



Castor Crete "A" Clear

ICP Construction Inc.

Version No: 5.5
Safety Data Sheet according to OSHA HazCom Standard (2012) requirements

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L.GHS.USA.EN

SECTION 1 Identification

Product Identifier

Product name	Castor Crete "A" Clear
Synonyms	Not Available
Proper shipping name	Environmentally hazardous substance, liquid, n.o.s.
Other means of identification	Not Available

Recommended use of the chemical and restrictions on use

Relevant identified uses	Special Floor Coating Resin
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Name, address, and telephone number of the chemical manufacturer, importer, or other responsible party

Registered company name	ICP Construction Inc.
Address	150 Dascomb Road Andover, MA 01810 United States
Telephone	1-866-667-5119 1-978-623-9987
Fax	Not Available
Website	www.icpgroup.com
Email	sds@icpgroup.com

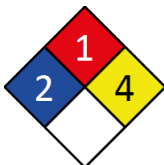
Emergency phone number

Association / Organisation	ChemTel
Emergency telephone numbers	1-800-255-3924
Other emergency telephone numbers	1-813-248-0585

SECTION 2 Hazard(s) identification

Classification of the substance or mixture

NFPA 704 diamond



Note: The hazard category numbers found in GHS classification in section 2 of this SDSs are NOT to be used to fill in the NFPA 704 diamond. Blue = Health Red = Fire Yellow = Reactivity White = Special (Oxidizer or water reactive substances)

Classification	Serious Eye Damage/Eye Irritation Category 2A, Hazardous to the Aquatic Environment Long-Term Hazard Category 2, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Skin Corrosion/Irritation Category 2, Reproductive Toxicity Category 1B, Sensitisation (Skin) Category 1
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Label elements

Hazard pictogram(s)	
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Signal word	Danger
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Hazard statement(s)

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H319	Causes serious eye irritation.
H411	Toxic to aquatic life with long lasting effects.
H335	May cause respiratory irritation.
H315	Causes skin irritation.
H360	May damage fertility or the unborn child.
H317	May cause an allergic skin reaction.

Hazard(s) not otherwise classified

Not Applicable

Precautionary statement(s) Prevention

P202	Do not handle until all safety precautions have been read and understood.
P261	Avoid breathing dust/fumes/gas/mist/vapors/spray
P264	Wash thoroughly after handling.
P271	Use only outdoors or in a well-ventilated area.
P272	Contaminated work clothing should not be allowed out of the workplace
P273	Avoid release to the environment
P280	Wear protective gloves/protective clothing/eye protection/face protection.

Precautionary statement(s) Response

P302+P352	IF ON SKIN: Wash with plenty of water
P333+P313	IF Skin irritation or rash occurs: Get medical advice/attention.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses if present and easy to do. Continue rinsing.
P337+P313	IF Eye irritation persists: Get medical advice/attention.
P308+P313	IF exposed or concerned: Get medical advice/attention.
P312	Call a POISON CENTER or doctor if you feel unwell
P362	Take off contaminated clothing and wash before reuse.
P391	Collect spillage

Precautionary statement(s) Storage

P405	Store locked up.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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Not Applicable

SECTION 3 Composition / information on ingredients**Substances**

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
8001-79-4	30-60	<u>castor oil</u>
61791-12-6	1-5	<u>castor oil, hydrogenated, ethoxylated</u>
64-19-7	0.1-1	<u>acetic acid glacial</u>
102-60-3	0.1-1	<u>tetrahydroxypropyl ethylenediamine</u>
85-68-7	10-30	<u>butyl benzyl phthalate</u>
94266-48-5	5-10	<u>pine oil, synthetic</u>

The specific chemical identity and/or exact percentage (concentration) of composition has been withheld as a trade secret.

SECTION 4 First-aid measures**Description of first aid measures**

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Wash out immediately with fresh running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
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Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation. <p>For thermal burns:</p> <ul style="list-style-type: none"> ▶ Decontaminate area around burn. ▶ Consider the use of cold packs and topical antibiotics. <p>For first-degree burns (affecting top layer of skin)</p> <ul style="list-style-type: none"> ▶ Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides. ▶ Use compresses if running water is not available. ▶ Cover with sterile non-adhesive bandage or clean cloth. ▶ Do NOT apply butter or ointments; this may cause infection. ▶ Give over-the counter pain relievers if pain increases or swelling, redness, fever occur. <p>For second-degree burns (affecting top two layers of skin)</p> <ul style="list-style-type: none"> ▶ Cool the burn by immerse in cold running water for 10-15 minutes. ▶ Use compresses if running water is not available. ▶ Do NOT apply ice as this may lower body temperature and cause further damage. ▶ Do NOT break blisters or apply butter or ointments; this may cause infection. ▶ Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape. <p>To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort):</p> <ul style="list-style-type: none"> ▶ Lay the person flat. ▶ Elevate feet about 12 inches. ▶ Elevate burn area above heart level, if possible. ▶ Cover the person with coat or blanket. ▶ Seek medical assistance. <p>For third-degree burns Seek immediate medical or emergency assistance.</p> <p>In the mean time:</p> <ul style="list-style-type: none"> ▶ Protect burn area cover loosely with sterile, nonstick bandage or, for large areas, a sheet or other material that will not leave lint in wound. ▶ Separate burned toes and fingers with dry, sterile dressings. ▶ Do not soak burn in water or apply ointments or butter; this may cause infection. ▶ To prevent shock see above. ▶ For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway. ▶ Have a person with a facial burn sit up. ▶ Check pulse and breathing to monitor for shock until emergency help arrives.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor, without delay.
Ingestion	<ul style="list-style-type: none"> ▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▶ Seek medical advice.

Most important symptoms and effects, both acute and delayed

See Section 11

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 Fire-fighting 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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Special protective equipment and precautions for fire-fighters

Fire Fighting	<ul style="list-style-type: none"> ▶ 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.
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Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ Combustible. ▶ Slight fire hazard when exposed to heat or flame. ▶ Heating may cause expansion or decomposition leading to violent rupture of containers. ▶ On combustion, may emit toxic fumes of carbon monoxide (CO). ▶ May emit acrid smoke. ▶ Mists containing combustible materials may be explosive. <p>Combustion products include: carbon dioxide (CO₂) acrolein nitrogen oxides (NO_x) other pyrolysis products typical of burning organic material.</p> <p>CARE: Water in contact with hot liquid may cause foaming and a steam explosion with wide scattering of hot oil and possible severe burns. Foaming may cause overflow of containers and may result in possible fire.</p>
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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	<p>Environmental hazard - contain spillage. Slippery when spilt.</p> <ul style="list-style-type: none"> ▶ 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	<p>Environmental hazard - contain spillage. Slippery when spilt. Moderate hazard.</p> <ul style="list-style-type: none"> ▶ 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.

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

SECTION 7 Handling and storage**Precautions for safe handling**

Safe handling	<ul style="list-style-type: none"> ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ Avoid smoking, naked lights or ignition sources. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ 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. ▶ DO NOT allow clothing wet with material to stay in contact with skin
Other information	<p>Consider storage under inert gas. Refrigerated storage normally required.</p> <ul style="list-style-type: none"> ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

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Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ DO NOT use aluminium or galvanised containers ▶ Metal can or drum ▶ Packaging as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<p>d-Limonene:</p> <ul style="list-style-type: none"> ▶ 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 <p>Phthalates:</p> <ul style="list-style-type: none"> ▶ react with strong acids, strong oxidisers, permanganates and nitrates ▶ attack some form of plastics <p>Acetic acid:</p> <ul style="list-style-type: none"> ▶ vapours forms explosive mixtures with air (above 39 C.) ▶ reacts violently with bases such as carbonates and hydroxides (giving off large quantities of heat), oxidisers, organic amines, acetaldehyde, potassium tert-butoxide ▶ reacts (sometimes violently), with strong acids, aliphatic amines, alkanolamines, alkylene oxides, epichlorohydrin, acetic anhydride, 2-aminoethanol, ammonia, ammonium nitrate, bromine pentafluoride, chlorosulfonic acid, chromic acid, chromium trioxide, ethylenediamine, ethyleneimine, hydrogen peroxide, isocyanates, oleum, perchloric acid, permanganates, phosphorus isocyanate, phosphorus trichloride, sodium peroxide, xylene ▶ attacks cast iron, stainless steel and other metals, forming flammable hydrogen gas ▶ attacks many forms of rubber, plastics and coatings <p>Terpenoids and terpenes, are generally unsaturated, are thermolabile, are often volatile and may be easily oxidised or hydrolysed depending on their respective structure.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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</p> <p>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.</p> <ul style="list-style-type: none"> - 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. <p>BREITHERICK: Handbook of Reactive Chemical Hazards, 4th Edition</p> <ul style="list-style-type: none"> - 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 <ul style="list-style-type: none"> ▶ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US OSHA Permissible Exposure Limits (PELs) Table Z-1	acetic acid glacial	Acetic acid	10 ppm / 25 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	acetic acid glacial	Acetic acid	10 ppm / 25 mg/m3	37 mg/m3 / 15 ppm	Not Available	Not Available
US ACGIH Threshold Limit Values (TLV)	acetic acid glacial	Acetic acid	10 ppm	15 ppm	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
acetic acid glacial	Not Available	Not Available	Not Available
tetrahydroxypropyl ethylenediamine	34 mg/m3	370 mg/m3	2,200 mg/m3
butyl benzyl phthalate	15 mg/m3	77 mg/m3	460 mg/m3

Ingredient	Original IDLH	Revised IDLH
castor oil	Not Available	Not Available
castor oil, hydrogenated, ethoxylated	Not Available	Not Available
acetic acid glacial	50 ppm	Not Available
tetrahydroxypropyl ethylenediamine	Not Available	Not Available
butyl benzyl phthalate	Not Available	Not Available
pine oil, synthetic	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
castor oil	E	≤ 0.1 ppm
castor oil, hydrogenated, ethoxylated	E	≤ 0.1 ppm
tetrahydroxypropyl ethylenediamine	E	≤ 0.1 ppm
butyl benzyl phthalate	C	> 1 to ≤ 10 parts per million (ppm)
pine oil, synthetic	E	≤ 0.1 ppm

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

MATERIAL DATA

Fragrance substance with is an established contact allergen in humans.
Scientific Committee on Consumer Safety SCCS OPINION on Fragrance allergens in cosmetic products 2012

Exposed individuals are **NOT** reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odour Safety Factor (OSF) is determined to fall into either Class C, D or E.

The Odour Safety Factor (OSF) is defined as:

OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm

Classification into classes follows:

Class OSF Description

- A 550 Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by working activities
- B 26-550 As "A" for 50-90% of persons being distracted
- C 1-26 As "A" for less than 50% of persons being distracted
- D 0.18-1 10-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached
- E <0.18 As "D" for less than 10% of persons aware of being tested

for d-Limonene:

CEL TWA: 30 ppm, 165.6 mg/m3 (compare WEEL-TWA*)

(CEL = Chemwatch Exposure Limit)

A Workplace Environmental Exposure Level* has been established by AIHA (American Industrial Hygiene Association) who have produced the following rationale:

d-Limonene is not acutely toxic. In its pure form it is not a sensitiser but is irritating to the skin. Although there is clear evidence of carcinogenicity in male rats, the effect has been attributed to an alpha-2u-globin (a2u-G) renal toxicity which is both species and gender specific. Humans do not synthesise a2u-G, and metabolism studies indicate that 75% to 95% of d-limonene is excreted in 2-3 days with different metabolites identified between humans and rats. In a 2-year study, liver effects were noted in male mice at 500 mg/kg and reduced survival was noted in female rats at 600 mg/kg. The no observable effect levels (NOELs) were 250 and 300 mg/kg, respectively. A WEEL of 30 ppm is recommended to protect against these effects.

for acetic acid:

NOTE: Detector tubes for acetic acid, measuring in excess of 1 ppm, are commercially available.

Exposure at or below the TLV-TWA and TLV-STEL is thought to protect the worker against conjunctival, nose and respiratory tract irritation.

Odour Safety Factor (OSF)

OSF=21 ("ACETIC ACID, GLACIAL")

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
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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.

Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. An approved self contained breathing apparatus (SCBA) may be required in some situations.
 Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	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.)

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity
3: Intermittent, low production.	3: High production, heavy use
4: Large hood or large air mass in motion	4: Small hood-local control only

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

Personal protection



Eye and face protection

- ▶ Safety glasses with side shields.
- ▶ Chemical goggles.
- ▶ 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

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

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	<ul style="list-style-type: none"> · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>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.</p> <p>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.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> · 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 <p>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.</p>
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ 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:
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Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NITRILE	C
NITRILE+PVC	C
PE	C
PE/EVAL/PE	C
PVC	C
SARANEX-23	C
TEFLON	C

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

Respiratory protection

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

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

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

^ - Full-face

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

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

SECTION 9 Physical and chemical properties**Information on basic physical and chemical properties**

Appearance	<p>Air sensitive. Heat sensitive.</p> <p>Glycerides, more correctly known as acylglycerols, are esters formed from glycerol and fatty acids.</p> <p>Glycerol has three hydroxyl functional groups, which can be esterified with one, two, or three fatty acids to form monoglycerides (MAGs), diglycerides (DAGs), and triglycerides (TAGs).</p> <p>Vegetable oils and animal fats contain mostly triglycerides, but are broken down by natural enzymes (lipases) into mono and diglycerides and free fatty acids and glycerol.</p> <p>Partial glycerides are esters of glycerol with fatty acids, where not all the hydroxyl groups are esterified. Since some of their hydroxyl groups are free their molecules are polar. Partial glycerides may be monoglycerides (two hydroxyl groups free) or diglycerides (one hydroxyl group free).</p> <p>Short chain partial glycerides are more strongly polar than long chain partial glycerides, and have excellent solvent properties for many hard-to-solubilise drugs, making them valuable as excipients in improving the formulation of certain pharmaceuticals. The most common forms of acylglycerol are triglycerides, having high caloric value and usually yielding twice as much energy per gram as carbohydrate</p> <p>Triglycerides are hydrophobic materials that range from oils, at the lowest molecular weights/shortest chain-lengths, to waxy solids, at the highest molecular weights/longest chain-lengths. Some triglycerides are produced synthetically via classical Fischer type esterification methods (i.e., reaction of carboxylic acids with a glycerin to produce carboxylic esters), although the reaction may be promoted by acid or base catalysis, or by the use of an acid chloride. However, some of these ingredients may be natural sourced and produced by transesterification (i.e., exchange of</p>
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	acid moieties to create a different ester product). For example, the triglycerides in natural oils can be reacted with intended length fatty acids to produce new triglycerides. Trisubstituted glycerols (TAGs; glycerolipids) represent the most abundant lipid class in oils and fats of animal origin, and comprise the bulk of storage fat in mammalian tissue. These molecules exist as enantiomers, since a center of asymmetry is created upon enzymatic biosynthesis at carbon 2 of the glycerol backbone. During the biosynthesis and digestion of TAGs, diacylglycerols (diglycerides, DAGs) and monoacylglycerols (monoglycerides, MAGs) are formed as intermediates, with two and one fatty acid substitution at the glycerol backbone, respectively		
Physical state	Liquid	Relative density (Water = 1)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Available
Flash point (°C)	Not Available	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Available	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (Not Available%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ▶ 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

Inhaled	<p>Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>Inhalation hazard is increased at higher temperatures.</p> <p>Not normally a hazard due to non-volatile nature of product</p> <p>Fine mists generated from plant/ vegetable (or more rarely from animal) oils may be hazardous. Extreme heating for prolonged periods, at high temperatures, may generate breakdown products which include acrolein and acrolein-like substances.</p>
Ingestion	<p>Accidental ingestion of the material may be damaging to the health of the individual.</p> <p>Fatty acid esters are relatively non-toxic in rats. Large doses of 20-60 gm/kg are lethal in rats.</p> <p>Castor oil is considered minimally toxic when administered orally to humans; the estimated lethal oral dose is 1-2 pints of undiluted oil (Gosselin et al., 1976). As a purgative, castor oil is ingested as a bolus. Since this would lead to higher concentrations of ricinoleic acid in the gastrointestinal tract than would occur with dietary exposure, it is not surprising that in an occupational setting there is no indications of loose or wet faeces.</p> <p>Constant use of purgatives/laxatives may decrease the sensitivity of the intestinal mucosa causing a diminished response to normal stimuli. The redevelopment of a normal habit is thus prevented.</p>

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	<p>In a 14-day study of butyl benzyl phthalate in rats, exposure to 25000 ppm or more resulted in lower body weight gains. Thymic atrophy occurred in all 100000 ppm rats and testicular degeneration was observed in all 50000 and 100000 ppm males. No compound related effects were seen in a companion study in mice receiving 25000 ppm in feed. In a similar 13-week study, lower body weight gains and testicular degeneration, characterised by loss of germinal epithelium of the seminiferous tubules were seen in male rats receiving 25000 ppm whereas compound related effects in mice were limited to lower body weight gains in male mice exposed to concentrations of 1600 ppm or more and in 12500 ppm females. The testicular degeneration in rats may be related to the conversion of the phthalate to monobutylphthalate which has been shown to produce testicular atrophy.</p> <p>Rats exposed to 2000 to 4000 mg in feed for 2-weeks showed dose-related posterior body stiffness and incoordination of the hind limbs which was more severe in males. These signs generally disappeared by the end of a 1-week recovery period. In a follow-up 6-week neurological study, body weight gains of rats exposed to 1500 or 3000 mg butyl benzyl phthalate/kg body weight were lower than those of the controls, and transient hind limb stiffness was observed in the 3000 mg/kg group, mainly in males.</p> <p>Histopathology of tissues from the central and peripheral nervous system no-observed-effect-level (NOEL) in a 90 day study with rats administered feed containing up to 2% of the phthalate, by weight, was 0.5%. After 14-weeks of a 2-year study with rats exposed to 6000 or 12000 ppm butyl benzyl phthalate in feed, compound related mortality in males resulted from unexplained internal haemorrhaging.</p> <p>It has been postulated that intraperitoneal injection produces acute depression of the central nervous system.</p> <p>Ricinoleic acid, the major fatty acid present in castor oil, has a variety of effects on the gastrointestinal tract, including inhibition of water and electrolyte absorption (Donowitz, 1979), stimulation of water secretion into the intestinal lumen (Ammon and Phillips, 1974), and depression of small bowel contractile activity (Ammon et al., 1974). The cathartic action of orally ingested castor oil traditionally has been attributed to irritant or stimulatory effects of ricinoleic acid on the gastrointestinal smooth muscle; the ricinoleic acid is liberated in the small intestine by the action of pancreatic lipase (Stewart and Bass, 1976). Moreover, absorption of ricinoleic acid occurs incompletely; substantial quantities remain in the gastrointestinal tract after oral administration (Stewart and Bass, 1976). Since diet palatability was not affected by the presence of castor oil, the poor absorption of ricinoleic acid and its potential to reduce absorption of other fatty acids could be responsible for the absence of more substantial body weight gains by rats and mice consuming castor oil-containing diets.</p>
<p>Skin Contact</p>	<p>The material may accentuate any pre-existing dermatitis condition</p> <p>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.</p> <p>Daily application of 0.5 ml of castor oil to the skin of adult female albino rabbits produced mild irritant reactions, including slight erythema and edema, acanthosis and disorganization of the basal layer, and slight inflammation of the dermis (Rantuccio et al., 1981)</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p> <p>The material produces severe skin irritation; evidence exists, or practical experience predicts, that the material either:</p> <ul style="list-style-type: none"> ▶ produces severe inflammation of the skin in a substantial number of individuals following direct contact, and/or ▶ produces significant and severe inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. ▶ Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. <p>NOTE: Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.</p>
<p>Eye</p>	<p>Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals. Repeated or prolonged eye contact may cause inflammation (similar to windburn) characterised by a temporary redness of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p>
<p>Chronic</p>	<p>Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. 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.</p> <p>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.</p> <p>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</p> <p>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.</p> <p>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.</p> <p>There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of:</p> <ul style="list-style-type: none"> - clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects. <p>The various phthalates have different uses, chemical structures and toxicity profiles. It is therefore difficult to generalise about the safety of all phthalates as a group. The main health concern associated with some phthalates is that animal studies have shown that high regular doses can affect the reproductive system in developing young, particularly males. While there is no significant risk to the general population, young children may experience higher exposures than the general population if they chew or suck on phthalate-containing toys, or if they ingest phthalates over a long period from other products containing high levels of phthalates.</p> <p>In animal tests, phthalates have been shown to "feminise" male animals, increasing the likelihood of small or undeveloped testes, undescended testicles, and low sperm counts. A 2005 study also linked higher foetal exposure to phthalates through the mother's blood with increased risk of developmental abnormalities in male infants. Higher phthalate levels are also associated with lower testosterone production and reduced sperm count in men.</p> <p>One study suggested that high levels of phthalates may be connected to the current obesity epidemic in children. It was found that obese children show greater exposure to phthalates than non-obese children. It was reported that the obesity risk increases according to the level of the chemical found in the children's bloodstream. In a national cross-section of U.S. men, concentrations of several prevalent phthalate metabolites showed statistically significant correlations with abnormal obesity and insulin resistance. A further study found that people with elevated phthalate levels had roughly twice the risk of developing diabetes compared with those with lower levels. This study also found that phthalates were associated with disrupted insulin production.</p> <p>Much of the current research on effects of phthalate exposure has been focused towards children and men's health, however, women may be at</p>

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higher risk for potential adverse health effects of phthalates due to increased cosmetic use. According to in vivo and observational studies there is an association between phthalate exposure and endocrine disruption leading to development of breast cancer. This finding may be associated with the demethylation of the oestrogen receptor complex in breast cancer cells.

A Russian study describes exposure by workers to mixed phthalates (and other plasticisers) - pain, numbness and spasms in the upper and lower extremities were related to duration of exposures. Symptoms usually developed after the sixth or seventh year of work. Neurological studies revealed the development of polyneuritis in about 30% of the workers involved in this study. About 30% of the workforce showed depression of the vestibular receptors. Because the study described mixed exposures it is difficult to determine what, if any, unique role was played by the phthalates. Increased incidences of anovulatory reproductive cycles and low oestrogen concentrations were reported among Russian women working with phthalate plasticisers; the abnormal cycles were associated with spontaneous abortion. The specific phthalates implicated, dose levels and other data were not reported. It has been alleged that the phthalates mimic or interfere with sex packaging) and are used as ingredients in paints, inks and adhesives. Their potential for entering the human body is marked. They have been added to a list of chemicals (including alkyl phenolics, polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs) and dioxins) which are implicated in reducing sperm counts and fertility in males a phenomenon which has apparently arisen since the mid 1960s.

Phthalates are generally considered to be in a class of endocrine disruptors known as "xenoestrogens," for their ability to mimic the effect of oestrogen on the body.

Although the human foetus is "bathed" in naturally occurring oestrogens during pregnancy it is suggested that it has developed a protective mechanism against natural oestrogens but is not safe from synthetic variants. These tend to accumulate in body fats which sets them apart from the natural product. During early pregnancy, fats are broken down and may flood the body with concentrated pollutants

Human phthalate exposure during pregnancy results in decreased anogenital distance among baby boys. Boys born to mothers with the highest levels of phthalates were 7 times more likely to have a shortened anogenital distance.

While anogenital distance is routinely used as a measure of foetal exposure to endocrine disruptors in animals, this parameter is rarely assessed in humans, and its significance is unknown

One study also found that female animals exposed to higher levels of phthalates experienced increased risk of miscarriage, a common symptom of excessive estrogen levels in human women, and stillbirth. Prematurity may also be linked to phthalate exposure.

Another study found a link between exposure to phthalates and increased rates of childhood obesity.

In adult human men, phthalates have been linked to greater waist circumference and higher insulin resistance, a common precursor to type 2 (adult onset) diabetes. They have been linked to thyroid irregularities, asthma, and skin allergies in both sexes. Though the exact mechanism is unclear, studies have linked higher rates of respiratory infections and other symptoms in children living in houses with vinyl floors. One possible explanation is inhalation of dust tainted by phthalates, which are used in cosmetics such as nail polishes and hand creams precisely because of their ability to bind to human tissues.

Animal studies have shown increased risks of certain birth defects (including the genital abnormalities and, in rats, extra ribs) and low birth rates in rats whose mothers were fed higher levels of phthalates.

These effects on foetal development are of particular concern because young women of childbearing age often have higher than average phthalate levels in the body thanks to their use of cosmetics, many of which contain phthalates.

The EU has applied limitations to the use of several phthalates in general food contact applications (packaging and closures) and medical device applications. The USA has introduced regulation of phthalate esters as components of children's toys and childcare articles for children under the age of 12 that could be 'placed in the mouth'.

Endocrine disruptors such as phthalates can add to the effects of other endocrine disruptors, so even very small amounts can interact with other chemicals to have cumulative, adverse "cocktail effects"

Large amounts of specific phthalates fed to rodents have been shown to damage their liver and testes, and initial rodent studies also indicated hepatocarcinogenicity. Later studies on primates showed that the mechanism is specific to rodents - humans are resistant to the effect. Studies conducted on mice exposed to phthalates in utero did not result in metabolic disorder in adults. However, "At least one phthalate, monoethylhexyl phthalate (MEHP) has been found to interact with all three peroxisome proliferator-activated receptors (PPARs) PPARs are members of the nuclear receptor superfamily involved in lipid and carbohydrate metabolism.

Prenatal exposure to phthalates may affect children's mental, motor and behavioral development during the preschool year.

A 2009 study found that prenatal phthalate exposure was related to low birth weight in infants. Low birth weight is the leading cause of death in children under 5 years of age and increases the risk of cardiovascular and metabolic disease in adulthood. Another study found that women who deliver prematurely have, on average, up to three times the phthalate level in their urine compared to women who carry to term.

Several findings point to a statistically significant correlation between urine phthalate concentrations in children and symptoms of attention deficit hyperactivity disorder (ADHD)

Glyceryl triesters (triglycerides), following ingestion, are metabolised to monoglycerides, free fatty acids and glycerol, all of which are absorbed in the intestinal mucosa and undergo further metabolism. Medium chain triglycerides (C8-C10) appear to have relatively rapid metabolism and elimination from blood and tissues compared to long chain triglycerides (C16-C18). Little or no acute, subchronic or chronic oral toxicity was seen in animal studies unless levels approached a significant percentage of calorific intake. Subcutaneous injections of tricaprilyn in rats over a five-week period caused granulomatous reaction characterised by oil deposits surrounded by macrophages. Diets containing substantial levels of tributyrin produced gastric lesions in rats fed for 3-35 weeks; the irritative effect of the substance was thought to be the cause of tissue damage. Dermal application was not associated with significant irritation in rabbit skin; ocular exposures were, at most, mildly irritating to rabbit eyes. No evidence of sensitisation or photo-sensitisation was seen in a guinea pig maximisation test. Most of the genotoxicity test systems were negative.

Tricaprylin, trioctanoin and triolein have been used, historically, as vehicles in carcinogenicity testing of other chemicals. In one study, subcutaneous injection of tricaprilyn, in newborn mice, produced more tumours in lymphoid tissue than were seen in untreated animals whereas, in another study, subcutaneous or intraperitoneal injection in 4- to 6-week old female mice produced no tumours. Trioctanoin injected subcutaneously in hamster produced no tumours; when injected intraperitoneally in pregnant rats there was an increase in mammary tumours among the off-spring but similar studies in pregnant hamsters and rabbits showed no tumours in the off-spring.

The National Toxicological Program conducted a 2-year study in rats given tricaprilyn by gavage. The treatment was associated with a statistically significant dose-related increase in pancreatic acinar cell hyperplasia and adenoma but there were no acinar carcinomas.

Tricaprylin is not teratogenic to mice or rats but some reproductive effects were seen in rabbits. A low level of foetal eye abnormalities and a small percentage of abnormal sperm were reported in mice injected with trioctanoin.

Trioctanoin was also used as a vehicle control in a sperm abnormality test. Ten male control mice received an intraperitoneal injection of 0.25 ml trioctanoin 0.05 g/kg of benz[a]pyrene (known reproductive toxicant and mutagen) daily for 5 days and sperm from caudae epididymides analysed. Based on these studies there is no sufficient evidence to classify the trioctanoin as reproductive toxicant.

In the human body, high levels of triglycerides in the bloodstream have been linked to atherosclerosis, heart disease and stroke. However, the relative negative impact of raised levels of triglycerides compared to that of LDL:HDL ratios is as yet unknown. The risk can be partly accounted for by a strong inverse relationship between triglyceride level and HDL-cholesterol level. But the risk is also due to high triglyceride levels increasing the quantity of small, dense LDL particles

Under the conditions of a 2-year feed study with benzyl butyl phthalate, there was some evidence of carcinogenic activity in male rats based on an increased incidence of pancreatic acinar cell adenoma and of acinar cell adenoma or carcinoma (combined). There was equivocal evidence of carcinogenic activity of butyl benzyl phthalate in female rats based on a marginally increased incidence of pancreatic acinar cell adenoma and of transitional epithelial papilloma of the urinary bladder. Exposure to rats of butyl

benzyl phthalate in feed for 2-years resulted in focal hyperplasia in the pancreas in male rats and in transitional hyperplasia in the urinary bladder of female rats. Results from in vitro mutagenicity tests were uniformly negative; in vivo studies with mice showed bone marrow sister chromatid exchange at 23 and 42 hours while chromosome aberrations were induced in bone marrow cells of male mice sampled 17 hours after intraperitoneal injection of 5000 mg/kg butyl benzyl phthalate.

Embryolethality, independent of maternal toxicity, has been demonstrated in rats fed 2% butyl benzyl phthalate. Foetal malformations consisting of cleft palate and fusion of the sternbrae has been demonstrated in rats; results indicate that the susceptibility of the teratogenic effect of butyl benzyl phthalate varies with the development stage at the time of administration. Exposure during the first half of pregnancy resulted in embryolethality; similar exposure during the second half caused marked teratogenicity.

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Prolonged use of purgatives/ laxatives may produce watery diarrhoea with excessive loss of water and electrolytes (particularly potassium), muscular weakness and weight loss. Changes in intestinal musculature associated with malabsorption, and dilation of the bowel, similar to ulcerative colitis and to megacolon may also result. Cardiac and renal symptoms have also been reported.

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.

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.

Castor Crete "A" Clear	TOXICITY	IRRITATION
	Not Available	Not Available
castor oil	TOXICITY	IRRITATION
	Oral (Rat) LD50; >4800 mg/kg ^[1]	Eye (rabbit): 500 mg mild
		Skin (human): 50 mg/48h mild Skin (rabbit): 100 mg/24h SEVERE
castor oil, hydrogenated, ethoxylated	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50; >20000 mg/kg ^[2]	Skin (human): non irritant Skin: no adverse effect observed (not irritating) ^[1]
acetic acid glacial	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 1060 mg/kg ^[2]	Eye (rabbit): 0.05mg (open)-SEVERE
	Inhalation(Mouse) LC50; 1.405 mg/L4h ^[2]	Skin (human):50mg/24hr - mild Skin (rabbit):525mg (open)-SEVERE
tetrahydroxypropyl ethylenediamine	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): Non irritant *
	Oral (Rat) LD50; 3280 mg/kg ^[2]	Skin (rabbit): Non irritant *
butyl benzyl phthalate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >10000 mg/kg ^[2]	Not Available
	Inhalation(Rat) LC50; >6.7 mg/l4h ^[2]	
pine oil, synthetic	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[2]	Eye (rabbit): Severe *
	Inhalation(Rat) LC50; >4.76 mg/L4h ^[2]	Skin (rabbit) : Severe*
Oral (Rat) LD50; >2000 mg/kg ^[2]		
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	

Castor Crete "A" Clear

<p>Castor Crete "A" Clear</p>	<p>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 prohaptens 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. A prohaptens is a chemical that itself is non- or low-sensitising but that is transformed into a hapten in the skin (bioactivation) usually via enzyme catalysis. It is not always possible to know whether a particular allergen that is not directly reactive acts as a prohaptens or as a prohaptens, or both, because air oxidation and bioactivation can often give the same product (geraniol is an example). Some chemicals might act by all three pathways.</p> <p>Prohaptens Compounds that are bioactivated in the skin and thereby form haptens are referred to as prohaptens.</p> <p>In the case of prohaptens, 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.</p> <p>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 sensitizers has not been studied in detail. Skin sensitising prohaptens 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.</p> <p>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 prohaptens) 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 prohaptens. 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</p>
<p>CASTOR OIL, HYDROGENATED, ETHOXYLATED</p>	<p>This product contains partially hydrogenated fatty acids and/ or trans fatty acids.</p> <p>The consumption of trans fats increases the risk of coronary heart disease by raising levels of LDL cholesterol and lowering levels of "good" HDL cholesterol. There is an ongoing debate about a possible differentiation between trans fats of natural origin and trans fats of man-made origin but so far no scientific consensus has been found. Two Canadian studies have shown that the natural trans fat vaccenic acid, found in beef and dairy products, may have an opposite health effect and could actually be beneficial compared to hydrogenated vegetable shortening, or a mixture of pork lard and soy fat, by lowering total and LDL cholesterol and triglyceride levels. In lack of recognized evidence and scientific agreement, nutritional authorities consider all trans fats as equally harmful for health and recommend that consumption of trans fats be reduced to trace amounts.</p> <p>The use of hydrogenated oils in foods has never been completely satisfactory. Because the center arm of the triglyceride is shielded somewhat by the end fatty acids, most of the hydrogenation occurs on the end fatty acids, While full hydrogenation produces largely saturated fatty acids, partial hydrogenation results in the transformation of unsaturated cis fatty acids to trans fatty acids in the oil mixture due to the heat used in hydrogenation. Partially hydrogenated oils and their trans fats have increasingly been viewed as "unhealthy".</p> <p>Trans fat is the common name for unsaturated fat with trans-isomer (E-isomer) fatty acid(s). Because the term refers to the configuration of a double carbon-carbon bond, trans fats are sometimes monounsaturated or polyunsaturated, but never saturated. Trans fats do exist in nature but also occur during the processing of polyunsaturated fatty acids in food production. Trans fats occur naturally in a limited number of cases: vaccenyl and conjugated linoleyl (CLA) containing trans fats occur naturally in trace amounts in meat and dairy products from ruminants.</p> <p>The exact biochemical methods by which trans fats produce specific health problems are a topic of continuing research. One theory is that the human lipase enzyme works only on the cis configuration and cannot metabolise a trans fat. A lipase is a water-soluble enzyme that helps digest, transport, and process dietary lipids such as triglycerides, fats, and oils in most - if not all - living organisms. While the mechanisms through which trans fats contribute to coronary heart disease are fairly well understood, the mechanism for trans fat's effect on diabetes is still under investigation. Trans fatty acids may impair the metabolism of long-chain polyunsaturated fatty acids (LCPUFAs), but maternal pregnancy trans fatty acid intake has been inversely associated with LCPUFAs levels in infants at birth thought to underlie the positive association between breastfeeding and intelligence.</p> <p>There are suggestions that the negative consequences of trans fat consumption go beyond the cardiovascular risk. In general, there is much less scientific consensus asserting that eating trans fat specifically increases the risk of other chronic health problems:</p> <p>It has been suggested that the intake of both trans fats and saturated fats promote the development of Alzheimer disease, although not confirmed in an animal model. It has been found that trans fats impaired memory and learning in middle-age rats. The rats' brains of trans-fat eaters had fewer proteins critical to healthy neurological function. Inflammation in and around the hippocampus, the part of the brain responsible for learning and memory. These are the exact types of changes normally seen at the onset of Alzheimer's, but seen after six weeks, even though the rats were still young.</p> <p>There is a growing concern that the risk of type 2 diabetes increases with trans fat consumption.[52] However, consensus has not been reached. For example, one study found that risk is higher for those in the highest quartile of trans fat consumption. Another study has found no diabetes risk once other factors such as total fat intake and BMI were accounted for.</p> <p>Research indicates that trans fat may increase weight gain and abdominal fat, despite a similar caloric intake. A 6-year experiment revealed that monkeys fed a trans fat diet gained 7.2% of their body weight, as compared to 1.8% for monkeys on a mono-unsaturated fat diet. Although obesity is frequently linked to trans fat in the popular media, this is generally in the context of eating too many calories; there is not a strong scientific consensus connecting trans fat and obesity, although the 6-year experiment did find such a link, concluding that "under controlled feeding conditions, long-term TFA consumption was an independent factor in weight gain. TFAs enhanced intra-abdominal deposition of fat, even in the absence of caloric excess, and were associated with insulin resistance, with evidence that there is impaired post-insulin receptor binding signal transduction.</p> <p>Liver Dysfunction: Trans fats are metabolised differently by the liver than other fats and interfere with delta 6 desaturase. Delta 6 desaturase is an enzyme involved in converting essential fatty acids to arachidonic acid and prostaglandins, both of which are important to the functioning of cells.</p> <p>Infertility in women: One 2007 study found, "Each 2% increase in the intake of energy from trans unsaturated fats, as opposed to that from carbohydrates, was associated with a 73% greater risk of ovulatory infertility...".</p> <p>Major depressive disorder: Spanish researchers analysed the diets of 12,059 people over six years and found those who ate the most trans fats had a 48 per cent higher risk of depression than those who did not eat trans fats. One mechanism may be trans-fats' substitution for docosahexaenoic acid (DHA) levels in the orbitofrontal cortex (OFC). Very high intake of trans-fatty acids (43% of total fat) in mice from 2 to 16 months of age was associated with lowered DHA levels in the brain (p=0.001) When the brains of 15 major depressive subjects who had committed suicide were examined post-mortem and compared against 27 age-matched controls, the suicidal brains were found to have 16% less (male average) to 32% (female average) less DHA in the OFC. The OFC is known to control reward, reward expectation and empathy, which are all negatively impacted in depressive mood disorders, as well as regulating the limbic system></p> <p>Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will</p>

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	<p>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.</p> <p>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.</p> <p>On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing.</p> <p>Allergic Contact Dermatitis—Formation, Structural Requirements, and Reactivity of Skin Sensitizers. Ann-Therese Karlberg et al; Chem. Res. Toxicol. 2008, 21, 53-69</p> <p>Polyethylene glycols (PEGs) have a wide variety of PEG-derived mixtures due to their readily linkable terminal primary hydroxyl groups in combination with many possible compounds and complexes such as ethers, fatty acids, castor oils, amines, propylene glycols, among other derivatives. PEGs and their derivatives are broadly utilized in cosmetic products as surfactants, emulsifiers, cleansing agents, humectants, and skin conditioners.</p> <p>PEGs and PEG derivatives were generally regulated as safe for use in cosmetics, with the conditions that impurities and by-products, such as ethylene oxides and 1,4-dioxane, which are known carcinogenic materials, should be removed before they are mixed in cosmetic formulations. Most PEGs are commonly available commercially as mixtures of different oligomer sizes in broadly- or narrowly-defined molecular weight (MW) ranges. For instance, PEG-10,000 typically designates a mixture of PEG molecules (n = 195 to 265) having an average MW of 10,000. PEG is also known as polyethylene oxide (PEO) or polyoxyethylene (POE), with the three names being chemical synonyms. However, PEGs mainly refer to oligomers and polymers with molecular masses below 20,000 g/mol, while PEOs are polymers with molecular masses above 20,000 g/mol, and POEs are polymers of any molecular mass. Relatively small molecular weight PEGs are produced by the chemical reaction between ethylene oxide and water or ethylene glycol (or other ethylene glycol oligomers), as catalyzed by acidic or basic catalysts. To produce PEO or high-molecular weight PEGs, synthesis is performed by suspension polymerization. It is necessary to hold the growing polymer chain in solution during the course of the poly-condensation process. The reaction is catalyzed by magnesium-, aluminum-, or calcium-organoelement compounds. To prevent coagulation of polymer chains in the solution, chelating additives such as dimethylglyoxime are used</p> <p>Safety Evaluation of Polyethylene Glycol (PEG) Compounds for Cosmetic Use: Toxicol Res 2015; 31:105-136 The Korean Society of Toxicology http://doi.org/10.5487/TR.2015.31.2.105</p>
ACETIC ACID GLACIAL	<p>for acid mists, aerosols, vapours</p> <p>Data from assays for genotoxic activity <i>in vitro</i> 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 <i>in vivo</i> 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 <i>in vivo</i> 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 <i>in vitro</i>.</p> <p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>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.</p> <p>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.</p> <p>NOAELs following repeated exposure to acetic acid and its salts range from 210 mg/kg bw/day (2-4 month acetic acid drinking water study; systemic toxicity) to 3600 mg/kg bw/day (acetic acid, sodium salt, 4 week dietary study; no effects reported). Signs of irritation/corrosion at the site of contact as well as systemic toxicity have been reported. Prolonged inhalation exposure to acetic acid results in muscle imbalance, increase in blood cholinesterase activity, decreases in albumins and decreased growth at concentrations greater than 0.01 mg/m³/day. Groups of 20 mice/sex were given 0.025% sodium acetate in drinking water (about 60 mg/kg bw/day) for 1 week before breeding, during a 9-day breeding period and (females only) throughout pregnancy, lactation and until the offspring were weaned at 3 weeks of age. No effects on fertility were observed. The male offspring were given the same solution until they were 5-7 weeks old and were then examined in a 24-hour activity test. Examination of the litters revealed no overt deformities and normal pup weights at day 1 and day 21. The activity of offspring of the treated group was lower than that of controls during the first 12 hours but was similar during the second 12 hours. It is unknown if the decreased activity observed in the sodium acetate treated group to was a result of exposure <i>in utero</i> and/or post-weaning, since the pups were exposed during both time periods.). Acetic acid had no effects on implantation or on maternal or fetal survival in rats, mice or rabbits dosed via gavage during gestation days 6-19 at doses up to 1600 mg/kg/day. The number of abnormalities seen in either soft or skeletal tissues of the test groups did not differ from the number occurring in the controls. Sodium acetate had no effect on pregnant mice or offspring when mice were administered 1000 mg/kg bw, by gavage on days 8-12 of gestation.</p>
TETRAHYDROXYPROPYL ETHYLENEDIAMINE	<p>Non mutagenic by Ames test * [BASF]</p>
BUTYL BENZYL PHTHALATE	<p>The material may produce peroxisome proliferation. Peroxisomes are single, membrane limited, cytoplasmic organelles that are found in the cells of animals, plants, fungi and protozoa. Peroxisome proliferators include certain hypolipidaemic drugs, phthalate ester plasticisers, industrial solvents, herbicides, food flavours, leukotriene D4 antagonists and hormones. Numerous studies in rats and mice have demonstrated the hepatocarcinogenic effects of peroxisome proliferators, and these compounds have been unequivocally established as carcinogens. However it is generally conceded that compounds inducing proliferation in rats and mice have little, if any, effect on human liver except at very high doses or extreme conditions of exposure.</p> <p>Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).</p> <p>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. Reproductive effector in rats.</p>
PINE OIL, SYNTHETIC	<p>Ames tests (with and without metabolic activation). OECD guideline 471. Negative: Chromosome aberration test. OECD guideline 473. Negative * Chemox Pound Pine Oil 85% terpene alcohols Standard (synthetic)</p> <p>Epoxidation of double bonds is a common bioactivation pathway for alkenes. The allylic epoxides, so formed, were found to possess sensitising capacity <i>in vivo</i> and <i>in vitro</i> 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 prohaptenes, 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.</p> <p>Allergic Contact Dermatitis—Formation, Structural Requirements, and Reactivity of Skin Sensitizers. Ann-Therese Karlberg et al; Chem. Res. Toxicol. 2008, 21, pp 53–69 http://ftp.cdc.gov/pub/Documents/OEL/06.%20Dotson/References/Karlberg_2008.pdf</p> <p>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</p>

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	<p>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.</p> <p>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 sensitizers.</p> <p>Prehapten</p> <p>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 sensitizers. 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.</p> <p>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.</p> <p>Prohapten</p> <p>Compounds that are bioactivated in the skin and thereby form haptens are referred to as prohapten.</p> <p>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.</p> <p>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 sensitizers 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.</p> <p>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.</p>
<p>Castor Crete "A" Clear & ACETIC ACID GLACIAL & TETRAHYDROXYPROPYL ETHYLENEDIAMINE</p>	<p>Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.</p>
<p>Castor Crete "A" Clear & TETRAHYDROXYPROPYL ETHYLENEDIAMINE & PINE OIL, SYNTHETIC</p>	<p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>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.</p>
<p>Castor Crete "A" Clear & PINE OIL, SYNTHETIC</p>	<p>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 conjugal contact dermatitis occur.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a sufficient degree of fragrance contact</p>

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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, 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.

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 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 adaptation 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.

Some tumorigenic effects have been reported in animal studies using castor oil

The castor seed contains ricin, a toxic protein. Heating during the oil extraction process denatures and inactivates the protein. However, harvesting castor beans may not be without risk. Allergenic compounds found on the plant surface can cause permanent nerve damage, making the harvest of castor beans a human health risk.

The United States Food and Drug Administration (FDA) has categorized castor oil as "generally recognized as safe and effective" (GRASE) for over-the-counter use as a laxative with its major site of action the small intestine where it is digested into ricinoleic acid. Despite castor oil being widely used to start labor in pregnant women, to date there is not enough research to show whether it is effective to ripen the cervix or induce labour

Due to its foul taste a heavy dose of castor oil was formerly used as a humiliating punishment for children and adults. Victims of this treatment did sometimes die, as the dehydrating effects of the oil-induced diarrhea; however, even those victims who survived had to bear the humiliation of the laxative effects resulting from excessive consumption of the oil.

Several instances of sensitization to castor oil in cosmetics have been reported, including an allergic reaction to a make-up remover and contact dermatitis caused by use of a lipstick containing castor oil. Hypersensitivity reactions such as angioedema, rhinitis, asthma, and scarlatiniform rashes have been reported in factory workers involved in the extraction of castor oil, or in association with ingesting it.

Relatively few studies of castor oil toxicity have been conducted with experimental animals, and no studies were located concerning its absorption, distribution, metabolism, or excretion. Subcutaneous injection of 0.1 ml of castor oil in adult C57Bl/6 mice, daily for 4 weeks, was associated with the presence of electron dense lipid inclusions in parenchymal cells of the zona fasciculata of the adrenal gland. Gavage administration of 1 ml/kg to rhesus monkeys, daily for 4 days, caused mild morphological changes in the small intestine, characterized by lipid droplets along the mucosal epithelium and in the underlying lamina propria. This was considered a possible indication that castor oil had reduced lipid metabolism in the intestinal epithelium.

Because of widespread human exposure, large annual production and use, and the lack of studies characterizing the effect of exposures of moderate duration, the subchronic toxicity of castor oil was evaluated by administering diet formulations to F344/N rats and B6C3F1 mice for 13 weeks. Exposure to castor oil in the diet at concentrations up to 10% had no effect on survival of F344/N rats. No significant differences in average food consumption among each sex were observed, although food consumption of male and female rats receiving diets containing 10% castor oil was slightly lower than that of controls. Hematological effects of the castor oil diets among male rats included a slight decrease in MCHC at day 21 in those receiving the 10% diet; a statistically significant decrease in MCV among the 10% group; a decrease in MCH among the 5% and 10% groups; and an increase in platelets among the 1.25%, 5%, and 10% groups. The only change observed among female rats was a statistically significant decrease in reticulocyte counts at day 5 in groups receiving the 0.62% or 10% diets. None of these changes was considered biologically significant.

A treatment- and dose-related increase in the activity of serum alkaline phosphatase was observed in male and female rats at days 5 and 21, and at study termination. Total bile acids were increased among males receiving the higher dietary levels at days 5 and 21 but were not increased at

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study termination. Other minor changes included increases in albumin observed at study termination in males receiving 5% diets and at day 5 in females receiving 10% diets, and an increase in urea nitrogen at study termination in males that received 0.62% diets and a decrease at day 5 in females that received castor oil at 10% in the diet. Absolute liver weights and the liver-to-body-weight ratio were increased in male rats that received diets containing 10% castor oil. Heart-to-body-weight ratios were increased in groups of male rats receiving 0.62%, 2.5%, and 10% diets; however, absolute heart weights were not increased, and the differences in body weight ratios were small and not considered treatment related. Using light microscopy, it was determined there were no morphologic changes associated with the slight differences in organ weights between groups. In male rats, there was a slight decrease in epididymal weight (6-7%) which occurred in the middle- and high-dose groups, but this was not dose-related. There were no effects on any other male rat reproductive endpoint, or on any female rat reproductive endpoint. Although there was some variation in epididymal weights, their small magnitude and the absence of changes in other endpoints suggested that there was little or no evidence of any reproductive toxicity associated with castor oil exposure. Histopathologic examination revealed an absence of compound-related lesions in any organ or tissue of rats exposed to castor oil in the diet.

In genetic toxicity studies, castor oil (100-10,000 ug/plate) was not mutagenic in *Salmonella typhimurium* strains TA100, TA1535, TA97, or TA98 when tested with a preincubation protocol in the presence and the absence of exogenous metabolic activation (S9). Castor oil did not induce sister-chromatid exchanges or chromosome aberrations in Chinese hamster ovary cells treated with concentrations up to 5000 Oug/ml with and without S9. No induction of micronuclei was observed in peripheral blood erythrocytes of male and female B6C3F1 mice sampled at the termination of the 13-week study.

Castor oil was found not to be mutagenic or clastogenic in several in vitro genetic toxicity assays, and administration of diets containing up to 10% castor oil was not associated with toxicity to any specific organ, organ system, or tissue in this study

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 ug C16/cm² and 91.84 ug 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 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 GEs.

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.

For triglycerides:

Carboxylic acid esters will undergo enzymatic hydrolysis by ubiquitously expressed GI esterases. The rate of hydrolysis is dependant on the structure of the ester, and may therefore be rapid or rather slow. Thus, due to hydrolysis, predictions on oral absorption based on the physico-chemical characteristics of the intact parent substance alone may no longer apply.

When considering the hydrolysis product glycerol, absorption is favoured based on passive and active absorption of glycerol.

The Cosmetic Ingredient Review (CIR) Expert Panel has issued three final reports on the safety of 25 triglycerides, i.e., fatty acid triesters of glycerin

High purity is needed for the triglycerides. Previously the Panel published a final report on a diglycerides, and concluded that the ingredients in the diglyceride family are safe in the present practices of use and concentration provided the content of 1,2-diester is not high enough to induce epidermal hyperplasia. The Panel discussed that there was an increased level of concern because of data regarding the induction of protein kinase C (PKC) and the tumor promotion potential of 1,2-diacylglycerols. The Panel noted that, nominally, glyceryl-1,3-diester contain 1,2-diester, raising the concern that 1,2-diester could potentially induce hyperplasia. The Panel did note that these compounds are more likely to cause these effects when the fatty acid chain length is ≤ 14 carbons, when one fatty acid is saturated and one is not, and when given at high doses, repeatedly. Although minimal percutaneous absorption of triolein has been demonstrated in vivo using guinea pigs (but not hairless mice) and in vitro using full-thickness skin from hairless mice, the Expert Panel recognizes that, reportedly, triolein and tricaprylin can enhance the skin penetration of other chemicals, and recommends that care should be exercised in using these and other glyceryl triesters in cosmetic products.

The Panel acknowledged that some of the triglycerides may be formed from plant-derived or animal-derived constituents. The Panel thus expressed concern regarding pesticide residues and heavy metals that may be present in botanical ingredients. They stressed that the cosmetics industry should continue to use the necessary procedures to sufficiently limit amounts of such impurities in an ingredient before blending them into cosmetic formulations. Additionally, the Panel considered the risks inherent in using animal-derived ingredients, namely the transmission of infectious agents. Although tallow may be used in the manufacture of glyceryl tallowate and is clearly animal-derived, the Panel notes that tallow is highly processed, and tallow derivatives even more so. The Panel agrees with determinations by the U.S. FDA that tallow derivatives are not risk materials for transmission of infectious agents.

Finally, the Panel discussed the issue of incidental inhalation exposure, as some of the triglycerides are used in cosmetic sprays and could possibly be inhaled. For example, triethylhexanoin and trisostearin are reported to be used at maximum concentrations of 36% and 30%, respectively, in perfumes, and 14.7% and 10.4%, respectively, in face powders. The Panel noted that in aerosol products, 95% – 99% of droplets/particles would not be respirable to any appreciable amount. Furthermore, droplets/particles deposited in the nasopharyngeal or bronchial regions of the respiratory tract present no toxicological concerns based on the chemical and biological properties of these ingredients.

Coupled with the small actual exposure in the breathing zone and the concentrations at which the ingredients are used, the available information indicates that incidental inhalation would not be a significant route of exposure that might lead to local respiratory or systemic effects

Cosmetic Ingredient Review (CIR) : Amended Safety Assessment of Triglycerides as Used in Cosmetics August 2017

Glyceryl triesters are also known as triglycerides; ingested triglycerides are metabolized to monoglycerides, free fatty acids, and glycerol, all of which are absorbed in the intestinal mucosa and undergo further metabolism. Dermal absorption of Triolein in mice was nil; the oil remained at the application site. Only slight absorption was seen in guinea pig skin. Tricaprylin and other glyceryl triesters have been shown to increase the skin penetration of drugs. Little or no acute, subchronic, or chronic oral toxicity was seen in animal studies unless levels approached a significant percentage of caloric intake. Subcutaneous injections of Tricaprylin in rats over a period of 5 weeks caused a granulomatous reaction characterized by oil deposits surrounded by macrophages. Dermal application was not associated with significant irritation in rabbit skin. Ocular exposures were, at most, mildly irritating to rabbit eyes. No evidence of sensitization or photosensitization was seen in a guinea pig maximization test. Most of the genotoxicity test systems were negative. Tricaprylin, Trioctanoin, and Triolein have historically been used as vehicles in carcinogenicity testing of other chemicals. In one study, subcutaneous injection of Tricaprylin in newborn mice produced more tumors in lymphoid tissue than were seen in untreated animals, whereas neither subcutaneous or intraperitoneal injection in 4- to 6-week-old female mice produced any tumors in another study. Trioctanoin injected subcutaneously in hamsters produced no tumors. Trioctanoin injected intraperitoneally in pregnant rats was associated with an increase in mammary tumors in the offspring compared to that seen in offspring of untreated animals, but similar studies in pregnant hamsters and rabbits showed no tumors in the offspring. One study of Triolein injected subcutaneously in rats showed no tumors at the injection site. As part of an effort to evaluate vehicles used in carcinogenicity studies, the National Toxicology Program conducted a 2-year carcinogenicity study in rats given Tricaprylin by gavage. This treatment was associated with a statistically significant dose-related increase in pancreatic acinar cell hyperplasia and adenoma, but there were no acinar carcinomas, the incidence of mononuclear leukemia was less, and nephropathy findings were reduced, all compared to corn oil controls. Overall, the study concluded that Tricaprylin did not offer significant advantages over corn oil as vehicles in carcinogenicity studies. Trilaurin was found to inhibit the formation of neoplasms initiated by dimethylbenzanthracene (DMBA) and promoted by croton oil. Tricaprylin was not teratogenic in mice or rats, but some reproductive effects were seen in rabbits. A low level of fetal eye abnormalities and a small percentage of abnormal sperm were reported in mice injected with Trioctanoin as a vehicle control. Clinical tests of Trilaurin at 36.3% in a commercial product applied to the skin produced no irritation reactions. Trilaurin, Tristearin, and Tribehenin at 40%, 1.68%, and 0.38%, respectively, in commercial products were also negative in repeated-insult patch tests. Tristearin at 0.32% in a commercial product induced transient, mild to moderate, ocular irritation after instillation into the eyes of human subjects. Based on the enhancement of penetration of other chemicals by skin treatment with glyceryl triesters, it is recommended that care be exercised in using them in cosmetic products.

Cosmetic Ingredient Review (CIR) Expert Panel: Final Report on the Safety Assessment of Trilaurin etc: Int J Toxicol, 20 Suppl 4, 61-94 2001

For Group E aliphatic esters (polyol esters):

According to a classification scheme described by the American Chemistry Council' Aliphatic Esters Panel, Group E substances are esters of monoacids, mainly common fatty acids, and trihydroxy or polyhydroxyalcohols or polyols, such as pentaerythritol (PE), 2-ethyl-2-(hydroxymethyl)-1,3-propanediol or trimethylolpropane (TMP), and dipentaerythritol (diPE). The Group E substances often are referred to as "polyol esters" The polyol esters are unique in their chemical characteristics since they lack beta-tertiary hydrogen atoms, thus leading to stability against oxidation and elimination. The fatty acids often range from C5-C10 to as high as C18 (e.g., oleic, stearic, isostearic, tall oil fatty acids) in carbon number and generally are derived from naturally occurring sources. Group E esters may have multiple ester linkages and may include mixed esters derived from different carbon-length fatty acid mixtures. The lack of beta-tertiary hydrogen atoms in the structure of the polyol esters makes them characteristically and chemically stable against oxidation and elimination in comparison to other ester classes or groups. For these reasons, trimethylolpropane (TMP) and pentaerythritol (PE) esters with fatty acids of C5 to C10 carbon-chain length have applications as synthetic lubricants for passenger car motor oil and military and civilian jet engines. TMP and PE esters of C18 acids (e.g., isostearic and oleic acids) also have found use in synthetic lubricant applications, including refrigeration lubricants and hydraulic fluids. Because of their higher thermal stability characteristics, they also find use in a variety of high temperature applications such as industrial oven chain oils, high temperature greases, fire resistant transformer coolants and turbine engines

Polyol esters that are extensively esterified also have greater polarity, less volatility and enhanced lubricity characteristics. **Acute toxicity:** Depending on the degree of esterification, the polyol esters can be resistant or slow towards chemical or enzymatic hydrolysis (i.e., esterase or

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lipases) as a result of steric hindrance. PE and diPE esters that are capable of being enzymatically hydrolyzed will generate pentaerythritol or dipentaerythritol, and the corresponding fatty acids which, for most of the Group E esters, are comprised mainly of oleic, linoleic and stearic acids as well as the fatty acids in the C5-10 carbon-length. Similarly, TMP esters can undergo metabolism to yield trimethylolpropane (2-ethyl-2-hydroxymethyl-1,3-propanediol) and fatty acid constituents. Pentaerythritol and trimethylolpropane have been reported to have a low order of toxicity. The acute oral LD50 for these substances was greater than 2000 mg/kg indicating a relatively low order of toxicity. The similarity in the low order of toxicity for these substances is consistent with their similar chemical structure and physicochemical properties.

Metabolic studies of polyglyceryl esters indicated that these esters are hydrolyzed in the gastrointestinal (GI) tract, and utilization and digestibility studies supported the assumption that the fatty acid moiety is metabolized in the normal manner. Analytical studies have produced no evidence of accumulation of the polyglycerol moiety in body tissues.

In an acute dermal toxicity study in rats, the LD50 of 1,2,3-propanetriol, homopolymer, diisooctadecanoate was >5000 mg/kg. Low toxicity was reported in acute oral studies. In rats, the LD50 >2000 mg/kg for polyglyceryl-3 caprate, polyglyceryl-3 caprylate, polyglyceryl-4 caprate, diisostearoyl polyglyceryl-3 dimer diinoleate, and the LD50 was >5000 mg/kg for polyglyceryl-3 iso-stearate, polyglyceryl-3-oleate, polyglyceryl-2 diisostearate and polyglyceryl-3 diisostearate.

The ability to enhance skin penetration was examined for several of the polyglyceryl fatty acid esters.

Repeat dose toxicity: Polyol esters are generally well tolerated by rats in 28-day oral toxicity studies. NOAEL for these substances was 1000 mg/kg/day in Sprague-Dawley rats. The TMP ester of heptanoic and octanoic acid did not produce signs of overt systemic toxicity at any dose levels tested (i.e., 100, 300, and 1000 mg/kg/day). There were no treatment-related clinical in-life, functional observation battery, or gross postmortem findings. There were no treatment related mortality, and no adverse effects on body weight, food consumption, clinical laboratory parameters, or organ weights. However, there were increased numbers of hyaline droplets in the proximal cortical tubular epithelium of the 300 and 1000 mg/kg/day in male rats. Based on these findings (hyaline droplets), the NOAEL for this polyol ester was established at 100 mg/kg/day for male rats. Hyaline droplet formation observed in the male kidneys is believed to be a sex/species condition specific to only male rats, which has little relevance to humans.

The results from these repeated dose dermal toxicity studies suggest that polyol esters exhibit a low order of toxicity following repeated application. This may be attributable to similarities in their chemical structures, physicochemical properties, and common metabolic pathways (i.e., esters can be enzymatically hydrolyzed to the corresponding polyalcohol and the corresponding fatty acids). The polyol, hexanedioic acid, mixed esters with decanoic acid, heptanoic acid, octanoic acid and PE, was applied to the skin of groups of 10 (male and female) rats for five days a week for four (4) weeks at dose levels of 0, 125, 500 and 2000 mg/kg/day. Treated animals exhibited no signs indicative of systemic toxicity. No visible signs of irritation were observed at treatment sites. Microscopically, treated skin (viz., greater than or equal to 500 mg/kg/day) exhibited a dose-related increased incidence and severity of hyperplasia and hyperkeratosis of the epidermis and sebaceous gland hyperplasia. These effects were reversible. None of the minor changes in haematology and serum chemistry parameters were considered biologically significant. High dose females (2000 mg/kg/day) exhibited a significant increase in relative adrenal and brain weights when compared to the controls. These differences were attributed to the lower final body weight of the female animals. The NOAEL in this study for systemic toxicity was established as 500 mg /kg/day and 125 mg/kg/day for skin irritation.

Two 28-day study conducted with fatty acids, C5-10, esters with pentaerythritol (CAS RN: 68424-31-7) and dipentaerythritol ester of n-C5/iso-C9 acids (CAS RN: 647028-25-9) showed no signs of overt toxicity. The 90-day study pentaerythritol ester of pentanoic acids and isononanoic acid (CAS RN: 146289-36-3) did not show any signs of overt toxicity. However, increased kidney and liver weights in the male animals was observed. In conclusion, since the effects observed are not considered to be systemic and relevant for humans, the NOAEL was found to exceed 1000 mg/kg bw for all substances based on the result from the 28 and 90-day studies.

Reproductive and developmental toxicity: Since metabolism of the polyol esters can occur, leading to the generation of the corresponding fatty acids and the polyol alcohol (such as pentaerythritol, trimethylolpropane, and dipentaerythritol), the issue of whether these metabolites may pose any potential reproductive/developmental toxicity concerns is important. However, the polyol alcohols such as pentaerythritol, trimethylolpropane, and dipentaerythritol, would be expected to undergo further metabolism, conjugation and excretion in the urine. Available evidence indicates that these ester hydrolysates (i.e., hydrolysis products), primarily fatty acids (e.g., heptanoic, octanoic, and decanoic acids) and secondarily the polyol alcohols should exhibit a low order of reproductive toxicity. It can be concluded that this group of high molecular weight polyol esters should not produce profound reproductive effects in rodents.

Genotoxicity: Polyols tested for genetic activity in the Salmonella assay, have been found to be inactive. Several polyol esters have been adequately tested for chromosomal mutation in the in vitro mammalian chromosome aberration assay, and all were inactive. Two TMP esters were also tested for in vivo chromosomal aberration in rats, and both demonstrated no activity. Thus, it is unlikely that these substances are chromosomal mutagens.

Carcinogenicity: In a 2-yr study, 28 male and 28 female rats were fed 5% polyglyceryl ester in the diet. No adverse effects on body weight, feed consumption, haematology values, or survival rate were noted. Liver function tests and renal function tests performed at 59 and 104 wks of the study were comparable between the test group and a control group fed 5% ground nut oil. The carcass fat contained no polyglycerol, and the levels of free fatty acid, unsaponifiable residue and fatty acid composition of carcass fat were not different from the controls. Organ weights, tumour incidence and tumour distribution were similar in control and test groups. A complete histological examination of major organs showed nothing remarkable.

for benzyl butyl phthalate:

Repeat dose toxicity: The repeated-dose toxicity of BBP has been well investigated in studies, primarily in the rat, in which dose-response was well characterised. Effects observed consistently have been decreases in body weight gain (often accompanied by decreases in food consumption) and increases in organ to body weight ratios, particularly for the kidney and liver. Histopathological effects on the pancreas and kidney and haematological effects have also been observed. At higher doses, degenerative effects on the testes and, occasionally, histopathological effects on the liver have been reported. In specialised investigations, peroxisomal proliferation in the liver has been observed, although potency in this regard was less than that for other phthalates, such as bis(2-ethylhexyl) phthalate (DEHP).

Reproductive Toxicity and Teratology Studies

Groups of male F344/N rats given 20, 200, or 2200 mg/kg body weight butyl benzyl phthalate daily in feed for 10 weeks resulted in significantly decreased prostate gland, right cauda, right epididymis, and right testis weights at the highest dose versus those of the controls (NTP, 1997). Additionally, the epididymal spermatozoal concentrations in males given the 200 and 2200 mg/kg levels were significantly less than the controls. Females mated to 20 and 200 mg/kg males exhibited maternal body weights similar to those of females mated to control males. Litter data between the two dose groups and controls were also similar. Females mated to 2200 mg/kg males were initially found to be sperm positive; however, at necropsy, none of the females were pregnant. The fertility indices of the males and females were observed to be significantly lower than those of the controls.

Developmental Toxicity: In several well-conducted studies in rats and mice, BBP has induced marked developmental effects, but only at dose levels that induce significant maternal toxicity.

Carcinogenicity Studies

In a 2-year study, groups of male F344/N rats were given 120, 240, or 500 mg/kg body weight butyl benzyl phthalate daily in feed and females were given 300, 600, or 1200 mg/kg/day (NTP, 1997). At the highest dose, the incidences of pancreatic acinar cell adenoma and adenoma or carcinoma (combined) were significantly greater in males than those in the controls. In females, the incidence of transitional epithelial hyperplasia was significantly greater than that in the controls. Specifically, two transitional epithelial papillomas in the urinary bladder were seen.

It was concluded that there was "some evidence" of carcinogenicity in male rats, based on an increased incidence of pancreatic tumours, and equivocal evidence in female rats, based on marginal increases in pancreatic and bladder tumours. Dietary restriction prevented full expression of the pancreatic tumours and delayed appearance of the bladder tumours. There was no evidence of carcinogenicity in mice.

Genotoxicity Studies

At concentrations up to 11,550 ug/plate butyl benzyl phthalate in *Salmonella typhimurium* strains TA98, TA100, TA1535, and TA1537, no mutagenic response was obtained, in the presence or absence of metabolic activation (S9) (NTP, 1997). *In vitro* studies with L5178Y mouse lymphoma cells and cultured Chinese hamster ovary cells, both conducted with and without S9, were also negative. In germ cells of male *Drosophila melanogaster*, no induction of sex-linked recessive lethal mutations was observed. In contrast to these results, butyl benzyl phthalate gave positive responses in two *in vivo* mouse studies. In one experiment, sister chromatid exchanges were weakly positive at 23 and 42 hours. In the other study, chromosomal aberrations were induced in bone marrow cells 17 hours after intraperitoneal injection of 5000 mg/kg of the compound.

Castor Crete "A" Clear & BUTYL BENZYL PHTHALATE

Castor Crete "A" Clear

CASTOR OIL & PINE OIL, SYNTHETIC	No significant acute toxicological data identified in literature search.			
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

Castor Crete "A" Clear	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
castor oil	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	72h	Algae or other aquatic plants	>100mg/l	2
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
castor oil, hydrogenated, ethoxylated	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	504h	Crustacea	<0.001mg/l	2
	LC50	96h	Fish	>7.33mg/l	2
	EC50	72h	Algae or other aquatic plants	6.61mg/l	2
EC50	48h	Crustacea	>25mg/l	2	
acetic acid glacial	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	24h	Algae or other aquatic plants	0.08mg/l	2
	LC50	96h	Fish	31.3-67.6mg/l	2
	EC50	72h	Algae or other aquatic plants	29.23mg/l	2
EC50	48h	Crustacea	18.9mg/l	2	
tetrahydroxypropyl ethylenediamine	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	72h	Algae or other aquatic plants	4.25mg/l	2
	LC50	96h	Fish	~4600mg/l	2
	EC50	72h	Algae or other aquatic plants	150.67mg/l	2
ErC50	72h	Algae or other aquatic plants	150.67mg/l	2	
butyl benzyl phthalate	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	72h	Algae or other aquatic plants	0.1mg/l	1
	LC50	96h	Fish	0.46-0.55mg/l	4
	EC50	72h	Algae or other aquatic plants	0.5mg/l	1
	EC50	48h	Crustacea	0.97mg/l	1
EC50	96h	Algae or other aquatic plants	>2.69mg/l	1	
pine oil, synthetic	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	72h	Algae or other aquatic plants	~3.9mg/l	2
	LC50	96h	Fish	>=62<=80mg/l	2
	EC50	72h	Algae or other aquatic plants	~17mg/l	2
	EC50(ECx)	48h	Crustacea	15.3-25.2mg/L	4
	LC50	96h	Fish	14.4-18.9mg/L	4
EC50	48h	Crustacea	15.3-25.2mg/L	4	

Legend: Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data

On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and or observed environmental fate and behaviour, the material may present a danger, immediate or long-term and /or delayed, to the structure and/ or functioning of natural ecosystems.

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.

Continued...

Castor Crete "A" Clear

When spilled this product may act as a typical oil, causing a film, sheen, emulsion or sludge at or beneath the surface of the body of water. The oil film on water surface may physically affect the aquatic organisms, due to the interruption of the oxygen transfer between the air and the water

Oils of any kind can cause:

- drowning of water-fowl due to lack of buoyancy, loss of insulating capacity of feathers, starvation and vulnerability to predators due to lack of mobility
- lethal effects on fish by coating gill surfaces, preventing respiration
- asphyxiation of benthic life forms when floating masses become engaged with surface debris and settle on the bottom and
- adverse aesthetic effects of fouled shoreline and beaches

In case of accidental releases on the soil, a fine film is formed on the soil, which prevents the plant respiration process and the soil particle saturation. It may cause deep water infestation.

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.

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.

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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 greenhouse 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 Group A aliphatic esters (fatty acid esters):

Environmental fate:

Group A substances are rather lipophilic (log Kow 10-15) in character due to the large number of carbons in the ester molecule (e.g., 24,26, 31 carbons) and have relatively high boiling points. Owing to the non-volatile nature of these esters, their vapor pressures are very low and difficult to determine experimentally. Water solubility is also very low. Hydrolysis half lives and atmospheric photodegradation rates were calculated by EPIWIN. The monoester hydrolysis rates were determined to be quite low and not a significant environmental fate route. Fugacity modeling indicates that the fatty acid esters have similar distribution patterns in the environmental compartments (e.g., air, water, soil, sediment). Biodegradation of alkyl fatty acid esters are expected to occur extensively based on the reported 28 day test results (80-85% biodegradation, OECD 301D) for decyl oleate and for the 2-ethylhexyl ester of C16- 18 saturated and C18 unsaturated fatty acids (CAS 85049-37-2). Group A Substances are expected to be extensively biodegraded since the fatty acids in these esters are primarily comprised of palmitic, stearic or oleic acids, which are known to be rapidly biodegraded

Ecotoxicity:

Aquatic toxicity results have been reported for decyl oleate and fatty acid, C16- 18 saturated and C18 unsaturated, 2-ethylhexanoate They are not acutely toxic to fish (LC50 3200 mg/L). In daphnids, the acute LC50 was reported to be 17 mg/L and in algae, the LC50 was reported to be 40-42 mg/L based on biomass and growth rate endpoints. Because of their limited water solubility, the alkyl fatty acid esters and Group A esters are not likely to cause acute aquatic toxicity.

For butyl benzyl phthalate (BBP)

log Kow : 4.78-4.91

Half-life (hr) air : 24-120

Henry's atm m3 /mol: 1.30E-06

BCF : 663

Environmental fate:

Terrestrial Fate: A measured soil adsorption constant for BBP is 68-350; thus if released to land it will sorb to soil and should not leach appreciably, although it has been detected in ground water. The most significant fate process for BBP in soil will be biodegradation . Because of its low volatility, evaporation of BBP from soil is not expected to be significant.

Aquatic Fate: BBP has a log Kow of 4.77 . Thus, BBP released to waters will partition to solids such as sediment and biota. The primary fate mechanism for BBP will be biodegradation . At an initial concentration of 1 mg/L in a lake water microcosm, primary degradation accounted for >95% loss of BBP in 7 days; after 28 days, 51-65% of it had mineralized (ultimate degradation) . Based on the estimated Henry's Law constant, volatilization of BBP from water will not be significant except from shallow rivers or during high wind activity . Photodegradation and hydrolysis will not be significant, since the half-lives for these processes are >100 days.

Atmospheric Fate: BBP released to the atmosphere has an estimated half-life of 1-5 days . However, volatilization of BBP to the atmosphere is not expected to be a significant transport mechanism, since its vapor pressure is only 8.6 x 10⁻⁶ mm Hg at 20 C and its Henry's Law constant is <1.0 x 10⁻⁶ atm/mol m³

Ecotoxicity:

Fish LC50 (96 h): 1.7-43 mg/L

Invertebrate LC50 (96 h): 3.7 mg/L

Bioaccumulation : little

Anaerobic effects : sig degrad

Effects on algae and plankton: LC50(96)0.4-1mg/L

Degradation Biological: sig

processes Abiotic: not sig

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)	Major Stable Products produced following reaction with ozone.
Occupants (exhaled breath, ski oils, personal care products)	Isoprene, nitric oxide, squalene, unsaturated sterols, oleic acid and other unsaturated fatty acids, unsaturated oxidation products	Methacrolein, methyl vinyl ketone, nitrogen dioxide, acetone, 6MHQ, geranyl acetone, 4OPA, formaldehyde, nonanol, decanal, 9-oxo-nonanoic acid, azelaic acid, nonanoic acid.
Soft woods, wood flooring, including cypress, cedar and silver fir boards, houseplants	Isoprene, limonene, alpha-pinene, other terpenes and sesquiterpenes	Formaldehyde, 4-AMC, pinoaldehyde, pinic acid, pinonic acid, formic acid, methacrolein, methyl vinyl ketone, SOAs including ultrafine particles
Carpets and carpet backing	4-Phenylcyclohexene, 4-vinylcyclohexene, styrene, 2-ethylhexyl acrylate, unsaturated fatty acids and esters	Formaldehyde, acetaldehyde, benzaldehyde, hexanal, nonanal, 2-nonanal
Linoleum and paints/polishes containing linseed oil	Linoleic acid, linolenic acid	Propanal, hexanal, nonanal, 2-heptenal, 2-nonanal, 2-decanal, 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-ol, 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, 4OPA, 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-ol, 5-ethenyl-dihydro-5-methyl-2(3H) furanone, SOAs including ultrafine particles
Overall home emissions	Limonene, alpha-pinene, styrene	Formaldehyde, 4-AMC, pinaldehyde, 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

Castor Crete "A" Clear

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 cassiicola*, *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.

Acetic acid and its salts (the acetates) can be grouped together because of their close structural relationships, their natural occurrence in plants and animals, and their fundamental role in cell metabolism, particularly in the tricarboxylic acid cycle (also known as the citric acid or Krebs cycle), which is where humans get their energy.

- ▶ Acetic acid is degraded photochemically in the atmosphere to produce hydroxyl radicals (estimated typical half-life of 22 days). Physical removal of acetates on atmospheric particulates may occur via wet or dry deposition.
- ▶ Natural water will neutralise dilute solutions of acetic acid.
- ▶ Spills of acetic acid on soil will readily biodegrade - the biodegradation rate for acetic acid after 14 days under aerobic conditions is 74 days.
- ▶ In invertebrates the toxicity of acetic acid (EC50 = 50-450 mg/L, depending on test species) - under static conditions, the 48 hour EC50 value for acetic acid is 65 mg/L for aquatic invertebrates (the test media was not neutralised). When the test solutions are neutralised, to form acetates, the static 48 hour EC50 for acetic acid is 6000 mg/L. In renewal systems with aquatic invertebrates, 48 hour EC50s for acetic acid are 100 mg/L and 180 mg/L.
- ▶ Fish LC50 (96 h): 75-88 mg/L.
- ▶ Acetic acid is not expected to bioconcentrate in the aquatic system.
- ▶ Low concentrations of acetic acid are harmful to fish.
- ▶ Drinking water standards: none available.
- ▶ Soil Guidelines: none available.
- ▶ Air Quality Standards: none available

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
acetic acid glacial	LOW	LOW
tetrahydroxypropyl ethylenediamine	HIGH	HIGH
butyl benzyl phthalate	HIGH (Half-life = 180 days)	LOW (Half-life = 2.5 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
acetic acid glacial	LOW (LogKOW = -0.17)
tetrahydroxypropyl ethylenediamine	LOW (LogKOW = -2.0822)
butyl benzyl phthalate	MEDIUM (BCF = 663)

Mobility in soil

Ingredient	Mobility
acetic acid glacial	HIGH (KOC = 1)
tetrahydroxypropyl ethylenediamine	LOW (KOC = 53.97)
butyl benzyl phthalate	LOW (KOC = 9359)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ Containers may still present a chemical hazard/ danger when empty. ▶ Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> ▶ 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. <p>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.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> ▶ Reduction ▶ Reuse ▶ Recycling ▶ Disposal (if all else fails) <p>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.</p> <ul style="list-style-type: none"> ▶ DO NOT allow wash water from cleaning or process equipment to enter drains. ▶ It may be necessary to collect all wash water for treatment before disposal. ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▶ Where in doubt contact the responsible authority.
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Continued...

Castor Crete "A" Clear

- ▶ 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.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	

Land transport (DOT)

UN number	3082	
UN proper shipping name	Environmentally hazardous substance, liquid, n.o.s.	
Transport hazard class(es)	Class	9
	Subrisk	Not Applicable
Packing group	III	
Environmental hazard	Environmentally hazardous	
Special precautions for user	Hazard Label	9
	Special provisions	8, 146, 173, 335, IB3, T4, TP1, TP29

For Individual Packages of Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082 that contain LESS THAN the reportable quantity (5000 lbs) - Not Regulated

For Individual Packages of Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082 that contain MORE THAN the reportable quantity (5000 lbs) - Regulated and classified as below:

Air transport (ICAO-IATA / DGR)

UN number	3082	
UN proper shipping name	Environmentally hazardous substance, liquid, n.o.s. *	
Transport hazard class(es)	ICAO/IATA Class	9
	ICAO / IATA Subrisk	Not Applicable
	ERG Code	9L
Packing group	III	
Environmental hazard	Environmentally hazardous	
Special precautions for user	Special provisions	A97 A158 A197 A215
	Cargo Only Packing Instructions	964
	Cargo Only Maximum Qty / Pack	450 L
	Passenger and Cargo Packing Instructions	964
	Passenger and Cargo Maximum Qty / Pack	450 L
	Passenger and Cargo Limited Quantity Packing Instructions	Y964
	Passenger and Cargo Limited Maximum Qty / Pack	30 kg G

Sea transport (IMDG-Code / GGVSee)

UN number	3082	
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S.	
Transport hazard class(es)	IMDG Class	9
	IMDG Subrisk	Not Applicable
Packing group	III	
Environmental hazard	Marine Pollutant	
Special precautions for user	EMS Number	F-A, S-F
	Special provisions	274 335 969
	Limited Quantities	5 L

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

Not Applicable

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

Product name	Group
castor oil	Not Available
castor oil, hydrogenated, ethoxylated	Not Available
acetic acid glacial	Not Available
tetrahydroxypropyl ethylenediamine	Not Available
butyl benzyl phthalate	Not Available
pine oil, synthetic	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
castor oil	Not Available
castor oil, hydrogenated, ethoxylated	Not Available
acetic acid glacial	Not Available
tetrahydroxypropyl ethylenediamine	Not Available
butyl benzyl phthalate	Not Available
pine oil, synthetic	Not Available

SECTION 15 Regulatory information**Safety, health and environmental regulations / legislation specific for the substance or mixture****castor oil is found on the following regulatory lists**

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

US TSCA Chemical Substance Inventory - Interim List of Active Substances

castor oil, hydrogenated, ethoxylated is found on the following regulatory lists

US List of Active Substances Exempt from the TSCA Inventory Notifications (Active-Inactive) Rule

US TSCA Chemical Substance Inventory - Interim List of Active Substances

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

acetic acid glacial is found on the following regulatory lists

US - Massachusetts - Right To Know Listed Chemicals

US NIOSH Recommended Exposure Limits (RELs)

US ACGIH Threshold Limit Values (TLV)

US OSHA Permissible Exposure Limits (PELs) Table Z-1

US CWA (Clean Water Act) - List of Hazardous Substances

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

US DOE Temporary Emergency Exposure Limits (TEELs)

US TSCA Chemical Substance Inventory - Interim List of Active Substances

tetrahydroxypropyl ethylenediamine is found on the following regulatory lists

US DOE Temporary Emergency Exposure Limits (TEELs)

US TSCA Chemical Substance Inventory - Interim List of Active Substances

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

butyl benzyl phthalate is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List

US CWA (Clean Water Act) - Priority Pollutants

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

US CWA (Clean Water Act) - Toxic Pollutants

US - California - Biomonitoring - Priority Chemicals

US DOE Temporary Emergency Exposure Limits (TEELs)

US - California Proposition 65 - Maximum Allowable Dose Levels (MADLs) for Chemicals Causing Reproductive Toxicity

US EPA Integrated Risk Information System (IRIS)

US - California Proposition 65 - Reproductive Toxicity

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

US - California Safe Drinking Water and Toxic Enforcement Act of 1986 - Proposition 65 List

US TSCA Chemical Substance Inventory - Interim List of Active Substances

US - Massachusetts - Right To Know Listed Chemicals

pine oil, synthetic is found on the following regulatory lists

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

US TSCA Chemical Substance Inventory - Interim List of Active Substances

Federal Regulations**Superfund Amendments and Reauthorization Act of 1986 (SARA)****Section 311/312 hazard categories**

Flammable (Gases, Aerosols, Liquids, or Solids)	No
Gas under pressure	No
Explosive	No
Self-heating	No
Pyrophoric (Liquid or Solid)	No
Pyrophoric Gas	No

Continued...

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Corrosive to metal	No
Oxidizer (Liquid, Solid or Gas)	No
Organic Peroxide	No
Self-reactive	No
In contact with water emits flammable gas	No
Combustible Dust	No
Carcinogenicity	No
Acute toxicity (any route of exposure)	No
Reproductive toxicity	Yes
Skin Corrosion or Irritation	Yes
Respiratory or Skin Sensitization	Yes
Serious eye damage or eye irritation	Yes
Specific target organ toxicity (single or repeated exposure)	No
Aspiration Hazard	No
Germ cell mutagenicity	No
Simple Asphyxiant	No
Hazards Not Otherwise Classified	No

US. EPA CERCLA Hazardous Substances and Reportable Quantities (40 CFR 302.4)

Name	Reportable Quantity in Pounds (lb)	Reportable Quantity in kg
acetic acid glacial	5000	2270
butyl benzyl phthalate	100	45.4

State Regulations

US. California Proposition 65

WARNING: This product can expose you to chemicals including **butyl benzyl phthalate**, which is known to the State of California to cause birth defects or other reproductive harm. For more information, go to www.P65Warnings.ca.gov.

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (castor oil; castor oil, hydrogenated, ethoxylated; acetic acid glacial; tetrahydroxypropyl ethylenediamine; butyl benzyl phthalate; pine oil, synthetic)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	No (tetrahydroxypropyl ethylenediamine)
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	05/02/2022
Initial Date	08/15/2021

CONTACT POINT

PLEASE NOTE THAT TITANIUM DIOXIDE IS NOT PRESENT IN CLEAR OR NEUTRAL BASES

SDS Version Summary

Version	Date of Update	Sections Updated
4.5	05/02/2022	Exposure Standard, Ingredients, Supplier Information, Name

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.

Continued...

Castor Crete "A" Clear

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
AII: 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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