EO < 5 gives Irritant (Xi) with R38 (Irritating to skin) and R41 (Risk of serious damage to eyes)

EO > 5-15 gives Harmful (Xn) with R22 (Harmful if swallowed) - R38/41

EO > 15-20 gives Harmful (Xn) with R22-41

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>20 EO is not classified (CESIO 2000)

Oxo-AE, C13 EO10 and C13 EO15, are Irritating (Xi) with R36/38 (Irritating to eyes and skin) .

AE are not included in Annex 1 of the list of dangerous substances of the Council Directive 67/548/EEC

In general, alcohol ethoxylates (AE) are readily absorbed through the skin of guinea pigs and rats and through the gastrointestinal mucosa of rats. AE are quickly eliminated from the body through the urine, faeces, and expired air (CO₂). Orally dosed AE was absorbed rapidly and extensively in rats, and more than 75% of the dose was absorbed. When applied to the skin of humans, the doses were absorbed slowly and incompletely (50% absorbed in 72 hours). Half of the absorbed surfactant was excreted promptly in the urine and smaller amounts of AE appeared in the faeces and expired air (CO₂). The metabolism of C12 AE yields PEG, carboxylic acids, and CO₂ as metabolites. The LD50 values after oral administration to rats range from about 1-15 g/kg body weight indicating a low to moderate acute toxicity.

The ability of nonionic surfactants to cause a swelling of the stratum corneum of guinea pig skin has been studied. The swelling mechanism of the skin involves a combination of ionic binding of the hydrophilic group as well as hydrophobic interactions of the alkyl chain with the substrate. One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been established that there is a connection between the potential of surfactants to denature protein in vitro and their effect on the skin.

Nonionic surfactants do not carry any net charge and, therefore, they can only form hydrophobic bonds with proteins. For this reason, proteins are not deactivated by nonionic surfactants, and proteins with poor solubility are not solubilized by nonionic surfactants. A substantial amount of toxicological data and information in vivo and in vitro demonstrates that there is no evidence for alcohol ethoxylates (AEs) being genotoxic, mutagenic or carcinogenic. No adverse reproductive or developmental effects were observed. The majority of available toxicity studies revealed NOAELs in excess of 100 mg/kg bw/d but the lowest NOAEL for an individual AE was established to be 50 mg/kg bw/day. This value was subsequently considered as a conservative, representative value in the risk assessment of AE. The effects were restricted to changes in organ weights with no histopathological organ changes with the exception of liver hypertrophy (indicative of an adaptive response to metabolism rather than a toxic effect). It is noteworthy that there was practically no difference in the NOAEL in oral studies of 90-day or 2 years of duration in rats. A comparison of the aggregate consumer exposure and the systemic NOAEL (taking into account an oral absorption value of 75%) results in a Margin of Exposure of 5,800. Taking into account the conservatism in the exposure assessment and the assigned systemic NOAEL, this margin of exposure is considered more than adequate to account for the inherent uncertainty and variability of the hazard database and inter and intra-species extrapolations.

AEs are not contact sensitizers. Neat AE are irritating to eyes and skin. The irritation potential of aqueous solutions of AEs depends on concentrations. Local dermal effects due to direct or indirect skin contact in certain use scenarios where the products are diluted are not of concern as AEs are not expected to be irritating to the skin at in-use concentrations. Potential irritation of the respiratory tract is not a concern given the very low levels of airborne AE generated as a consequence of spray cleaner aerosols or laundry powder detergent dust.

In summary, the human health risk assessment has demonstrated that the use of AE in household laundry and cleaning detergents is safe and does not cause concern with regard to consumer use.

For high boiling ethylene glycol ethers (typically triethylene- and tetraethylene glycol ethers):

Skin absorption: Available skin absorption data for triethylene glycol ether (TGBE), triethylene glycol methyl ether (TGME), and triethylene glycol ethylene ether (TGEE) suggest that the rate of absorption in skin of these three glycol ethers is 22 to 34 micrograms/cm²/hr, with the methyl ether having the highest permeation constant and the butyl ether having the lowest. The rates of absorption of TGBE, TGEE and TGME are at least 100-fold less than EGME, EGEE, and EGBE, their ethylene glycol monoalkyl ether counterparts, which have absorption rates that range from 214 to 2890 micrograms/cm²/hr. Therefore, an increase in either the chain length of the alkyl substituent or the number of ethylene glycol moieties appears to lead to a decreased rate of percutaneous absorption. However, since the ratio of the change in values of the ethylene glycol to the diethylene glycol series is larger than that of the diethylene glycol to triethylene glycol series, the effect of the length of the chain and number of ethylene glycol moieties on absorption diminishes with an increased number of ethylene glycol moieties. Therefore, although tetraethylene glycol methyl ether (TetraME) and tetraethylene glycol butyl ether (TetraBE) are expected to be less permeable to skin than TGME and TGBE, the differences in permeation between these molecules may only be slight.

Metabolism: The main metabolic pathway for metabolism of ethylene glycol monoalkyl ethers (EGME, EGEE, and EGBE) is oxidation via alcohol and aldehyde dehydrogenases (ALD/ADH) that leads to the formation of an alkoxy acids. Alkoxy acids are the only toxicologically significant metabolites of glycol ethers that have been detected *in vivo*. The principal metabolite of TGME is believed to be 2-[2-(2-methoxyethoxy)ethoxy] acetic acid. Although ethylene glycol, a known kidney toxicant, has been identified as an impurity or a minor metabolite of glycol ethers in animal studies it does not appear to contribute to the toxicity of glycol ethers.

The metabolites of category members are not likely to be metabolized to any large extent to toxic molecules such as ethylene glycol or the mono alkoxy acids because metabolic breakdown of the ether linkages also has to occur

Acute toxicity: Category members generally display low acute toxicity by the oral, inhalation and dermal routes of exposure. Signs of toxicity in animals receiving lethal oral doses of TGBE included loss of righting reflex and flaccid muscle tone, coma, and heavy breathing. Animals administered lethal oral doses of TGEE exhibited lethargy, ataxia, blood in the urogenital area and piloerection before death.

Irritation: The data indicate that the glycol ethers may cause mild to moderate skin irritation. TGEE and TGBE are highly irritating to the eyes. Other category members show low eye irritation.

Repeat dose toxicity: Results of these studies suggest that repeated exposure to moderate to high doses of the glycol ethers in this category is required to produce systemic toxicity

In a 21-day dermal study, TGME, TGEE, and TGBE were administered to rabbits at 1,000 mg/kg/day. Erythema and oedema were observed. In addition, testicular degeneration (scored as trace in severity) was observed in one rabbit given TGEE and one rabbit given TGME.

Testicular effects included spermatid giant cells, focal tubular hypospermatogenesis, and increased cytoplasmic vacuolisation. Due to a high incidence of similar spontaneous changes

in normal New Zealand White rabbits, the testicular effects were considered not to be related to treatment. Thus, the NOAELs for TGME, TGEE and TGBE were established at 1000 mg/kg/day. Findings from this report were considered unremarkable.

A 2-week dermal study was conducted in rats administered TGME at doses of 1,000, 2,500, and 4,000 mg/kg/day. In this study, significantly-increased red blood cells at 4,000 mg/kg/day and significantly-increased urea concentrations in the urine at 2,500 mg/kg/day were observed. A few of the rats given 2,500 or 4,000 mg/kg/day had watery caecal contents and/or haemolysed blood in the stomach. These gross pathologic observations were not associated with any histologic abnormalities in these tissues or alterations in haematologic and clinical chemistry parameters. A few males and females treated with either 1,000 or 2,500 mg/kg/day had a few small scabs or crusts at the test site. These alterations were slight in degree and did not adversely affect the rats.

In a 13-week drinking water study, TGME was administered to rats at doses of 400, 1,200, and 4,000 mg/kg/day. Statistically-significant changes in relative liver weight were observed at 1,200 mg/kg/day and higher. Histopathological effects included hepatocellular cytoplasmic vacuolisation (minimal to mild in most animals) and hypertrophy (minimal to mild) in males at all doses and hepatocellular hypertrophy (minimal to mild) in high dose females. These effects were statistically significant at 4,000 mg/kg/day. Cholangiofibrosis was observed in 7/15 high-dose males; this effect was observed in a small number of bile ducts and was of mild severity. Significant, small decreases in total test session motor activity were observed in the high-dose animals, but no other neurological effects were observed. The changes in motor activity were secondary to systemic toxicity

Mutagenicity: Mutagenicity studies have been conducted for several category members. All in vitro and in vivo studies were negative at concentrations up to 5,000 micrograms/plate and 5,000 mg/kg, respectively, indicating that the category members are not genotoxic at the concentrations used in these studies. The uniformly negative outcomes of various mutagenicity studies performed on category members lessen the concern for carcinogenicity.

Reproductive toxicity: Although mating studies with either the category members or surrogates have not been performed, several of the repeated dose toxicity tests with the surrogates have included examination of reproductive organs. A lower molecular weight glycol ether, ethylene glycol methyl ether (EGME), has been shown to be a testicular toxicant. In addition, results of repeated dose toxicity tests with TGME clearly show testicular toxicity at an oral dose of 4,000 mg/kg/day four times greater than the limit dose of 1,000 mg/kg/day recommended for repeat dose studies. It should be noted that TGME is 350 times less potent for testicular effects than EGME. TGBE is not associated with testicular toxicity, TetraME is not likely to be metabolised by any large extent to 2-MAA (the toxic metabolite of EGME), and a mixture containing predominantly methylated glycol ethers in the C5-C11 range does not produce testicular toxicity (even when administered intravenously at 1,000 mg/kg/day).

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Developmental toxicity: The bulk of the evidence shows that effects on the foetus are not noted in treatments with . 1,000 mg/kg/day during gestation. At 1,250 to 1,650 mg/kg/day TGME (in the rat) and 1,500 mg/kg/day (in the rabbit), the developmental effects observed included skeletal variants and decreased body weight gain.

for nonylphenol:

Nonylphenol was studied for oral toxicity in rats in a 28-day repeat dose toxicity test at doses of 0, 4, 15, 60 and 250 mg/kg/day. Changes suggesting renal dysfunction were mainly noted in both sexes given 250 mg/kg. Liver weights were increased in males given 60 mg/kg and in both sexes given 250 mg/kg group. Histopathologically, hypertrophy of the centrilobular hepatocytes was noted in both sexes given 250 mg/kg. Kidney weights were increased in males given 250 mg/kg and macroscopically, disseminated white spots, enlargement and pelvic dilatation were noted in females given 250 mg/kg. Histopathologically, the following lesions were noted in the 250 mg/kg group: basophilic change of the proximal tubules in both sexes, single cell necrosis of the proximal tubules, inflammatory cell infiltration in the interstitium and casts in females, basophilic change and dilatation of the collecting tubules in both sexes, simple hyperplasia of the pelvic mucosa and pelvic dilatation in females. In the urinary bladder, simple hyperplasia was noted in both sexes given 250 mg/kg. In the caecum, macroscopic dilatation was noted in both sexes given 250 mg/kg. Almost all changes except those in the kidney disappeared after a 14-day recovery period. The NOELs for males and females are considered to be 15 mg/kg/day and 60 mg/kg/day, respectively, under the conditions of the present study.

Nonylphenol was not mutagenic to *Salmonella typhimurium*, TA100, TA1535, TA98, TA1537 and *Escherichia coli* WP2 uvrA, with or without an exogenous metabolic activation system.

Nonylphenol induced neither structural chromosomal aberrations nor polyploidy in CHL/IU cells, in the absence or presence of an exogenous metabolic activation system.

D-LIMONENE

Tumorigenic by RTECS criteria

The following information refers to contact allergens as a group and may not be specific to this product.

Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.

Epoxidation of double bonds is a common bioactivation pathway for alkenes. The allylic epoxides, so formed, were found to possess sensitising capacity in vivo and in vitro and to chemically reactive towards a common hexapeptide containing the most common nucleophilic amino acids. Further-more, a SAR study of potentially prohaptenic alkenes demonstrated that conjugated dienes in or in conjunction with a six-membered ring are prohaptens, whereas related alkenes containing isolated double bonds or an acyclic conjugated diene were weak or nonsensitizing compounds. This difference in sensitizing capacity of conjugated dienes as compared to alkenes with isolated double bonds was found to be due to the high reactivity and sensitizing capacity of the allylic epoxides metabolically formed from conjugated dienes.

Allergic Contact Dermatitis—Formation, Structural Requirements, and Reactivity of Skin Sensitizers.

Ann-Therese Karlberg et al: Chem. Res. Toxicol. 2008, 21, pp 53–69

https://ftp.cdc.gov/pub/Documents/OEL/06.%20Dotson/References/Karlberg_2008.pdf

Adverse reactions to fragrances in perfumes and in fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, photosensitivity, immediate contact reactions (contact urticaria), and pigmented contact dermatitis. Airborne and connubial contact dermatitis occur.

Intolerance to perfumes, by inhalation, may occur if the perfume contains a sensitising principal. Symptoms may vary from general illness, coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea, acute respiratory illness, hayfever, and other respiratory diseases (including asthma). Perfumes can induce hyper-reactivity of the respiratory tract without producing an IgE-mediated allergy or demonstrable respiratory obstruction. This was shown by placebo-controlled challenges of nine patients to "perfume mix". The same patients were also subject to perfume provocation, with or without a carbon filter mask, to ascertain whether breathing through a filter with active carbon would prevent symptoms. The patients breathed through the mouth, during the provocations, as a nose clamp was used to prevent nasal inhalation. The patient's earlier symptoms were verified; breathing through the carbon filter had no protective effect. The symptoms were not transmitted via the olfactory nerve but they may have been induced by trigeminal reflex via the respiratory tract or by the eyes. Cases of occupational asthma induced by perfume substances such as isoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to give persistent symptoms even though the exposure is below occupational exposure limits.

Inhalation intolerance has also been produced in animals. The emissions of five fragrance products, for one hour, produced various combinations of sensory irritation, pulmonary irritation, decreases in expiratory airflow velocity as well as alterations of the functional observational battery indicative of neurotoxicity in mice. Neurotoxicity was found to be more severe after mice were repeatedly exposed to the fragrance products, being four brands of cologne and one brand of toilet water.

Contact allergy to fragrances is relatively common, affecting 1 to 3% of the general population, based on limited testing with eight common fragrance allergens and about 16 % of patients patch tested for suspected allergic contact dermatitis.

Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a sufficient degree of fragrance contact allergens. Contact allergy is a life-long, specifically altered reactivity in the immune system. This means that once contact allergy is developed, cells in the immune system will be present which can recognise and react towards the allergen. As a consequence, symptoms, i.e. allergic contact dermatitis, may occur upon re-exposure to the fragrance allergen(s) in question. Allergic contact dermatitis is an inflammatory skin disease characterised by erythema, swelling and vesicles in the acute phase. If exposure continues it may develop into a chronic condition with scaling and painful fissures of the skin. Allergic contact dermatitis to fragrance ingredients is most often caused by cosmetic products and usually involves the face and/or hands. It may affect fitness for work and the quality of life of the individual. Fragrance contact allergy has long been recognised as a frequent and potentially disabling problem. Prevention is possible as it is an environmental disease and if the environment is modified (e.g. by reduced use concentrations of allergens), the disease frequency and severity will decrease. Fragrance contact allergy is mostly non-occupational and related to the personal use of cosmetic products. Allergic contact dermatitis can be severe and widespread, with a significant impairment of quality of life and potential consequences for fitness for work. Thus, prevention of contact sensitisation to fragrances, both in terms of primary prevention (avoiding sensitisation) and secondary prevention (avoiding relapses of allergic contact dermatitis in those already sensitised), is an important objective of public health risk management measure.

Hands: Contact sensitisation may be the primary cause of hand eczema, or may be a complication of irritant or atopic hand eczema. The number of positive patch tests has been reported to correlate with the duration of hand eczema, indicating that long-standing hand eczema may often be complicated by sensitisation. Fragrance allergy may be a relevant problem in patients with hand eczema; perfumes are present in consumer products to which their hands are exposed. A significant relationship between hand eczema and fragrance contact allergy has been found in some studies based on patients investigated for contact allergy. However, hand eczema is a multi-factorial disease and the clinical significance of fragrance contact allergy in (severe) chronic hand eczema may not be clear.

Axillae Bilateral axillary (underarm) dermatitis may be caused by perfume in deodorants and, if the reaction is severe, it may spread down the arms and to other areas of the body. In individuals who consulted a dermatologist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy.

Face Facial eczema is an important manifestation of fragrance allergy from the use of cosmetic products (16). In men, after-shave products can cause an eczematous eruption of the beard area and the adjacent part of the neck and men using wet shaving as opposed to dry have been shown to have an increased risk of being fragrance allergic.

Irritant reactions (including contact urticaria): Irritant effects of some individual fragrance ingredients, e.g. citral are known. Irritant contact dermatitis from perfumes is believed to be common, but there are no existing investigations to substantiate this. Many more people complain about intolerance or rashes to perfumes/perfumed products than are shown to be allergic by testing. This may be due to irritant effects or inadequate diagnostic procedures. Fragrances may cause a dose-related contact urticaria of the non-immunological type (irritant contact urticaria). Cinnamal, cinnamic alcohol, and Myroxylon pereirae are well recognised causes of contact urticaria, but others, including menthol, vanillin and benzaldehyde have also been reported. The reactions to Myroxylon pereirae may be due to cinnamates. A relationship to delayed contact hypersensitivity was suggested, but no significant difference was found between a fragrance-allergic group and a control group in the frequency of immediate reactions to fragrance ingredients in keeping with a nonimmunological basis for the reactions seen.

Pigmentary anomalies: The term "pigmented cosmetic dermatitis" was introduced in 1973 for what had previously been known as melanosis faciei feminae when the mechanism (type IV allergy) and causative allergens were clarified.. It refers to increased pigmentation,

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usually on the face/neck, often following sub-clinical contact dermatitis. Many cosmetic ingredients were patch tested at non-irritant concentrations and statistical evaluation showed that a number of fragrance ingredients were associated: jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol, geranium oil.

Photo-reactions Musk ambrette produced a considerable number of allergic photocontact reactions (in which UV-light is required) in the 1970s and was later banned from use in the EU. Nowadays, photoallergic contact dermatitis is uncommon. Furocoumarins (psoralens) in some plant-derived fragrance ingredients caused phototoxic reactions with erythema followed by hyperpigmentation resulting in Berloque dermatitis. There are now limits for the amount of furocoumarins in fragrance products. Phototoxic reactions still occur but are rare.

General/respiratory: Fragrances are volatile and therefore, in addition to skin exposure, a perfume also exposes the eyes and naso-respiratory tract. It is estimated that 2-4% of the adult population is affected by respiratory or eye symptoms by such an exposure. It is known that exposure to fragrances may exacerbate pre-existing asthma. Asthma-like symptoms can be provoked by sensory mechanisms. In an epidemiological investigation, a significant association was found between respiratory complaints related to fragrances and contact allergy to fragrance ingredients, in addition to hand eczema, which were independent risk factors in a multivariate analysis.

Monomethyltin chloride, thioglycolate esters, and tall oil ester reaction product:

Monomethyltin trichloride (MMTC, CAS RN: 993-16-8), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (EHTG); MMT (2-EHMA), CAS RN: 57583-34-3), monomethyltin tris[isooctylmercaptoacetate (MMT(IOTG), CAS RN: 54849-38-6) and methyltin reverse ester tallate reaction product (TERP, CAS RNs: 201687-58-3, 201687-57-2, 68442-12-6, 151436-98-5) are considered one category of compounds for mammalian studies via the oral route. The justification for this category is based on structural similarities and the demonstrated rapid conversion of all of the esters to the MMTC when placed in simulated mammalian gastric contents [0.07M HCl] under physiological conditions. For the MMT(EHTG) >90% conversion to MMTC occurred within 0.5 hours. For TERP, 68% of the monomethyltin portion of the compound was converted to MMTC within 1 hour. Thus, MMTC is the appropriate surrogate for mammalian toxicology studies via the oral route.

TERP is a reaction product of MMTC and dimethyltin dichloride (DMTC), Na₂S, and tall oil fatty acid [a mixture of carboxylic acids, predominantly C-18]. The reaction product is a mixture of carboxylic esters and includes short oligomers of mono/dimethyltins bridged by sulfide groups. Although the tall oil component of TERP is not structurally similar to EHTG, TERP's conversion to MMTC justifies its inclusion. While the contribution of the various ligands to the overall toxicity may vary, the contribution is expected to be small relative to that of the MMTC. Further, the EHTG ligand from MMT(EHTG) is likely to be more toxic than the oleic or linoleic acid from TERP so inclusion of TERP in the category is a rather conservative approach. The other possible degradate of tall oil and EHTG is 2-mercaptoethanol (2-ME), and it is common to both ligands.

Data for MMT(EHTG) and MMT(IOTG) are used interchangeably because they are isomers differing only slightly in the structure of the C-8 alcohol of the mercaptoester ligand. In addition, the breakdown products of MMT(EHTG) and MMT(IOTG) are the thioglycolate esters (EHTG and IOTG), which have the common degradates, thioglycolic acid and C-8 alcohols (either 2-ethylhexanol or isooctanol). EHTG and IOTG also have similar physicochemical and toxicological properties.

The chemistry of the alkyl organotin has been well studied. For organotin, like MMT(EHTG), the alkyl groups are strongly bound to tin and remain bound to tin under most reaction conditions. However, other ligands, such as carboxylates or sulfur based ligands (EHTG), are more labile and are readily replaced under mild reaction conditions. To assess the reactivity of MMT(EHTG) under physiological conditions simulating the mammalian stomach, an in-vitro hydrolysis test was performed. This in vitro test provides chemical information that strongly suggests both the probable in vivo metabolic pathway and the toxicokinetics of the MMT(EHTG) substance. This result verifies that under physiological conditions MMT(EHTG) is rapidly and essentially completely converted to the corresponding monomethyltin chloride, MMTC.

Acute toxicity:

The majority of toxicology studies were conducted with commercial mixtures having high monoalkyltin to dialkyltin ratios.

Gastric hydrolysis studies were conducted with TERP and MMT(EHTG) in which simulated gastric fluid [0.07M HCl under physiological conditions] converted these substances to methyltin chloride and the respective organic acids. Based on data for DMTC and DMT esters the dermal penetration of MMTC and its esters is expected to be low.

Oral:

Acute oral LD₅₀ values for MMTC, MMT(EHTG), MMT(IOTG), and TERP indicated low to moderate toxicity; the most reliable data place the LD₅₀s in the range of 1000 mg/kg.

The acute oral LD₅₀ of MMT(2-EHMA) was 880 mg/kg in rats. Clinical observations included depression, comatose, piloerection, eye squinting, hunched posture, laboured breathing, ataxia, faecal/urine stains, and masticatory movement. No gross pathological changes were reported in surviving animals.

Dermal

Acute dermal LD₅₀ values were =1000 mg/kg bw, and inhalation LC₅₀ was >200 mg/L. MMTC was corrosive to skin and assumed corrosive to eyes.

The acute dermal LD₅₀ of MMT(2-EHMA) in rabbits was 1000 (460 to 2020) mg/kg for females and 2150 (1000 to 4620) mg/kg for males.

There were no deaths at 215 and 464 mg/kg, 0/2 males and 1/2 females died at 1000 mg/kg and 1/2 males and 2/2 females died at 2150 mg/kg. All animals died at 4640 and 10 000 mg/kg. A variety of clinical abnormalities were observed and disappeared in surviving animals by the end of the exposure period. Clinical signs included death, uncoordinated movements, shaking, and hypersensitivity to external stimuli.

Gross necropsy results for animals that died during the study included irritated intestines; blanched stomach; reddened lungs; pale or congested kidneys; and oral, ocular and/or nasal discharges

Inhalation:

The acute inhalation LC₅₀ of MMT(2-EHMA) was 240 mg/L.

The study reported an acute inhalation LC₅₀ of 240 (212 to 271) mg/L in a 1-hr aerosol exposure to male and female rats. The mortality rate was 2/10, 6/10, 9/10 and 10/10 animals at dose levels of 200, 250, 300 and 250 mg/L/hr, respectively. Gross findings included blood in lungs, dark spleen, pale kidneys, fluid in the chest cavity, and heart failure. The slope of the dose-response curve was 1.22 (1.04 to 1.43).

Irritation:

MMT(IOTG)/(EHTG) are irritating to skin, but not to eyes.

Sensitisation:

No data on sensitization are available on MMT(EHTG)/(IOTG), but the hydrolysis products EHTG or IOTG are sensitizers. No primary irritation data were available for TERP, but it was a sensitizer in the mouse Local Lymph Node Assay.

Topical application with 5, 25 and 50 % v/v MMT(2-EHMA) elicited a stimulation index (SI) of 2.13, 7.25 and 9.05, respectively in a local lymph node assay (OECD 429), thus the material is a sensitizer.

Repeat dose toxicity:

There are no repeated-dose studies for the category members via the dermal or inhalation routes.

In a 90-day repeated dose oral study of MMTC, treatment-related changes were limited to the high dose group (750 ppm in diet; 50 mg/kg bw/d with some gender-related variation). Organ weight changes (adrenal, kidney, thymus, spleen, brain, epididymides), haematology, clinical chemistry, and urinalysis changes were noted, but histopathology only confirmed effects in the thymus and brain. The critical toxic effects were neurotoxicity and thymic atrophy. Both sexes had decreased cortex/medulla ratios in the thymus. In the brain there was loss of perikarya of neuronal cells in the pyramidal layer of the Hippocampus CA1/2 in both sexes, and in males there was loss of perikarya in the piriform cortex. The NOAEL was 150 ppm (10 mg/kg bw/d). Another 90-day dietary study using MMTC showed increased relative kidney weights and slight to moderate epithelial hyperplasia of the bladder in females at the lowest dose (NOAEL <20 ppm in diet [$<1.3.6$ mg/kg bw/d]) and additional effects including increased relative thymus weights in females and urinalysis results in both sexes at higher doses.

A 90-day dietary study with dose levels of 30, 100, 300, and 1000 ppm TERP in the diet resulted in slightly decreased food intake, body and organ weight changes, and decreased specific gravity of the urine at the highest dose. The NOAEL was 300 ppm in diet (equivalent to 15 mg/kg bw/d). A 28-day gavage study using TERP showed changes in clinical chemistry and slight differences in haematology at 150 mg/kg bw/d and higher. The NOAEL was 50 mg/kg bw/d.

The effects of MMT(IOTG) were evaluated in a 90-day dietary study using doses of 100, 500, and 1500 ppm (decreased from 2500 ppm) in the diet. Based on clinical chemistry effects at 500 ppm and other effects at higher doses, the NOAEL was 100 ppm in diet (approximately 6-21 mg/kg bw/d).

Neurotoxicity:

In a guideline 90-day subchronic dietary study conducted in Wistar rats, effects occurred at the high dose of 750 ppm MMT(2-EHMA, (equivalent to 49.7 mg/kg bw/day in males and 53.6 mg/kg bw/day in females), which consisted of changes in neurobehavioral parameters and associated brain histopathology. The NOAEL was the next lower dose of 150 ppm (equivalent to 9.8 mg/kg bw/day in males and 10.2 mg/kg bw/day in females)

Immunotoxicity:

Immune function was assessed in male Sprague-Dawley rats exposed to the mixture of organotin used in PVC pipe production.

Continued...

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Adult male rats were given drinking water for 28 d containing a mixture of dibutyltin dichloride (DBTC), dimethyltin dichloride (DMTC), monobutyltin trichloride (MBT), and monomethyltin trichloride (MMT) in a 2:2:1:1 ratio, respectively, at 3 different concentrations (5:5:2.5:2.5, 10:10:5:5, or 20:20:10:10 mg organotin/L). Rats were also exposed to MMT alone (20 or 40 mg MMT/L) or plain water as a control. Delayed-type hypersensitivity, antibody synthesis, and natural killer cell cytotoxicity were evaluated in separate endpoint groups immediately after exposure ended.

The evaluated immune functions were not affected by the mixture or by MMT alone. The data suggest that immunotoxicity is unlikely to result from the concentration of organotin present in drinking water delivered via PVC pipes, as the concentrations used were several orders of magnitude higher than those expected to leach from PVC pipes

Genotoxicity:

In a guideline 90-day subchronic dietary study in rats, with MMT(2-EHMA), based on the changes in neurobehavioral parameters and associated brain histopathology that occurred at the high dose of 750 ppm (equivalent to 49.7 mg/kg bw/day in males and 53.6 mg/kg bw/day in females), as well as changes in haematology, clinical chemistry, urinalysis, organ weights, and pathology of the thymus at the same dose, the NOAEL was the next lower dose of 150 ppm (equivalent to 9.8 mg/kg bw/day in males and 10.2 mg/kg bw/day in females). The monomethyltin compounds as a class are not mutagenic in the Ames test. TERP was positive in a human lymphocyte assay. MMTC was equivocal for induction of micronucleated polychromatic erythrocytes (MPEs) in an in vivo rat micronucleus test (OECD 474). In this study a statistically significant increase in MPE was observed only at 24 h and not at 48 h after treatment and there was no dose-response. Based on these observations the overall conclusion is that MMTC does not have genotoxic potential.

From the results obtained in a micronucleus test with MMT(2-EHMA), it was demonstrated that the substance was weakly genotoxic to bone marrow cells of rats and that the substance has the potential to induce damage to the mitotic spindle apparatus of the bone marrow target cells.

Carcinogenicity:

In a limited carcinogenicity study, MMT(EHTG) produced no compound-related macroscopic or microscopic changes in rats fed 100 ppm in the diet for two years.

Toxicity to reproduction:

In the reproductive satellite portion of the 90-day study using MMTC (with dose levels of 30, 150, and 750 ppm in the diet), post-implantation loss, decreased litter size and increased neonatal mortality occurred at 750 ppm (26-46 mg/kg bw/d for females). Maternal gestational body weights were transiently suppressed and other maternal toxicity was inferred from the repeated dose results at this dose. There were no malformations observed at any dose. The NOAEL for maternal toxicity, and reproductive, and foetotoxic effects was 150 ppm in the diet (6-12 mg/kg bw/d).

SIDS Initial Assessment Profile (SIAM 23 2006)

ECHA Registration Dossier for MMT(2-EHMA) (ethylhexyl 10-ethyl-4-[[2-[(2-ethylhexyl)oxy]-2-oxoethyl]thio]-4-methyl-7-oxo-8-oxa-3,5-dithia-4-stannatetradecanoate)

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.

Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein.

However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A **prehapten** is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems.

In the case of prehapten, it is possible to prevent activation outside the body to a certain extent by different measures, e.g. prevention of air exposure during handling and storage of the ingredients and the final product, and by the addition of suitable antioxidants. When antioxidants are used, care should be taken that they will not be activated themselves and thereby form new sensitisers.

Prehapten

Most terpenes with oxidisable allylic positions can be expected to autoxidise on air exposure due to their inherent properties. Depending on the stability of the oxidation products that are formed, a difference in the sensitisation potency of the oxidised terpenes can be seen. Autoxidation is a free radical chain reaction in which hydrogen atom abstraction in combination with addition of oxygen forms peroxy radicals. The reaction shows selectivity for positions where stable radicals can be formed. So far, all fragrance substances that have been investigated with regard to the influence of autoxidation on the allergenic potential, including identification of formed oxidation products, have oxidisable allylic positions that are able to form hydroperoxides and/or hydrogen peroxide as primary oxidation products upon air exposure. Once the hydroperoxides have been formed outside the skin they form specific antigens and act as skin sensitisers. Secondary oxidation products such as aldehydes and epoxides can also be allergenic, thus further increasing the sensitisation potency of the autoxidation mixture. The process of photoactivation may also play a role, but further research is required to establish whether this activation route is currently underestimated in importance due to insufficient knowledge of the true haptens in this context.

It should be noted that activation of substances via air oxidation results in various haptens that might be the same or cross-reacting with other haptens (allergens). The main allergens after air oxidation of linalool and linalyl acetate are the hydroperoxides. If linalyl acetate is chemically hydrolysed outside the skin it can thereafter be oxidised to the same haptens as seen for linalool. A corresponding example is citronellol and citronellyl acetate. In clinical studies, concomitant reactions to oxidised linalool and oxidised linalyl acetate have been observed. Whether these reactions depend on cross-reactivity or are due to exposure to both fragrance substances cannot be elucidated as both have an allergenic effect themselves. Linalool and linalyl acetate are the main components of lavender oil. They autoxidise on air exposure also when present in the essential oil, and form the same oxidation products found in previous studies of the pure synthetic terpenes. Experimental sensitisation studies showed that air exposure of lavender oil increased the sensitisation potency. Patch test results in dermatitis patients showed a connection between positive reactions to oxidised linalool, linalyl acetate and lavender oil.

Prohapten

Compounds that are bioactivated in the skin and thereby form haptens are referred to as prohapten.

In the case of prohapten, the possibility to become activated is inherent to the molecule and activation cannot be avoided by extrinsic measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Crossreactivity has been shown for certain alcohols and their corresponding aldehydes, i.e. between geraniol and geranial (citral) and between cinnamyl alcohol and cinnamal. The human skin expresses enzyme systems that are able to metabolise xenobiotics, modifying their chemical structure to increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases: phase I and phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask hydrophilic functional groups. If the metabolites are sufficiently polar at this point they will be eliminated. However, many phase I products have to undergo subsequent phase II transformations, i.e. conjugation to make them sufficiently water soluble to be eliminated. Although the purpose of xenobiotic metabolism is detoxification, it can also convert relatively harmless compounds into reactive species. Cutaneous enzymes that catalyse phase I transformations include the cytochrome P450 mixed-function oxidase system, alcohol and aldehyde dehydrogenases, monoamine oxidases, flavin-containing monooxygenases and hydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferases and sulfotransferases are examples of phase II enzymes that have been shown to be present in human skin. These enzymes are known to catalyse both activating and deactivating biotransformations, but the influence of the reactions on the allergenic activity of skin sensitisers has not been studied in detail. Skin sensitising prohapten can be recognised and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or in vivo and in vitro studies of sensitisation potential and chemical reactivity.

QSAR prediction: The relationships between molecular structure and reactivity that form the basis for structural alerts are based on well established principles of mechanistic organic chemistry. Examples of structural alerts are aliphatic aldehydes (alerting to the possibility of sensitisation via a Schiff base reaction with protein amino groups), and alpha,beta-unsaturated carbonyl groups, C=C-CO- (alerting to the possibility of sensitisation via Michael addition of protein thiol groups). Prediction of the sensitisation potential of compounds that can act via abiotic or metabolic activation (pre- or prohapten) is more complex compared to that of compounds that act as direct haptens without any activation. The autoxidation patterns can differ due to differences in the stability of the intermediates formed, e.g. it has been shown that autoxidation of the structural isomers linalool and geraniol results in different major haptens/allergens. Moreover, the complexity of the prediction increases further for those compounds that can act both as pre- and prohapten. In such cases, the impact on the sensitisation potency depends on the degree of abiotic activation (e.g. autoxidation) in relation to the metabolic activation.

d-Limonene is readily absorbed by inhalation and ingestion. Dermal absorption is reported to be lower than by the inhalation route. d-Limonene is rapidly distributed to different tissues in the body, readily metabolised and eliminated primarily through the urine.

Limonene exhibits low acute toxicity by all three routes in animals. Limonene is a skin irritant in both experimental animals and humans.

Limited data are available on the potential to cause eye and respiratory irritation. Autooxidised products of d-limonene have the potential to be skin sensitisers. Limited data are available in humans on the potential to cause respiratory sensitisation. Autooxidation of limonene occurs

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	readily in the presence of light and air forming a variety of oxygenated monocyclic terpenes. Risk of skin sensitisation is high in situations where contact with oxidation products of limonene occurs. Renal tumours induced by limonene in male rats is thought to be sex and species specific and are not considered relevant to humans. Repeated exposure affects the amount and activity of liver enzymes, liver weight, blood cholesterol levels and bile flow in animals. Increase in liver weight is considered a physiological adaption as no toxic effects on the liver have been reported. From available data it is not possible to identify an NOAEL for these effects. Limonene is neither genotoxic or teratogenic nor toxic to the reproductive system.
PELARGONIC ACID & NONYLPHENOL ETHOXYLATES	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.

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

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

SECTION 12 Ecological information

Toxicity

Titan Pelargonic 525 (Titan Pelargonic 525 Herbicide)	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
pelargonic acid	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	8.5mg/l	2
	EC50	48h	Crustacea	57.984-107.814mg/L	4
	EC50(ECx)	240h	Algae or other aquatic plants	0.5mg/l	4
	LC50	96h	Fish	7.4mg/l	Not Available
nonylphenol ethoxylates	Endpoint	Test Duration (hr)	Species	Value	Source
	BCF	1008h	Fish	<0.2	7
	EC50	48h	Crustacea	12.2mg/L	4
	EC50	96h	Algae or other aquatic plants	12mg/l	4
	NOEC(ECx)	2400h	Fish	0.035mg/L	4
	LC50	96h	Fish	1-1.8mg/L	4
d-limonene	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	0.214mg/l	2
	EC50	48h	Crustacea	0.307mg/l	2
	NOEC(ECx)	0h	Algae or other aquatic plants	<0.05-1.5mg/L	4
	LC50	96h	Fish	0.46mg/l	2
Legend:	Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. US EPA, Ecotox database - Aquatic Toxicity Data 4. ECETOC Aquatic Hazard Assessment Data 5. NITE (Japan) - Bioconcentration Data 6. METI (Japan) - Bioconcentration Data 7. Vendor Data				

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.
Toxic to flora.
Toxic to soil organisms.
Ecotoxicity:
The tolerance of water organisms towards pH margin and variation is diverse. Recommended pH values for test species listed in OECD guidelines are between 6.0 and almost 9. Acute testing with fish showed 96h-LC50 at about pH 3.5
For aliphatic fatty acids and alcohols:
Environmental fate:
Saturated fatty acids are very stable in air, whereas unsaturated (C=C bonds) fatty acids are susceptible to oxidation.
Unsaturation increases the rate of metabolism although the degree of unsaturation and positioning of double bonds is not highly significant.
The available data indicate all fatty acid salt chain lengths up to and including C18 can be metabolised under aerobic conditions and can be considered to be readily biodegradable
All tests showed that fatty acids and lipids are readily biodegradable
The aliphatic acids are of similar very weak acid strength (approximately pKa 5), i.e., partially dissociate in aqueous solution; the salts of the aliphatic acids are highly dissociated in water solution such that the anion is the same for homologous salts and acids.
Slight (although inconsistent) effects on the trend for decreasing vapour pressure are also observed with the mono-, di- and tri-unsaturated substances as compared to the corresponding saturated substances.

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Dicarboxylic acids: Compared to their corresponding single acid substances (C8-10 single component, saturated), the dicarboxylic acids exhibit modestly higher melting/ boiling points and water solubility, and lower partition coefficients and vapour pressures. The trends described above for changes in physical chemical properties with increasing carbon chain length apply.

Salts: As expected, the salts differ in physical / chemical properties as compared to their homologous single component substances. However the trends described above for single components with regard to changes in physical chemical properties with increasing carbon chain length apply

Models also indicate that the aliphatic acids will distribute primarily to soil and water, with lesser amounts to air and sediment. With increasing chain length, the percent distributions to soil and sediment generally increase and the percent distributions to water and air generally decrease.

The rate of degradation of fatty acids was investigated in two non-GLP studies.

The total fatty acids residue exhibits low persistence in soil. From the pattern of peaks decline, it was hypothesised a degradation pathway by the sequential elimination of C2 fragments. Consequently, the major soil metabolites of a given fatty acid would be other fatty acids with shorter chains.

Although mineralisation was not measured in these experiments, formation of CO₂ is the expected terminal step of this process. Fatty acids undergo aerobic biodegradation by the process of beta-oxidation. Beta-oxidation of the parent fatty acid forms acetate and a new fatty acid of two less carbon atoms. This process repeats itself until the compound is completely broken down. The hydrocarbon will eventually be degraded to CO₂ and H₂O. For this reason, the length of the fatty acid chain does not preclude biodegradation, but it may take longer to achieve complete mineralisation. The beta-oxidation sequence does not necessarily require the presence of molecular oxygen, and fatty acid biodegradation may proceed under anaerobic conditions.

Hydrolysis is not an important fate path in the environment due to the fact that the substances lack hydrolysable functional groups. Aliphatic acids are hydrolytically stable in aqueous solution.

Water solubility:

In general, the water solubility of single carbon chain length substances followed a pattern of decreasing solubility as carbon chain length increases, especially at C16 and higher. In addition, greater solubility is seen for dicarboxylic acids as compared to their homologous single acids:

In reviewing the physical/ chemical properties of the aliphatic acids, two predominant trends are clearly evident with increasing alkyl chain length and include: i) increasing melting point, boiling point, and partition coefficient, and ii) decreasing water solubility and vapour pressure. Within a given carbon chain length, melting point increases with increasing saturation and decreases with increasing unsaturation. The noted general trends with increasing alkyl chain length are observed when an entire single component group (12 saturated, 4 mono-unsaturated, 2 di-unsaturated, and 1 tri-unsaturated substances) is evaluated together; that is the degree of saturation or unsaturation does not alter the properties trend. The effect of mono-unsaturation (C14:1 to C22:1) appears to be a slight increase in water solubility and a slight decrease in the partition coefficient, as compared to the corresponding saturated substances; a similar trend is noted for the C18 di- or tri-unsaturated substances.

Fatty acids (including methyl esters) were stable to hydrolysis in the pH range of 1-14. It is not expected that photolysis would significantly contribute to the degradation of fatty acids in water.

According to modelling, the aliphatic acids are subject to photodegradation in air. Estimated half-lives generally increase with decreasing chain length and range from 0.6 hours to 17.5 hours.

Methyl (and other) esters are estimated to exhibit high mobility and the acids very high mobility. Mobility may be expected to be higher for the salts than for the corresponding acids and methyl esters

Biodegradation studies or model estimations for single and multi-component aliphatic acids generally confirm that the extent of biodegradation observed in 28 days meets the ready biodegradability criterion (>60%). When the 10-day window was not met or less than 60%, biodegradation was observed in 28 days, it is likely that the aliphatic acids tested were not fully in solution.

Biodegradability tests demonstrated that pelargonic acid (C9), potassium salts and methyl octanoate / methyl decanoate are readily biodegradable. It can be assumed that both acids and methyl esters fatty acids C7-C18 are readily biodegradable.

No experimental bioaccumulation data appear to be available but log Kow data from various sources are higher than 4, which indicates that fatty acids and natural lipids have a potential for bioaccumulating in aquatic organisms.

Fatty alcohols up to chain length C18 are biodegradable, with length up to C16 biodegrading within 10 days completely. Chains C16 to C18 were found to biodegrade from 62% to 76% in 10 days. Chains greater than C18 were found to degrade by 37% in 10 days. Field studies at waste-water treatment plants have shown that 99% of fatty alcohols lengths C12-C18 are removed.

A review of soaps (including calcium and magnesium salts) states that the available data indicate all fatty acid salt chain lengths up to and including C18 can be metabolised under aerobic conditions and can be considered to be biodegradable. Biodegradability did not appear to be influenced by even or odd chain length, degree of saturation or unsaturation or branching. For example odd/even chain length C8 and C9 are readily biodegradable; Saturation/unsaturation: C18(saturated) and C18 (di-unsaturated) are biodegradable, while C18 (mono-unsaturated) are readily biodegradable; branching or hydroxylation: the C18 hydroxylated substance was readily biodegradable and the C18 methyl branched substance was biodegradable.

Higher water solubility of the potassium, sodium and ammonium salts make these a lower ranked analogy for the aquatic toxicity endpoints for the (non-salt) aliphatic acids (and vice versa), while lower water solubility of the magnesium and calcium salts make these a lower ranked analogy for all other members of the category

The aliphatic acids also undergo biodegradation under anaerobic conditions.

Estimated bioconcentration factor values are calculated using EPI Suite v4.10. The aliphatic acids have BCF values less than 100, indicating a low potential for bioaccumulation

Fate prediction using fugacity modeling has shown that fatty alcohols with chain lengths of C10 and greater in water partition into sediment. Lengths C14 and above are predicted to stay in the air upon release. Modeling shows that each type of fatty alcohol will respond independently upon environmental release

Ecotoxicity

Structure-activity relationships based on carbon chain length are evident in the available data on the aquatic ecotoxicity of substances of this category (aquatic toxicity increases with increasing chain length up to a "cutoff" at or near 12 carbons).

The aliphatic acids category members possess properties indicating a hazard for the environment (acute toxicity to fish: between 1-100 mg/L for carbon chain lengths C6 through C12, and multi-component sodium or potassium salts C16-18; acute toxicity to aquatic invertebrates: between 1 and 100 mg/L for carbon chain lengths C6 through C9 (including sodium salts) and less than 1 mg/L for sodium salts single component aliphatic acids C18 and multi component sodium salt aliphatic acids with carbon chain lengths including C14 through C18; and, acute toxicity to aquatic plants: between 1-100 mg/L for carbon chain length C12, including sodium or ammonium salts).

There are a number of acute data for fatty acids and fatty acid salts to aquatic organisms although there is a predominance of data for fatty acid. There are few toxicity values for terrestrial organisms. Data availability / quality covering all the taxonomic groups for specific fatty acid salt chain lengths is poor. The chronic data set is very limited.

For chain lengths >C12, solubility decreases to a degree where an adverse effect would not be expected in the environment due to reduced bioavailability. Data for longer chain lengths have been generated using solvents which makes interpretation more difficult.

The most of few available data indicate low toxicity towards aquatic organisms with EC/LC50 values above 1000 mg/l. However, EC/LC50 values below 100 mg/l are not unusual either

Fish, invertebrates and algae experience similar levels of toxicity with fatty alcohols although it is dependent on chain length with the shorter chain having greater toxicity potential. Longer chain lengths show no toxicity to aquatic organisms.

The available toxicity data indicated low acute and short-term (for birds only) toxicity to birds and mammals. Given that fatty acids are an essential component of the diet of birds and mammals a low risk is expected. On the basis that fatty acids are readily biodegradable and are an essential component of the diet of birds and mammals, a low reproductive risk is expected.

No toxicity data were available for higher aquatic plants and therefore a risk assessment cannot be performed. As pelargonic acid, fatty acid/salt and C8-C10 methyl esters are used as herbicides and plant growth regulators, a data gap to address the risk to higher aquatic plants was identified

A low risk to natural populations of bees and non-target arthropods was concluded for representative greenhouses uses of potassium salts of fatty acids, fatty acid/salt and C8-C10 methyl esters.

Given that fatty acids are readily biodegradable a low risk to sewage treatment organisms was concluded for all of the representative uses.

For Terpenes such as Limonene and Isoprene:

Atmospheric Fate: Contribute to aerosol and photochemical smog formation. When terpenes are introduced to the atmosphere, may either decrease ozone concentrations when oxides of nitrogen are low or, if emissions take place in polluted air (i.e. containing high concentrations of nitrogen oxides), leads to an increase in ozone concentrations. Lower terpenoids can react with unstable reactive gases and may act as precursors of photochemical smog therefore indirectly influencing community and ecosystem properties. The reactions of ozone with larger unsaturated compounds, such as the terpenes can give rise to oxygenated species with low vapour pressures that subsequently condense to form secondary organic aerosol.

Aquatic Fate: Complex chlorinated terpenes such as toxaphene (a persistent, mobile and toxic insecticide) and its degradation products were produced by photoinitiated reactions in an aqueous system, initially containing limonene and other monoterpenes, simulating pulp bleach conditions.

Substances containing unsaturated carbons are ubiquitous in indoor environments. They result from many sources (see below). Most are reactive with environmental ozone and many produce stable products which are thought to adversely affect human health. The potential for surfaces in an enclosed space to facilitate reactions should be considered.

Source of unsaturated substances Unsaturated substances (Reactive Emissions)

Occupants (exhaled breath, ski oils, personal care products) Isoprene, nitric oxide, squalene, unsaturated sterols, oleic acid and other unsaturated fatty acids, unsaturated oxidation products

Soft woods, wood flooring, including cypress, cedar and silver Isoprene, limonene, alpha-pinene, other terpenes and sesquiterpenes

Major Stable Products produced following reaction with ozone.

Methacrolein, methyl vinyl ketone, nitrogen dioxide, acetone, 6MHQ, geranyl acetone, 4OPA, formaldehyde, nonanol, decanal, 9-oxo-nonanoic acid, azelaic acid, nonanoic acid.

Formaldehyde, 4-AMC, pinoaldehyde, pinic acid, pinonic acid, formic acid, methacrolein, methyl vinyl ketone, SOAs including ultrafine particles

Continued...

Titan Pelargonic 525 (Titan Pelargonic 525 Herbicide)

fir boards, houseplants		
Carpets and carpet backing	4-Phenylcyclohexene, 4-vinylcyclohexene, styrene, 2-ethylhexyl acrylate, unsaturated fatty acids and esters	Formaldehyde, acetaldehyde, benzaldehyde, hexanal, nonanal, 2-nonenal
Linoleum and paints/polishes containing linseed oil	Linoleic acid, linolenic acid	Propanal, hexanal, nonanal, 2-heptenal, 2-nonenal, 2-decenal, 1-pentene-3-one, propionic acid, n-butyric acid
Latex paint	Residual monomers	Formaldehyde
Certain cleaning products, polishes, waxes, air fresheners	Limonene, alpha-pinene, terpinolene, alpha-terpineol, linalool, linalyl acetate and other terpenoids, longifolene and other sesquiterpenes	Formaldehyde, acetaldehyde, glycoaldehyde, formic acid, acetic acid, hydrogen and organic peroxides, acetone, benzaldehyde, 4-hydroxy-4-methyl-5-hexen-1-al, 5-ethenyl-dihydro-5-methyl-2(3H)-furanone, 4-AMC, SOAs including ultrafine particles
Natural rubber adhesive	Isoprene, terpenes	Formaldehyde, methacrolein, methyl vinyl ketone
Photocopier toner, printed paper, styrene polymers	Styrene	Formaldehyde, benzaldehyde
Environmental tobacco smoke	Styrene, acrolein, nicotine	Formaldehyde, benzaldehyde, hexanal, glyoxal, N-methylformamide, nicotinaldehyde, cotinine
Soiled clothing, fabrics, bedding	Squalene, unsaturated sterols, oleic acid and other saturated fatty acids	Acetone, geranyl acetone, 6MHO, 40PA, formaldehyde, nonanal, decanal, 9-oxo-nonanoic acid, azelaic acid, nonanoic acid
Soiled particle filters	Unsaturated fatty acids from plant waxes, leaf litter, and other vegetative debris; soot; diesel particles	Formaldehyde, nonanal, and other aldehydes; azelaic acid; nonanoic acid; 9-oxo-nonanoic acid and other oxo-acids; compounds with mixed functional groups (=O, -OH, and -COOH)
Ventilation ducts and duct liners	Unsaturated fatty acids and esters, unsaturated oils, neoprene	C5 to C10 aldehydes
"Urban grime"	Polycyclic aromatic hydrocarbons	Oxidized polycyclic aromatic hydrocarbons
Perfumes, colognes, essential oils (e.g. lavender, eucalyptus, tea tree)	Limonene, alpha-pinene, linalool, linalyl acetate, terpinene-4-ol, gamma-terpinene	Formaldehyde, 4-AMC, acetone, 4-hydroxy-4-methyl-5-hexen-1-al, 5-ethenyl-dihydro-5-methyl-2(3H)-furanone, SOAs including ultrafine particles
Overall home emissions	Limonene, alpha-pinene, styrene	Formaldehyde, 4-AMC, pinonaldehyde, acetone, pinic acid, pinonic acid, formic acid, benzaldehyde, SOAs including ultrafine particles

Abbreviations: 4-AMC, 4-acetyl-1-methylcyclohexene; 6MHQ, 6-methyl-5-heptene-2-one, 4OPA, 4-oxopentanal, SOA, Secondary Organic Aerosols

Reference: Charles J Weschler; Environmental Health Perspectives, Vol 114, October 2006

For Surfactants: Kow cannot be easily determined due to hydrophilic/hydrophobic properties of the molecules in surfactants. BCF value: 1-350.

Aquatic Fate: Surfactants tend to accumulate at the interface of the air with water and are not extracted into one or the other liquid phases.

Terrestrial Fate: Anionic surfactants are not appreciably sorbed by inorganic solids. Cationic surfactants are strongly sorbed by solids, particularly clays. Significant sorption of anionic and non-ionic surfactants has been observed in activated sludge and organic river sediments. Surfactants have been shown to improve water infiltration into soils with moderate to severe hydrophobic or water-repellent properties.

Ecotoxicity: Some surfactants are known to be toxic to animals, ecosystems and humans, and can increase the diffusion of other environmental contaminants. The acute aquatic toxicity generally is considered to be related to the effects of the surfactant properties on the organism and not to direct chemical toxicity. Surfactants should be considered to be toxic to aquatic species under conditions that allow contact of the chemicals with the organisms. Surfactants are expected to transfer slowly from water into the flesh of fish.

During this process, readily biodegradable surfactants are expected to be metabolized rapidly during the process of bioaccumulation. Surfactants are not to be considered to show bioaccumulation potential if they are readily biodegradable.

for alkylphenols and their ethoxylates, or propoxylates:

Environmental fate: Alkylphenols are ubiquitous in the environmental after the introduction, generally as wastes, of their alkoxylated forms (ethoxylates and propoxylates, for example); these are extensively used throughout industry and in the home.

Alkylphenol ethoxylates are widely used surfactants in domestic and industrial products, which are commonly found in wastewater discharges and in sewage treatment plant (STP) effluent's. Degradation of APEs in wastewater treatment plants or in the environment generates more persistent shorter-chain APEs and alkylphenols (APs) such as nonylphenol (NP), octylphenol (OP) and AP mono- to triethoxylates (NPE1, NPE2 and NPE3). There is concern that APE metabolites (NP, OP, NPE1-3) can mimic natural hormones and that the levels present in the environment may be sufficient to disrupt endocrine function in wildlife and humans. The physicochemical properties of the APE metabolites (NP, NPE1-4, OP, OPE1-4), in particular the high Kow values, indicate that they will partition effectively into sediments following discharge from STPs. The aqueous solubility data for the APE metabolites indicate that the concentration in water combined with the high partition coefficients will provide a significant reservoir (load) in various environmental compartments. Data from studies conducted in many regions across the world have shown significant levels in samples of every environmental compartment examined. In the US, levels of NP in air ranged from 0.01 to 81 ng/m³, with seasonal trends observed. Concentrations of APE metabolites in treated wastewater effluents in the US ranged from < 0.1 to 369 ug/l, in Spain they were between 6 and 343 ug/l and concentrations up to 330 ug/l were found in the UK. Levels in sediments reflected the high partition coefficients with concentrations reported ranging from < 0.1 to 13,700 ug/kg for sediments in the US. Fish in the UK were found to contain up to 0.8 ug/kg NP in muscle tissue. APEs degraded faster in the water column than in sediment. Aerobic conditions facilitate easier further biotransformation of APE metabolites than anaerobic conditions. Nonylphenols are susceptible to photochemical degradation. Using natural, filtered, lake water it was found that nonylphenol had a half-life of approximately 10-15 h under continuous, noon, summer sun in the surface water layer, with a rate approximately 1.5 times slower at depths 20-25 cm. Photolysis was much slower with ethoxylated nonylphenol, and so it is unlikely to be a significant event in removal of the ethoxylates.

Air: Alkylphenols released to the atmosphere will exist in the vapour phase and is thought to be degraded by reaction with photochemically produced hydroxyl radicals, with a calculated half-life, for nonylphenol, of 0.3 days.

Water: Abiotic degradation of alkylphenol is negligible. Biodegradation does not readily take place. The half-life in surface water may be around 30 days.

Degradation: Alkylphenol ethoxylates (APES) may abiotically degrade into the equivalent alkylphenol. During degradation ethylene oxide units are cleaved off the ethylene oxide chain until only short-chain alkylphenol ethoxylates remain, typically mono- and diethylene oxides. Oxidation of these oligomers creates the corresponding carboxylic acids. This leaves several degradation products: short-chain ethoxylates, their carboxylic acids, and alkylphenols.

Biodegradation: Alkylphenols are not readily biodegradable. Several mechanisms of microbial aromatic ring degradation have been reported, the most common being formation of catechol from phenol, followed by ring scission between or adjacent to the two hydroxyl groups.

The full breakdown pathway for APES has not yet been determined, and all studies have so far focused on identification of intermediates in bacterial culture media, rather than studying cell-free systems or purified enzymes. It is, however, likely that microbial metabolism usually starts by an attack on the ethoxylate chain, rather than on the ring or the hydrophobic chain. The ethoxylate groups are progressively removed, either by ether cleavage, or by terminal alcohol oxidation followed by cleavage of the resulting carboxylic acid.

Biodegradation of APES produces less biodegradable products: alkylphenol mono- and di-ethoxylates, alkylphenoxy acetic and alkylphenoxy polyethoxy acetic acids, and alkylphenols. These metabolites frequently persist through sewage treatment and in rivers. Anaerobic conditions generally lead to the accumulation of alkylphenols. The rate of biodegradation seems to decrease with increasing length of the ethylene oxide chain.

Bioaccumulation: Metabolites of APES accumulate in organisms, with bioconcentration factors varying from ten to several thousand, depending on species, metabolite and organ.

The metabolites of APES are generally more toxic than the original compounds. APES have LC50s above about 1.5 mg/l, whereas alkylphenols, such as nonylphenol, have LC50s are generally around 0.1 mg/l.

Oestrogenic activity: The role of alkyl chain length and branching, substituent position, number of alkylated groups, and the requirement of a phenolic ring structure was assessed in fish. The results showed that most alkylphenols were oestrogenic, although with 3-300 thousand times lower potency than the endogenous estrogen 17beta-estradiol. Mono-substituted tertiary alkylphenols with moderate (C4-C5) and long alkyl chain length (C8-C9) in the para position exhibited the highest oestrogenic potency. Substitution with multiple alkyl groups, presence of substituents in the ortho- and meta-position and lack of a hydroxyl group on the benzene ring reduced the oestrogenic activity, although several oestrogenic alkylated non-phenolics were identified.

Human exposure: Alkylphenols were first found to be oestrogenic (oestrogen-mimicking) in the 1930s, but more recent research has highlighted the implications of these effects. The growth of cultured human breast cancer cells is affected by nonylphenol at concentrations as low as 1 uM (220 ug/l) or concentrations of octylphenol as low as 0.1 uM (20 ug/l). Oestrogenic effects have also been shown on rainbow trout hepatocytes, chicken embryo fibroblasts and a mouse oestrogen receptor.

The insecticide chlordecone (Kepone) shows similar behaviour to alkylphenols, accumulating in liver and adipose tissue, and eliciting oestrogenic activity. Workers exposed to this insecticide can suffer reproductive effects such as low sperm counts and sterility. In addition, the oestrogenic effects of chlordecone on MCF7 cells occur at similar concentrations to those of alkylphenols, suggesting that alkylphenols will be a similar health hazard if target cells are exposed to uM levels of these compounds.

By comparing environmental concentrations, bioconcentration factors and *in vitro* oestrogenic effect levels, current environmental levels of alkylphenolic compounds are probably high enough to affect the hormonal control systems of some organisms. It is also possible that human health could be being affected.

For Limonenes:

Atmospheric Fate: Due to the high volatility of limonene, the atmosphere is expected to be the major environmental sink for this chemical. The oxidation of limonene may contribute to aerosol and photochemical smog formation. The daytime atmospheric lifetime of d-limonene is estimated to range from 12 to 48 minutes depending upon local hydroxyl rate and ozone concentrations. Ozonolysis of limonene may also lead to the formation of hydrogen peroxide and organic peroxides, which have various toxic effects on plant cells and may damage forests. Reactions with nitrogen oxides produce aerosol formation as well as lower molecular weight products such as formaldehyde, acetaldehyde, formic acid, acetone and peroxyacetyl nitrate.

Terrestrial fate: When released to the ground, limonene is expected to have low to very low mobility in soil based on its physicochemical properties. It is expected that limonene will rapidly volatilize from both dry and moist soil, however; its absorption to soil may slow the process.

Aquatic fate: In the aquatic environment, limonene is expected to evaporate to a significant extent owing to its high volatility. The estimated half-life for volatilisation of limonene from a model river 1 m deep is 3.4 h. Some limonene is expected to absorb to sediment and suspended organic matter. Hydrolysis of limonene is not expected in terrestrial or in aquatic environments. The hydrolytic half-life of d-limonene is estimated to be >1000 days.

Ecotoxicity: Biotic degradation of limonene has been shown with some species of microorganisms such as *Penicillium digitatum*, *Corynespora cassicola*, *Diplodia gossypina* and a soil strain of *Pseudomonas* sp (SL strain). Limonene is readily biodegradable under aerobic conditions. Biodegradation has been assessed under anaerobic conditions; there was no indication of any metabolisms, possibly because of the toxicity to micro-organisms. Limonene may bioaccumulate in fish and other aquatic species. Technical limonene is practically nontoxic to birds and is slightly toxic to freshwater fish and invertebrates on an acute basis. Limonene has low subacute toxicity to bobwhite quail.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
pelargonic acid	LOW	LOW
d-limonene	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
pelargonic acid	LOW (LogKOW = 3.42)
nonylphenol ethoxylates	LOW (BCF = 1.4)
d-limonene	HIGH (LogKOW = 4.8275)

Mobility in soil

Ingredient	Mobility
pelargonic acid	LOW (Log KOC = 47.26)
d-limonene	LOW (Log KOC = 1324)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	- Containers may still present a chemical hazard/ danger when empty. - Return to supplier for reuse/ recycling if possible. Otherwise: - If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. - Where possible retain label warnings and SDS and observe all notices pertaining to the product. 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) 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. - Where in doubt contact the responsible authority. - Recycle wherever possible or consult manufacturer for recycling options. - Consult State Land Waste Authority for disposal. - Bury or incinerate residue at an approved site. - Recycle containers if possible, or dispose of in an authorised landfill.
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SECTION 14 Transport information

Labels Required

	
Marine Pollutant	
HAZCHEM	2X

Land transport (ADG)

14.1. UN number or ID number	1760
14.2. UN proper shipping name	CORROSIVE LIQUID, N.O.S. (contains pelargonic acid)
	Class8

14.3. Transport hazard class(es)	Subsidiary Hazard	Not Applicable
14.4. Packing group	III	
14.5. Environmental hazard	Environmentally hazardous	
14.6. Special precautions for user	Special provisions	223 274
	Limited quantity	5 L

Air transport (ICAO-IATA / DGR)

14.1. UN number	1760	
14.2. UN proper shipping name	Corrosive liquid, n.o.s. * (contains pelargonic acid)	
14.3. Transport hazard class(es)	ICAO/IATA Class	8
	ICAO / IATA Subsidiary Hazard	Not Applicable
	ERG Code	8L
14.4. Packing group	III	
14.5. Environmental hazard	Environmentally hazardous	
14.6. Special precautions for user	Special provisions	A3 A803
	Cargo Only Packing Instructions	856
	Cargo Only Maximum Qty / Pack	60 L
	Passenger and Cargo Packing Instructions	852
	Passenger and Cargo Maximum Qty / Pack	5 L
	Passenger and Cargo Limited Quantity Packing Instructions	Y841
	Passenger and Cargo Limited Maximum Qty / Pack	1 L

Sea transport (IMDG-Code / GGVSee)

14.1. UN number	1760	
14.2. UN proper shipping name	CORROSIVE LIQUID, N.O.S. (contains pelargonic acid)	
14.3. Transport hazard class(es)	IMDG Class	8
	IMDG Subsidiary Hazard	Not Applicable
14.4. Packing group	III	
14.5. Environmental hazard	Marine Pollutant	
14.6. Special precautions for user	EMS Number	F-A, S-B
	Special provisions	223 274
	Limited Quantities	5 L

14.7. Maritime transport in bulk according to IMO instruments

14.7.1. Transport in bulk according to Annex II of MARPOL and the IBC code
Not Applicable

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

Product name	Group
pelargonic acid	Not Applicable
nonylphenol ethoxylates	Not Applicable
d-limonene	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
pelargonic acid	Not Applicable
nonylphenol ethoxylates	Not Applicable
d-limonene	Not Applicable

SECTION 15 Regulatory information

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

- pelargonic acid 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
- Australian Inventory of Industrial Chemicals (AIIC)
- nonylphenol ethoxylates 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

d-limonene is found on the following regulatory lists
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

Additional Regulatory Information
Not Applicable

National Inventory Status	
National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (pelargonic acid; nonylphenol ethoxylates; d-limonene)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory 'Active'
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	Yes
UAE - Control List (Banned/Restricted Substances)	No (pelargonic acid; nonylphenol ethoxylates; d-limonene)
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	05/05/2026
Initial Date	24/09/2025

SDS Version Summary		
Version	Date of Update	Sections Updated
2.1	25/09/2025	Hazards identification - Classification
3.1	05/05/2026	Physical and chemical properties - Appearance

Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.

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

- Definitions and abbreviations**
- ▶ PC - TWA: Permissible Concentration-Time Weighted Average
 - ▶ PC - STEL: Permissible Concentration-Short Term Exposure Limit
 - ▶ IARC: International Agency for Research on Cancer
 - ▶ ACGIH: American Conference of Governmental Industrial Hygienists
 - ▶ STEL: Short Term Exposure Limit
 - ▶ TEEL: Temporary Emergency Exposure Limit,
 - ▶ IDLH: Immediately Dangerous to Life or Health Concentrations
 - ▶ ES: Exposure Standard
 - ▶ OSF: Odour Safety Factor
 - ▶ NOAEL: No Observed Adverse Effect Level
 - ▶ LOAEL: Lowest Observed Adverse Effect Level
 - ▶ TLV: Threshold Limit Value
 - ▶ LOD: Limit Of Detection
 - ▶ OTV: Odour Threshold Value
 - ▶ BCF: BioConcentration Factors
 - ▶ BEI: Biological Exposure Index
 - ▶ DNEL: Derived No-Effect Level
 - ▶ PNEC: Predicted no-effect concentration
 - ▶ MARPOL: International Convention for the Prevention of Pollution from Ships
 - ▶ IMSBC: International Maritime Solid Bulk Cargoes Code
 - ▶ IGC: International Gas Carrier Code
 - ▶ IBC: International Bulk Chemical Code

Titan Pelargonic 525 (Titan Pelargonic 525 Herbicide)

- ▶ AIIC: Australian Inventory of Industrial Chemicals
- ▶ DSL: Domestic Substances List
- ▶ NDSL: Non-Domestic Substances List
- ▶ IECSC: Inventory of Existing Chemical Substance in China
- ▶ EINECS: European INventory of Existing Commercial chemical Substances
- ▶ ELINCS: European List of Notified Chemical Substances
- ▶ NLP: No-Longer Polymers
- ▶ ENCS: Existing and New Chemical Substances Inventory
- ▶ KECI: Korea Existing Chemicals Inventory
- ▶ NZIoC: New Zealand Inventory of Chemicals
- ▶ PICCS: Philippine Inventory of Chemicals and Chemical Substances
- ▶ TSCA: Toxic Substances Control Act
- ▶ TCSI: Taiwan Chemical Substance Inventory
- ▶ INSQ: Inventario Nacional de Sustancias Químicas
- ▶ NCI: National Chemical Inventory
- ▶ FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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