FORSPEC PRESWELL

FORSPEC PROTECTIVE COATINGS

Chemwatch Hazard Alert Code: 3

Chemwatch: **9010-10**
Version No: **2.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Initial Date: **12/03/2026**
Revision Date: **12/03/2026**
Print Date: **11/05/2026**
L.GHS.AUS.EN.E

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

Product Identifier

Product name	FORSPEC PRESWELL
Chemical Name	Not Applicable
Synonyms	Hydrophilic Waterstop
Chemical formula	Not Applicable
Other means of identification	Not Available

Relevant identified uses of the substance or mixture and uses advised against

Relevant identified uses	Concrete gap sealing and waterproofing.

Details of the manufacturer or importer of the safety data sheet

Registered company name	**FORSPEC PROTECTIVE COATINGS**
Address	22/872 Canterbury Rd. Roselands NSW 2196 Australia
Telephone	+61 2 8021 3517
Fax	Not Available
Website	www.forspec.com.au
Email	info@forspec.com.au

Emergency telephone number

Association / Organisation	**FORSPEC PROTECTIVE COATINGS**
Emergency telephone number(s)	+61 2 8021 3517 (Mon-Fri 7.30am to 5pm; Sat 8.30am to 12.30pm)
Other emergency telephone number(s)	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

HAZARDOUS CHEMICAL. NON-DANGEROUS GOODS. According to the WHS Regulations and the ADG Code.

Chemwatch Hazard Ratings

	Min	Max
Flammability	1	
Toxicity	1	
Body Contact	2	
Reactivity	1	
Chronic	3	

0 = Minimum
1 = Low
2 = Moderate
3 = High
4 = Extreme

Poisons Schedule	Not Applicable
Classification [1]	Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 1, Sensitisation (Respiratory) Category 1, Carcinogenicity Category 1B, Hazardous to the Aquatic Environment Long-Term Hazard Category 2
Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI***

Label elements

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

Hazard statement(s)

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H317	May cause an allergic skin reaction.
H318	Causes serious eye damage.
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H350	May cause cancer.
H411	Toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P261	Avoid breathing dust/fumes.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P284	[In case of inadequate ventilation] wear respiratory protection.
P273	Avoid release to the environment.
P202	Do not handle until all safety precautions have been read and understood.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

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.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P342+P311	If experiencing respiratory symptoms: Call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Collect spillage.

Precautionary statement(s) Storage

P405	Store locked up.

Precautionary statement(s) Disposal

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

No further product hazard information.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
Not Available		Polymer Acrylic Waterstop:
9003-04-7	30-60	sodium polyacrylate
9004-32-4	1-10	sodium carboxymethylcellulose
9003-55-8	1-10	styrene/ butadiene rubber
9006-04-6	1-10	natural rubber
Not Available	1-10	Modified polyurethane (hydrophilic)
471-34-1	1-10	calcium carbonate
8013-07-8	1-10	soybean oil, epoxidised
95-33-0	<10	N-cyclohexyl-2-benzothiazolesulfenamide
7704-34-9.	<10	sulfur
Not Available	<10	Other additives
Not Available		Hot melt adhesive backing:
64742-52-5.	NotSpec	naphthenic distillate, heavy, hydrotreated (severe)
9003-27-4	NotSpec	isobutylene homopolymer
13463-67-7	NotSpec	C.I. Pigment White 6
25038-32-8	NotSpec	styrene/ isoprene copolymer
64742-16-1	NotSpec	hydrocarbon resin, postpolymerised with maleic anhydride
69430-35-9	NotSpec	hydrocarbons, C6-20, hydrogenated, polymers
6683-19-8	NotSpec	pentaerythritol tetrabutylhydroxyhydrocinnamate
31570-04-4	NotSpec	tris(2,4-di-tert-butylphenyl)phosphite
Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available***	

SECTION 4 First aid measures

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Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Immediately hold eyelids apart and flush the eye continuously with running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation. For thermal burns: ▸ Decontaminate area around burn. ▸ Consider the use of cold packs and topical antibiotics. For first-degree burns (affecting top layer of skin) ▸ 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. For second-degree burns (affecting top two layers of skin) ▸ 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. To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort): ▸ 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. For third-degree burns Seek immediate medical or emergency assistance. In the mean time: ▸ 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	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor.
Ingestion	▸ 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.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 Firefighting measures

Extinguishing media

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

Special hazards arising from the substrate or mixture

Fire Incompatibility	▸ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result

Advice for firefighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Use water delivered as a fine spray to control fire and cool adjacent area. ▸ Avoid spraying water onto liquid pools. ▸ **DO NOT** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	▸ Combustible. ▸ Slight fire hazard when exposed to heat or flame. ▸ Heating may cause expansion or decomposition leading to violent rupture of containers. ▸ On combustion, may emit toxic fumes of carbon monoxide (CO).

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	▸ May emit acrid smoke. ▸ Mists containing combustible materials may be explosive. Combustion products include: ▸ carbon dioxide (CO_2) nitrogen oxides (NO_x) phosphorus oxides (PO_x) sulfur oxides (SO_x) metal oxides ▸ other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes. **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.
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	Environmental hazard - contain spillage. ▸ Remove all ignition sources. ▸ Clean up all spills immediately. ▸ Avoid breathing vapours and contact with skin and eyes. ▸ Control personal contact with the substance, by using protective equipment. ▸ Contain and absorb spill with sand, earth, inert material or vermiculite. ▸ Wipe up. ▸ Place in a suitable, labelled container for waste disposal.
Major Spills	Environmental hazard - contain spillage. ▸ Clear area of personnel and move upwind. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by all means available, spillage from entering drains or water courses. ▸ Consider evacuation (or protect in place). ▸ No smoking, naked lights or ignition sources. ▸ Increase ventilation. ▸ Stop leak if safe to do so. ▸ Water spray or fog may be used to disperse / absorb vapour. ▸ Contain or absorb spill with sand, earth or vermiculite. ▸ Collect recoverable product into labelled containers for recycling. ▸ Collect solid residues and seal in labelled drums for disposal. ▸ Wash area and prevent runoff into drains. ▸ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▸ 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	▸ **DO NOT** allow clothing wet with material to stay in contact with skin ▸ Avoid skin contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Prevent concentration in hollows and sumps. ▸ **DO NOT** enter confined spaces until atmosphere has been checked. ▸ Avoid smoking, naked lights or ignition sources. ▸ Avoid contact with incompatible materials. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Keep containers securely sealed when not in use. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
Other information	▸ Store below 38 deg. C. ▸ Store in original containers. ▸ Keep containers securely sealed. ▸ No smoking, naked lights or ignition sources. ▸ Store in a cool, dry, well-ventilated area. ▸ Store away from incompatible materials and foodstuff containers. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	10M per roll packed by film placed in carton box. ▸ Glass container is suitable for laboratory quantities ▸ Metal can or drum ▸ Packaging as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.

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Storage incompatibility	Dilute solutions of all sugars are subject to fermentation, either by yeast or by other microorganisms or enzymes derived from these, producing gases which can pressurise and burst sealed containers. Some microorganisms will produce hydrogen or methane, adding a fire and explosion hazard. Cellulose and its derivatives may react vigorously with calcium oxide, bleaching powder, perchlorates, perchloric acid, sodium chlorate, fluorine, nitric acid, sodium nitrate and sodium nitrite. May be incompatible with aminacrine hydrochloride, chlorocresol, mercuric chloride, phenol, resorcinol, tannic acid and silver nitrate. ▸ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	calcium carbonate	Calcium carbonate	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Workplace exposure limits for airborne contaminants (WEL list) (Effective from 1 December 2026) - Appendix A - Workplace Exposure Limits	calcium carbonate	Calcium carbonate	10 mg/m3	Not Available	Not Available	Containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	naphthenic distillate, heavy, hydrotreated (severe)	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	C.I. Pigment White 6	Titanium dioxide	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Workplace exposure limits for airborne contaminants (WEL list) (Effective from 1 December 2026) - Appendix A - Workplace Exposure Limits	C.I. Pigment White 6	Titanium dioxide	10 mg/m3	Not Available	Not Available	Containing no asbestos and < 1% crystalline silica.

MATERIAL DATA

None assigned. Refer to individual constituents.

Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure. ▸ Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulated area. ▸ Work should be undertaken in an isolated system such as a "glove-box" . Employees should wash their hands and arms upon completion of the assigned task and before engaging in other activities not associated with the isolated system. ▸ Within regulated areas, the carcinogen should be stored in sealed containers, or enclosed in a closed system, including piping systems, with any sample ports or openings closed while the carcinogens are contained within. ▸ Open-vessel systems are prohibited. ▸ Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation. ▸ Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system. ▸ For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. ▸ Except for outdoor systems, regulated areas should be maintained under negative pressure (with respect to non-regulated areas). ▸ Local exhaust ventilation requires make-up air be supplied in equal volumes to replaced air. ▸ Laboratory hoods must be designed and maintained so as to draw air inward at an average linear face velocity of 0.76 m/sec with a minimum of 0.64 m/sec. Design and construction of the fume hood requires that insertion of any portion of the employees body, other than hands and arms, be disallowed.
Individual protection measures, such as personal protective equipment	
Eye and face protection	When handling very small quantities of the material eye protection may not be required. For laboratory, larger scale or bulk handling or where regular exposure in an occupational setting occurs: ▸ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent] ▸ Face shield. Full face shield may be required for supplementary but never for primary protection of eyes. ▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].
Skin protection	See Hand protection below
Hands/feet protection	**NOTE:**

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	▸ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▸ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. ▸ Rubber gloves (nitrile or low-protein, powder-free latex, latex/ nitrile). Employees allergic to latex gloves should use nitrile gloves in preference. ▸ Double gloving should be considered. ▸ PVC gloves. ▸ Change gloves frequently and when contaminated, punctured or torn. ▸ Wash hands immediately after removing gloves. ▸ Protective shoe covers. [AS/NZS 2210] ▸ Head covering.
Body protection	See Other protection below
Other protection	▸ Employees working with confirmed human carcinogens should be provided with, and be required to wear, clean, full body protective clothing (smocks, coveralls, or long-sleeved shirt and pants), shoe covers and gloves prior to entering the regulated area. [AS/NZS ISO 6529:2006 or national equivalent] ▸ Employees engaged in handling operations involving carcinogens should be provided with, and required to wear and use half-face filter-type respirators with filters for dusts, mists and fumes, or air purifying canisters or cartridges. A respirator affording higher levels of protection may be substituted. [AS/NZS 1715 or national equivalent] ▸ Emergency deluge showers and eyewash fountains, supplied with potable water, should be located near, within sight of, and on the same level with locations where direct exposure is likely. ▸ Prior to each exit from an area containing confirmed human carcinogens, employees should be required to remove and leave protective clothing and equipment at the point of exit and at the last exit of the day, to place used clothing and equipment in impervious containers at the point of exit for purposes of decontamination or disposal. The contents of such impervious containers must be identified with suitable labels. For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. ▸ Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. ▸ For quantities up to 500 grams a laboratory coat may be suitable. ▸ For quantities up to 1 kilogram a disposable laboratory coat or coverall of low permeability is recommended. Coveralls should be buttoned at collar and cuffs. ▸ For quantities over 1 kilogram and manufacturing operations, wear disposable coverall of low permeability and disposable shoe covers. ▸ For manufacturing operations, air-supplied full body suits may be required for the provision of advanced respiratory protection. ▸ Eye wash unit. ▸ Ensure there is ready access to an emergency shower. ▸ For Emergencies: Vinyl suit

Respiratory protection

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	A-AUS P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	-	A-AUS / Class 1 P2	-
up to 100 x ES	-	A-2 P2	A-PAPR-2 P2 ^

^ - 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.

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- 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

· Respirators may be necessary when engineering and administrative controls do not adequately prevent exposures.
· The decision to use respiratory protection should be based on professional judgment that takes into account toxicity information, exposure measurement data, and frequency and likelihood of the worker's exposure - ensure users are not subject to high thermal loads which may result in heat stress or distress due to personal protective equipment (powered, positive flow, full face apparatus may be an option).
· Published occupational exposure limits, where they exist, will assist in determining the adequacy of the selected respiratory protection. These may be government mandated or vendor recommended.
· Certified respirators will be useful for protecting workers from inhalation of particulates when properly selected and fit tested as part of a complete respiratory protection program.
· Where protection from nuisance levels of dusts are desired, use type N95 (US) or type P1 (EN143) dust masks. Use respirators and components tested and approved under appropriate government standards such as NIOSH (US) or CEN (EU)
· Use approved positive flow mask if significant quantities of dust becomes airborne.
· Try to avoid creating dust conditions.
Where significant concentrations of the material are likely to enter the breathing zone, a Class P3 respirator may be required.
Class P3 particulate filters are used for protection against highly toxic or highly irritant particulates.
Filtration rate: Filters at least 99.95% of airborne particles
Suitable for:
· Relatively small particles generated by mechanical processes eg. grinding, cutting, sanding, drilling, sawing.
· Sub-micron thermally generated particles e.g. welding fumes, fertilizer and bushfire smoke.
· Biologically active airborne particles under specified infection control applications e.g. viruses, bacteria, COVID-19, SARS
· Highly toxic particles e.g. Organophosphate Insecticides, Radionuclides, Asbestos
Note: P3 Rating can only be achieved when used with a Full Face Respirator or Powered Air-Purifying Respirator (PAPR). If used with any other respirator, it will only provide filtration protection up to a P2 rating.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Blue colour rubber strip with one side white hot melt self-adhesive. Blue Waterstop: Upon contact with water, it rapidly absorbs moisture and forms a non-toxic, odorless, transparent gel-like substance that is insoluble in water. White Adhesive: The white hot-melt adhesive is inherently water-insoluble; however, when immersed underwater for extended periods, the adhesive layer may separate from the main body of the waterstop strip.

Physical state	Manufactured	**Relative density (Water = 1)**	Blue Waterstop:1.35-1.4; White Adhesive:1.0
Odour	Slight smell of rosin	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Applicable	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	35000 cps @160C
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	*>260 (White Adhesive)	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Applicable
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 (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available
Heat of Combustion (kJ/g)	Not Available	**Ignition Distance (cm)**	Not Available
Flame Height (cm)	Not Available	**Flame Duration (s)**	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	**Enclosed Space Ignition Deflagration Density (g/m3)**	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

a) Acute Toxicity	Based on available data, the classification criteria are not met.

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

Inhaled	The material is not thought to produce respiratory irritation (as classified by EC Directives using animal models). Nevertheless inhalation of vapours, fumes or aerosols, especially for prolonged periods, may produce respiratory discomfort and occasionally, distress. Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual. Cellulose, after a single intratracheal dose (15 mg per animal) brought about fibrosing granulomatous bronchioloalveolitis and an increase of IgA production in the bronchioalveolar lavage. Fibrosing alveolitis showed moderate progression as a function of time. Injury of Type I pneumocytes and incomplete repair of Type II pneumocytes were detected. The damage of alveolar epithelium initiated and activated a series of processes that led to definite pulmonary alterations and pulmonary fibrosis leading to disintegration of the alveolo-capillary morphological functional unit. Tatrai, E. et al: Journal of Applied Toxicology; 16(2) 129-135 (1996) Some health effects associated with wood, cotton, flax, jute and hemp particles or fibres are not attributable to cellulose content but to other substances and/or impurities. Inhalation of oil droplets/ aerosols may cause discomfort and may produce chemical pneumonitis.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. Large doses of cellulose may be administered orally as non-nutritive bulk. Doses of up to 30 g/day can be tolerated as bulk laxative. Extremely large oral doses may produce gastrointestinal disturbances. Sulfonamides and their derivatives may precipitate in kidney tubules causing extensive damage. Haemolytic anaemia may also result from use or exposure. Overdose may cause acidosis or hypoglycaemia with confusion and coma resulting. Hypersensitivity reactions may occur in predisposed individuals including those who have been sensitised by topical application. Deaths associated with therapies based on sulfonamide appear to be a result of hypersensitivity reaction, agranulocytosis, aplastic anaemia, other blood dyscrasias and renal and hepatic failure. Doses of 2 to 5 gms have produced toxicity and fatalities. Pathological findings include crystalluria, and necrotic or inflammatory lesions of the heart, liver, kidneys, bone marrow or other organs. Sulfonamides may damage the stem cell which acts as the precursor to components of the blood. Loss of the stem cell may result in pancytopenia (a reduction in the number of red and white blood cells and platelets) with a latency period corresponding to the lifetime of the individual blood cells. Granulocytopenia (a reduction in granular leukocytes) develops within days and thrombocytopenia (a disorder involving platelets), within 1-2 weeks, whilst loss of erythrocytes (red blood cells) need months to become clinically manifest. Aplastic anaemia develops due to complete destruction of the stem cells. Sulfonamides cross the placental barrier, are excreted in the breast milk and may produce adverse effects in the foetus/ embryo and newborn including agranulocytosis, haemolytic anaemia, jaundice and kernicterus. High molecular weight material; on single acute exposure would be expected to pass through gastrointestinal tract with little change / absorption. Occasionally accumulation of the solid material within the alimentary tract may result in formation of a bezoar (concretion), producing discomfort. Polysaccharides are not substantially absorbed from the gastrointestinal tract but may produce a laxative effect. Larger doses may produce intestinal obstruction or stomach concretions. Large quantities of the substituted polysaccharide, methylcellulose (as with other bulk laxatives), may temporarily increase flatulence. Oesophageal obstruction, by swelling, may occur if the material is swallowed dry. Doses of 3-9 gm hydroxypropylcellulose, fed to human subjects, at least one week apart, were eliminated within 96 hours. Animals fed on diets containing 3% or less, experienced no adverse effects. Higher levels produced malnutrition due to excessive bulk but caused no organic damage. In one dog, an oral dose of hydroxypropylcellulose produced diarrhoea and blood cell depression. Ingestion of hetastarch (hydroxyethyl amylopectin) has reportedly produced fever, chills, urticaria and salivary gland enlargement. Several of these effects may be due to contamination by other naturally occurring macromolecules extracted from the source material. Large volumes of ingested hetastarch may interfere with coagulation mechanisms and increase the risk of haemorrhage. Anaphylaxis has occurred. Infusions of dextrans may occasionally produce allergic reactions such as urticaria, hypotension and bronchospasm. Severe anaphylactic reactions may occasionally occur and death may result from cardiac and respiratory arrest. Nausea, vomiting, fever, joint pains, and flushing may also occur. Similarly, allergic reactions, sometimes severe (but rare) have been reported following ingestion or inhalation of tragacanth gums.
Skin Contact	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. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected. The material produces severe skin irritation; evidence exists, or practical experience predicts, that the material either: ▸ 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. **NOTE:** Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation. Ophthalmic solutions containing sulfonamides are reported to produce local irritation, reactive hyperaemia, burning and transient stinging, blurred vision and temporary impairment of depth perception. Hypersensitivity reactions may occur in predisposed individuals. Possible eye changes produced by phototoxic agents such as the sulfonamides include kerato-conjunctivitis or corneal and lens opacities.
Chronic	Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial number of individuals at a greater frequency than would be expected from the response of a normal population. Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking. 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.

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Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.
Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers
Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.
Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.
On the basis, primarily, of animal experiments, the material may be regarded as carcinogenic to humans. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in cancer on the basis of:
- appropriate long-term animal studies
- other relevant information
Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.
Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.
Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects.

Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects.
Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.
The celluose derivatives pass essentially unchanged through the gastrointestinal tract following oral administration to rats, dogs and man. Acute, subchronic, chronic toxicity, reproductive and developmental toxicity, genotoxicity and carcinogenicity studies of cellulose derivatives indicated that they are practically non-toxic when administered by oral, intraperitoneal, subcutaneous or dermal routes. While no clinical inhalation studies have been conducted, long term exposure to the dusts of cellulose ethers in manufacturing operations has not lead to any significant adverse effects. Ocular and dermal irritation studies indicate that the cellulose derivatives are, at most, minimally irritating and are not dermal sensitisers. Clinical studies confirm these results.
Amended Safety Assessment of Cellulose and Related Polymers as used in Cosmetics: Final Report of the Cosmetic Ingredient Review (CIR) Expert Panel: March 2009
Inhalation studies indicate that cellulose fibres may be fibrogenic; this finding continues to be the subject of extensive research. Cellulose is not considered an inert substance because :
· in rats, it causes granulomatous fibrosing alveolitis at the end of the third month after exposure,
· in rats there was an increase in the secretion of plasminogen activator and interleukin 1 as well as the release of lactate dehydrogenase from macrophages, in a manner similar to asbestos,
· there were increases in the incidence of obstructive lung diseases and bronchial asthma in humans at work and in the residential environment where exposure to cellulose was common,
· the substance may induce free radical production in human leucocytes.
Byssinosis is an occupational disease of the lungs caused by inhalation of cotton dust or dusts from other vegetable fibres such as flax, hemp, or sisal. Byssinosis is a chronic, asthma-like narrowing of the airways. Also called brown lung disease, byssinosis occurs almost exclusively in people who work with unprocessed cotton.
Cotton dust disease, "byssinosis", is well known among cotton mill workers. Cotton dust consists largely of cellulose fibre. Exposure to two components of the total dust, the "respirable" and "medium" fraction correlated significantly with the prevalence of respiratory symptoms. Inhalation exposure to a concentration of 0.3 to 0.4 mg/m3 of "fly-free" dust results in a 20% occurrence of byssinosis. "Fly-free" dust is the sum of respirable and medium-length fibres. At 0.46 mg/m3, Grade II byssinosis occurs. A byssinosis (all grades) prevalence of 20%, at 0.3 mg/m3 occurs when the fibre length is less than 15 um (aerodynamic equivalent diameter). Byssinosis is not caused by mechanical irritation but by reactions caused by pharmacologically active substances producing oedema or contraction of the smooth musculature of the airways. The causative agent is suspected to be an endotoxin, in turn, thought to be a cell wall component of bacteria found in cotton. Symptoms of byssinosis include chest tightness, wheezing and dyspnoea. Symptoms usually appear after an absence from work and may subside after 2-days of exposure. As the disease progresses, symptoms may persist for longer periods until they are constant. The individual may eventually exhibit chronic bronchitis and emphysema. Increased physical exertion may produce shortness of breath.

Repeated ingestion of sulfonamides used for therapeutic purposes has caused nausea, vomiting, abdominal pain, diarrhoea, anorexia, stomatitis, impaired folic acid absorption, exacerbation of porphyria, acidosis, liver injury with jaundice and hypoprothrombinemia, and pancreatitis. Hepatitis has been reported and may be fatal. Renal effects are often prominent and may include crystalluria, haematuria, proteinuria, pain and frequent urination, necrosis of the tubules, nephritic syndrome, and toxic necrosis with oliguria or anuria with azotemia. Neurologic effects include headache, drowsiness, insomnia, vertigo, tinnitus, hearing loss, mental depression, hallucinations, ataxia, muscular paralysis, peripheral neuropathy, transient lesions of the posterior spinal column, transverse myelitis, convulsions and unconsciousness. Haematological effects include eosinophilia, thrombocytopenia, leukopenia, neutropenia, agranulocytosis, pancytopenia, megoblastic anaemia, Heinz body anaemia and aplastic anaemia; petechiae and purpura may result. Acute haemolytic anaemia may also result (possibly as a result of hypersensitivity reactions) with people of African descent apparently more susceptible than Europeans - glucose-6-phosphate deficiency also appears to be a factor. Methaemoglobinaemia, sulfhaemoglobinaemia and cyanosis may also occur. Ocular effects may include acute transient myopia, keratitis and conjunctivitis with inflammation and chemosis accompanied by swelling of the lids and in more severe cases, photophobia. Cross-sensitivity amongst the sulfonamides is common and allergic reaction may occur following systemic use or topical application. Sensitisation may produce generalised skin eruptions, urticaria and pruritus. Stevens-Johnson syndrome; a severe form of erythema multiforme associated with wide-spread lesions of the skin, mucous membranes and which may be fatal in about 25% of cases, has occurred in patients treated with sulfonamides. This syndrome may produce conjunctival and corneal scarring, serum sickness, periorbital oedema, angioedema, arthritis, arthralgia, allergic myocarditis, decreased pulmonary function and eosinophilic pneumonia. Other effects of long-term therapy include fever, chills, alopecia, vasculitis, lupus erythematosus, oligospermia, infertility, hypothyroidism and on occasion, goiter and diuresis.

More severe responses to treatment include irreversible neuromuscular and central nervous system changes and fibrosing alveolitis. During sulfonamide treatment, direct exposure to sunlight should be avoided as photosensitisation dermatitis may develop. This form of phototoxic dermatitis may be contrasted to photoallergic dermatitis produced by specific sensitising agents through immunological intervention. Phototoxic reactions have been described following contact, ingestion or injection of causal agents. The chemical may reach the skin by the circulatory system following ingestion or following parenteral administration. The actual skin changes vary with the agent and circumstances of the exposure. Swelling and redness (erythema) frequently occur, and blistering may also result; increased skin temperature and pruritus may follow. This is analagous to irritant contact dermatitis and occurs immediately following contact.

Hyperpigmentation may also follow the reaction. Photodermatitis of this type requires activation of a chemical substance on the skin surface by UV radiation (290 to 490 nm wavelength) for its clinical expression. In all cases, inflammation develops on the body surfaces normally exposed to sunlight (dorsal hands, arms, neck, face), provided that the responsible photosensitiser also contacts the anatomic areas. Covered skin, the eyelids, submental chin and upper ears covered by hair, are characteristically spared. Phototoxic reactions, analogous to irritant contact dermatitis, are typically accompanied by immediate burning, stinging or "smarting" of the skin shortly following sun exposure, and clinical inflammation appears more like an acute sunburn than an eczematous dermatitis. Photoallergic dermatitis may result from contact with the material; this is characterised by an increased reactivity of the skin to ultra- violet (UV) and/or visible radiation produced by a

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chemical agent on an immunological basis and occurs after a latent period of days or months. This type of response can be elicited only in individuals who have been previously allergically sensitised to the chemical agent and appropriate radiation.

Photoallergic dermatitis is relatively rare (certainly more so than phototoxic dermatitis produced by non-immunological principals) and presents, clinically, as an eczematous dermatitis in sun-exposed areas (distinguishing it from phototoxic dermatitis which is analogous to contact irritant dermatitis and produces swelling, redness and even blistering); photoallergic dermatitis may eventually spread to areas covered by clothes. Lichenification (thickening with increased skin markings) and chronic pigmentary changes may also develop.
Photoallergic reactions may sometimes be followed by a persistent state of light reactivity (persistent light reactor) where clinical dermatitis recurs following exposure to sunlight alone, in the absence of the original initiating chemical. Studies in rats have shown that long-term administration of sulfonamides may produce thyroid malignancies; rats, however, appear to be more susceptible to the goiterogenic effects of sulfonamides than do other animal species. Sulfonamides may cause kernicterus in the neonate and their use is not recommended during pregnancy. Studies in rats and mice given high oral doses have shown that certain sulfonamides cause a significant incidence of cleft palate and other bony abnormalities in the foetus.
Studies indicate that diets containing large amounts of non-absorbable polysaccharides, such as cellulose, might decrease absorption of calcium, magnesium, zinc and phosphorus.
Polysaccharides are polymeric carbohydrates that consist of monosaccharide units, which are connected together with glycosidic bonds. Due to the structural variation of different monosaccharides as well as the innumerable ways that these building blocks link with each other, polysaccharides can be considered as structurally complex biomacromolecules. Polysaccharides originating from plants (e.g., starch and guar gum), microbes (e.g., xanthan), algae (e.g., alginates and carrageenans) and animals (e.g., glycogen and chitin) are frequently used in food. Starch, a high molar mass compound consisting of (1->4)-linked alpha-D-glucopyranosyl units, is an important energy nutrient that is abundant in common foods, such as cereals and root crops. Although many other food polysaccharides are not digested in the upper gastrointestinal tract of humans, they often serve functions other than being components giving nutritional value. For example, plant cell-wall polysaccharides, such as arabinoxylans and beta-glucan, exist in cereal-based foods, and “plant gums” are used as thickeners, emulsifiers, emulsion stabilizers, gelling agents and encapsulating agents. These non-digestible polysaccharides are important for health because they are considered as dietary fibre, which promote colon health, regulate post-prandial blood glucose levels and reduce serum cholesterol levels.
Despite the fact that nature provides various sources of polysaccharides, and that scientific research on their exploitation as food materials is increasingly active, a relatively low number of polysaccharides are authorized for use as food ingredients. For example, in the European Union (EU) and in Switzerland, among the permitted food additives (identified by an E number) only a small percentage are polysaccharide-based (native or structurally modified). The difference between other food ingredients and food additives is mainly the quantity used in any given product. Food ingredients can be consumed alone as food (e.g., starch), whereas food additives (e.g., carboxymethyl cellulose) are used in small quantities (usually less than 2%) relative to the total food composition but they, nonetheless, play an important role in the food products. Regarding food additive use in Europe, the European Food Safety Authority (EFSA) has an expert Panel on Food Additives and Nutrient Sources Added to Food (ANS), which evaluates the safety of food additives. Similarly, if new ingredients are released into the market, EFSA's Panel on Dietetic Products, Nutrition and Allergies (NDA) has the responsibility of evaluating the safety of Novel Food ingredients
The vast majority of polysaccharides used as food ingredients are plant-based. In addition to the cellulosic polysaccharides, other types of food-grade ingredients or additives, such as, vanillin aroma, glycerol esters of wood rosins (E445), xylitol (E967) and steryls/stanols, are derived from wood. The main components of wood are polysaccharides: cellulose (40–50 wt%) and hemicelluloses (20–35%), while lignin comprises 15–30% of wood mass.
The material contains a substantial proportion of a polymer considered to be of low concern (PLC). The trend towards production of lower molecular weight polymers (thus reducing the required level of solvent use and creating a more "environmentally-friendly" material) has brought with it the need to define PLCs as those
having molecular weights of between 1000 and 10000 and containing less than 10% of the molecules with molecular weight below 500 and less than 25% of the molecules with a molecular weight below 1000. These may contain unlimited low concern functional groups or moderate concern reactive functional groups with a combined functional group equivalent weight (FGEW, a concept developed by the US EPA describing whether the reactive functional group is sufficiently diluted by polymeric material) of a 1000 or more (provided no high concern groups are present) or high concern reactive functional groups with a FGEW of 5000 or more (FGEW includes moderate concern groups if present).
having molecular weights exceeding 10000 (without restriction on reactive groups).
inhalation of polymers with molecular weights > 70,000 Da has been linked with irreversible lung damage due to lung overloading and impaired clearance of particles from the lung, particularly following repeated exposure. If the polymer is inhaled at low levels and/or infrequently, it is assumed that it will be cleared from the lungs.
Reactive functional groups are in turn classified as being of low, moderate or high concern Classification of the polymer as a PLC, in accordance with established criteria, does not mean that hazards will not be associated with the polymer (during its import, manufacture, use, storage, handling or disposal). The polymer may, for example, contain a large number of particles in the respirable range, a hazard which may need to assessed in the health and safety risk assessment. Similarly a polymer with low concern reactive may be released into the environment in large quantities and produce an environmental hazard.
Whilst it is generally accepted that polymers with a molecular weight exceeding 1000 are unlikely to pass through biological membranes, oligomers with lower molecular weight and specifically, those with a molecular weight below 500, may. Estimations based on a "highly" dispersed polymer population (polydispersity = 10) suggests that the molecular weight of the polymer carrying a reactive group of high concern must be 5000 to be considered a PLC; similarly a polymer of approximate molecular weight 1000 could contain no more than one reactive group of moderate concern (for two moderate concern groups, the molecular weight would be about 2500).
Although polymers with a molecular weight above 10000 are normally considered to be PLCs (polymers of low concern) (because these are not expected to be absorbed by biological systems), this rule does NOT apply to water-absorbing polymers.
A two year oncogenicity study with high molecular weight polyacrylate (1 million), with no reactive functional groups, showed a statistically significant induction of lung tumours in rats exposed at 0.8 mg/m3 respirable particles.
Various lung effects such as inflammation, hyperplasia (abnormal increase in the number of cells composing a tissue or organ), scarring (fibrosis), changes in the air sac (alveolar) ducts of the lung, and tumours were noted in laboratory studies with rodents inhaling concentrations of water-absorbent sodium polyacrylate dusts greater than 0.05 mg/m3 (respirable particles) for the majority of their lives. Furthermore, some lung or lung cell effects were found in rodent laboratory studies of shorter duration.
Inhalation of polymers with molecular weights > 70,000 Da has been linked with irreversible lung damage due to lung overloading and impaired clearance of particles from the lung, particularly following repeated exposure. If the polymer is inhaled at low levels and/or infrequently, it is assumed that it will be cleared from the lungs.
Respiratory sensitisation may result in allergic/asthma like responses; from coughing and minor breathing difficulties to bronchitis with wheezing, gasping.

FORSPEC PRESWELL	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
sodium polyacrylate	**TOXICITY**	**IRRITATION**
	Oral (Mouse) LD50; >16000 mg/kg[2]	Eye (Rodent - rabbit): 2mg - Moderate
sodium carboxymethylcellulose	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[2]	Not Available
	Inhalation (Rat) LC50: >5.8 mg/L4h[2]	
	Oral (Guinea) LD50; 16000 mg/kg[2]	

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styrene/ butadiene rubber	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >20000 mg/kg[2]	Eye (Rodent - rabbit): 500mg/24H - Mild
	Oral (Rat) LD50: 71000 mg/kg[2]	Skin (Rodent - rabbit): 500mg - Mild
natural rubber	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
calcium carbonate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (Rodent - rabbit): 750ug/24H - Severe
	Inhalation (Rat) LC50: >3 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	Skin (Rodent - rabbit): 500mg/24H - Moderate
		Skin: no adverse effect observed (not irritating)[1]
soybean oil, epoxidised	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >19900 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: 6000 mg/kg[2]	Skin (Rodent - rabbit): 500mg - Mild
		Skin: no adverse effect observed (not irritating)[1]
N-cyclohexyl-2-benzothiazolesulfenamide	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (Rodent - rabbit): 100% - Mild
	Oral (Rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
		Skin (Human): 1%
		Skin (Rodent - rabbit): 100% - Mild
		Skin: no adverse effect observed (not irritating)[1]
sulfur	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (Human): 8ppm
	Inhalation (Rat) LC50: >5.43 mg/L4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	Skin: adverse effect observed (irritating)[1]
naphthenic distillate, heavy, hydrotreated (severe)	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation (Rat) LC50: 2.18 mg/l4h[2]	Skin (Rodent - rabbit): 500mg - Severe
	Oral (Rat) LD50: >5000 mg/kg[2]	Skin (Rodent - rabbit): 500mg - Severe
		Skin: no adverse effect observed (not irritating)[1]
isobutylene homopolymer	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
C.I. Pigment White 6	**TOXICITY**	**IRRITATION**
	dermal (hamster) LD50: >=10000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation (Rat) LC50: >2.28 mg/l4h[1]	Skin (Human): 300ug/3D (intermittent) - Mild
	Oral (Rat) LD50: >=2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
styrene/ isoprene copolymer	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
hydrocarbon resin, postpolymerised with maleic anhydride	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
hydrocarbons, C6-20, hydrogenated, polymers	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[2]	Not Available
	Oral (Rat) LD50: >5000 mg/kg[2]	
pentaerythritol tetrabutylhydroxyhydrocinnamate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: 3160 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation (Rat) LC50: >1.95 mg/l4h[2]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[2]	

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tris(2,4-di-tert-butylphenyl)phosphite	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation (Rat) LC50: >20 mg/L4h[2]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >6000 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***

SODIUM POLYACRYLATE	Polycarboxylates are of low toxicity by all exposure routes examined. Homopolymers(P-AA) are of low acute toxicity to the rat (LD50 > 5 g/kg bw/d) and are not irritating to the rabbit s skin and, at the most, slightly irritating to the eye. Further P-AA has no sensitising potential. The adverse effect after repeated inhalation dosing (91-d/rat) was a mild, reversible pulmonary irritation. This effect is considered as not substance related owing to the physical property of the respirable dust, which caused local and not systemic lung effects. There was neither evidence for a genotoxic potential of PAA using a variety of genetic endpoints in-vitro and in-vivo,nor for developmental toxicity or reprotoxicity in the rat. Based upon the available data, it is considered that exposure to polycarboxylates does not imply any particular hazard to humans The Cosmetic Ingredient Review (CIR) Expert Panel noted that these crosslinked alkyl acrylates are macromolecules that are not expected to pass through the stratum corneum of the skin, so significant dermal absorption is not expected. Therefore, topically applied cosmetics are not expected to result in systemic or reproductive and developmental toxicity or to have genotoxic or carcinogenic effects upon use. The Panel noted that cosmetic products containing these ingredients are reportedly used around the eyes, on the lips, and on other mucous membranes. Thus, crosslinked alkyl acrylates could be absorbed systemically through the relatively moist,n stratum cornea of the conjunctiva, lips,and other mucous membranes, and through ingestion when applied to the lips. However, the Panel noted that any absorption through healthy intact mucous membranes is likely to be not significant,primarily because of the relatively large molecular sizes. Furthermore, the chemically inert nature of the polymers precludes degradation to smaller absorbable species. Absorption of the polymers and their residual monomers in cosmetic products also would be limited after application to the lips or eye area based on the relatively small fractions of the applied products that might be inadvertently ingested or make direct contact with the conjunctiva. The Carbomers (Carbopols) are synthetic, high molecular weight, nonlinear polymers of acrylic acid, cross-linked with a polyalkenyl polyether. The Carbomer polymers are used in cosmetics and emulsifying agents at concentrations up to 50%. Acute oral animal studies showed that Carbomers-910, -934, -934P, -940, and -941 have low toxicities when ingested. Rabbits showed minimal skin irritation and zero to moderate eye irritation when tested with Carbomers-910 and -934. Subchronic feeding of rats and dogs with Carbomer-934 in the diet resulted in lower than normal body weights, but no pathological changes were observed. Dogs chronically fed Carbomer-934P manifested gastrointestinal irritation and marked pigment deposition within Kupffer cells of the liver. Clinical studies with Carbomers showed that these polymers have low potential for skin irritation and sensitization at concentrations up to 100%. Carbomer-934 demonstrated low potential for phototoxicity and photo-contact allergenicity. On the basis of the available information presented and as qualified in the report, it is concluded that the Carbomers are safe as cosmetic ingredients. Little toxicity data is available for acrylic crosspolymers; the acute dermal and oral toxicity data that were found indicated that these ingredients are not very toxic. The little genotoxicity data that were available reported negative results in Ames tests. Carcinogenicity data were not found in the published literature for the polymers, but data were available for the monomers. In an alternative method study, acrylates/vinyl neodecanoate crosspolymer was predicted to be a non-irritant. The non-human studies reported no to slight irritation with undiluted and weak sensitization with 2% aq., acrylates/C10-30 alkyl acrylate crosspolymer, no irritation with acrylates crosspolymer at 30% in olive oil, and no irritation or sensitization with sodium acrylates crosspolymer-2 (concentration not specified). Mostly, human testing with undiluted acrylates/C10-30 alkyl acrylate crosspolymer, acrylates crosspolymer, and acrylates/ethylhexyl acrylate crosspolymer, up to 2.5% aq. acrylates/vinyl isodecanoate crosspolymer, 1% aq. dilutions of formulations containing 2% acrylates/vinyl neodecanoate crosspolymer, and formulations containing up to 2.6% lauryl methacrylate/glycol dimethacrylate crosspolymers do not indicate any dermal irritation or sensitization. The only exception was a weak irritant response noted during an intensified Shelanski human repeated insult patch test (HRIPT) with undiluted acrylates/C10-30 alkyl acrylate crosspolymer. Alternative test methods for ocular irritation indicated that acrylates/vinyl isodecanoate crosspolymer and a formulation containing 1% lauryl methacrylate/glycol dimethacrylate crosspolymer are not likely ocular irritants. In studies using rabbits, undiluted acrylates/C10-30 alkyl acrylate crosspolymer produced minimal to moderate irritation, and it was considered a borderline irritant in unrinsed rabbit eyes. Acrylates crosspolymer, at 50% in olive oil,and sodium acrylates crosspolymer-2 did not appear to be ocular irritants in rabbit eyes.Two different risk assessments evaluating the carcinogenic endpoint for benzene that may be present in acrylates/ C10-30 alkyl acrylates crosspolymer resulted in different lifetime risk. One found that the risk was within the range associated with a 10exp 6 cancer risk, while the other reported a 20-fold greater risk. Final Safety Assessment: Crosslinked Alkyl Acrylates as Used in Cosmetics. Nov 2011 Cosmetic Ingredient Review (CIR) Expert Panel https://ntp.niehs.nih.gov/ntp/roc/nominations/2013/publiccomm/attachmentcir_508.pdf The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.
SODIUM CARBOXYMETHYLCELLULOSE	Neoplastic by RTECS criteria While thought to be uncommon, case reports of severe reactions to carboxymethylcellulose exist. In one such instance, a woman was known to experience anaphylaxis following exposure. Skin testing is believed to be a useful diagnostic tool for this purpose. Effects on inflammation, microbiota-related metabolic syndrome, and colitis are a subject of research Carboxymethyl cellulose has been found to cause inflammation of the gut, altering microbiota, and was found to be a triggering factor of inflammatory bowel diseases such as ulcerative colitis and Crohn's disease
STYRENE/ BUTADIENE RUBBER	Occupational exposures in the rubber-manufacturing industry are carcinogenic to humans (Group 1).IARC Working Groups There is sufficient evidence in humans for the carcinogenicity of occupational exposures in the rubber-manufacturing industry. Occupational exposures in the rubber-manufacturing industry cause leukaemia, lymphoma, and cancers of the urinary bladder, lung, and stomach. Also, a positive association has been observed between occupational exposures in the rubber-manufacturing industry and cancers of the prostate, oesophagus, and larynx.IARC Working Group. The multiple genetic and cytogenetic effects observed among workers employed in the rubber-manufacturing industry provide strong evidence to support genotoxicity as one mechanism for the observed increase in cancer risks. However, due to the complexity and changing nature of the exposure mixture and the potential interactions between exposures in the rubber-manufacturing industry, other mechanisms are also likely to play a role. While it is clear that exposure to some agents in the rubber-manufacturing industry has been reduced over time, the results of recent cytogenetic studies continue to raise concerns about cancer risks.

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	The rubber-manufacturing industry has used and still uses a wide variety of substances that belong to many different chemical categories, e.g. carbon black, aromatic amines, PAH, N-nitrosamines, mineral oils, other volatile organic compounds from curing fumes, trace amounts of monomers from synthetic rubber like 1,3-butadiene, acetonitrile, styrene, vinyl chloride, ethylene oxide, etc.. For this reason, it has been difficult to relate the observed cancer hazards in the rubber-manufacturing industry to exposure to specific chemicals.
NATURAL RUBBER	Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens). Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis. Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure.
CALCIUM CARBONATE	No evidence of carcinogenic properties. No evidence of mutagenic or teratogenic effects. 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. The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
SOYBEAN OIL, EPOXIDISED	. For epoxidised oils and their derivatives (EOD): Epoxidised Oils and Derivatives are epoxidised fatty acid esters(not to be confused with glycidyl fatty acid esters where the epoxide group is linked to the carboxyl ester moiety) The oils from which these products are derived are naturally occurring long chain fatty acid sources, and there is considerable overlap in the composition of the fatty acid portion of these products. They are primarily the C18 acids: oleic, linoleic, and linolenic acid. The alcohols are primary alcohols, diols or triols. This category consists of related fatty acid esters. Fatty acids, tall-oil, epoxidised, 2-ethylhexyl esters (ETP) 9-octadecenoic acid (Z)-, epoxidised, ester w/propylene glycol (EODA) epoxidised soybean oil (ESBO) epoxidised linseed oil (ELSO or ELO). ETP is a monoester with 2-ethylhexanol. EODA is a diester with propylene glycol. ESBO and ELSO are triesters with glycerol (triglycerides). These materials are considered a category for purposes of environmental and health hazard screening assessments because of their similarities in metabolism in microbial, aquatic and mammalian systems. Although there are no data regarding metabolism on these materials, it is known that fats are metabolized by esterases, and the materials in this category are fats. Carboxylesterases have been demonstrated to be present in many families of fish and aquatic invertebrates as well as mammals. The action of the esterase will result in a mixture of epoxidised fatty acids and 2-ethylhexanol from ETP, epoxidised fatty acids and propylene glycol from EODA, and epoxidised fatty acids and glycerol from ESBO and ELSO. The similarity of the epoxidised fatty acids which are the primary constituents of the metabolic products of these materials make it possible to use the data of representative materials within this category to assess the potential hazards across the category. Following metabolism, the alcohols from each material are a minor constituent of the metabolic products, and not produced in sufficient quantity to influence the toxicity profile of the EOD materials. Acute toxicity: In mammalian species these materials are expected to be absorbed and metabolized in a similar fashion, resulting in the release of similar free epoxidised fatty acids and lesser substituted alcohols or glycerides. Lipase is an enzyme that assists in the breakdown and digestion of fat in the body. Pancreatic lipase works at the oil/water interface since triglycerides are insoluble. During metabolism in the GI tract, pancreatic lipase preferentially hydrolyzes triglycerides to release the free fatty acids from the SN-1 and SN-3 (terminal) positions of the glycerol backbone. Other products of metabolism are mono- and diglycerides. The monoglycerides, diglycerides, and fatty acids can be absorbed. In a similar manner, pancreatic lipase and other digestive enzymes have been shown to hydrolyse propylene glycol monoesters and diesters in vitro. The absorption, metabolism and hydrolysis of propylene glycol distearate (which is structurally similar to EODA) were studied in rats using isotopically labelled compounds. These processes for the propylene glycol esters were found to be similar to those of the glycerol esters. So, there is evidence that the propylene glycol esters of fatty acids are hydrolyzed to propylene glycol and fatty acids. The ETP structure is similar to that of the monoglycerides, formed from the ELSO and ESBO. Further hydrolysis can also occur via carboxylesterase activity. Single exposure (acute) studies indicate that ESBO is practically non-toxic if swallowed (rat LD50 22,400 mg/kg) or absorbed through skin (rat LD50 19,900 mg/kg) and slightly irritating to rabbit eyes and skin. Exposure to concentrated ESBO vapours for 8 hrs produced no deaths in rats. Numerous acute toxicity studies have been conducted with ETP, ESBO and ELSO via the dermal and oral route of exposure. Acute toxicity studies were not available for EODA. The dermal LD50s of ETP, ESBO and ELSO in rabbits range from greater than 16 mL/kg bw (ELSO) to greater than 20 mL/kg bw (ETP and ESBO). The oral LD50s of ETP, ESBO and ELSO in rats range from 14.9 mL/kg bw (ELSO) to greater than 16 mL/kg bw (ETP) up to 41.5 mL/kg bw (ESBO). Similar acute toxicity values would be expected for EODA. There are no valid definitive skin irritation studies for any of the EOD materials, however the results from dermal toxicity studies with ETP, ESBO and ELSO indicate transient erythema and desquamation (no to slight skin irritation), similar effects are anticipated for EODA. Eye irritation studies are available for ETP and ESBO indicating no damage is observed, similar effects (no eye irritation) are expected for ELSO and EODA. Skin sensitisation tests with ESBO and ETP indicate the EOD materials are not sensitisers. No skin allergy was observed in guinea pigs following repeated exposure of ESBO. Repeat dose toxicity: Long-term dietary administration of ESBO to rats produced increased mortality, reduced body weight gains, kidney and liver changes (enlarged, fatty infiltration of the liver), degeneration of the testes, and slight changes in the uterus. Life-time application to the skin of mice or life-time administration in the diet to rats did not increase the incidence of tumors. No effects were seen on the ability of male or female rats to reproduce or on the development of the offspring when exposed orally prior to mating. Repeat dose studies by the oral route of exposure (gavage or dietary) have been conducted with ETP and ESBO. The NOAEL for ETP in a standard OECD Guideline 422 (rat) study was considered to be 1000 mg/kg bw/d (the highest dose tested). In a 90 day subchronic study, male and female rats were given 0.04, 0.2, 1.0 or 5.0% (5% is approximately 1.25 g/kg bw/d) ESBO. (Liver weights were increased in rats fed ESBO at 1% or 5% in diet. Gross findings indicated a test article-related effect of ESBO on liver and kidney at 1% and 5%. The NOAEL was 0.2% ESBO; the LOAEL was 1% ESBO. In a 2-year rat feeding study, a LOAEL of 1% ESBO was described based on increased liver and kidney weights. No effects were

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	observed at .5% ESBO. Rats were given diets containing up to 5% ESBO for 15 weeks. Temporary slowed growth and liver and kidney enlargement at concentrations greater than 1.5% was observed. The LOAEL was greater than 1.5% ESBO. Groups of three dogs were fed up to 5% ESBO once per day for one year. Dogs fed 5% ESBO lost weight, and fatty liver changes (fatty infiltration) in one dog given 5% ESBO were observed. Food intake and body weight were decreased at 5% ESBO. The LOAEL was 5% ESBO. Genotoxicity: No genetic changes were observed in tests with ESBO using bacteria and human or animal cells In vitro, ETP and ESBO were negative in bacterial or mammalian gene mutations assays. ETP and ESBO did not induce chromosomal aberrations in Chinese hamster V79 or human lymphocyte cells. EODA and ELSO are not expected to be mutagenic in bacterial or mammalian systems based on the lack of mutagenicity of ETP and ESBO. Carcinogenicity: Chronic/carcinogenicity studies were conducted with ESBO by the oral route (dietary). ESBO was not carcinogenic when fed to rats at up to 2.5% of the diet. Further, the NOAEL was 2.5% providing an average daily intake of approximately 1.0 g/kg bw in males and 1.4 g/kg bw in females. There were no treatment-related histopathologic lesions and there was no evidence of carcinogenicity of ESBO in groups of male and female rats given ESBO in the diet at 0, 0.1, 0.5, 1.0, 2.5, and 5.0%. A similar lack of carcinogenic response is expected for ETP, EODA and ELSO. Reproductive and developmental toxicity: ETP and ESBO have been evaluated for the potential to cause developmental toxicity in rats and were not teratogenic, nor did they demonstrate reproductive toxicity. In a standard OECD Guideline 422 rat study with ETP, there were no adverse effects on reproductive parameters, and the NOAEL (maternal) and NOEL for reproduction was considered to be 1000 mg/kg bw/d. Daily administration of ESBO up to 1000 mg/kg bw/d did not induce any toxic effect in parent male or female animals, did not disturb their capacity for reproduction and did not impair development of the F1 offspring. Based on the lack of reproductive toxicity or developmental effects with ETP and ESBO, EODA and ELSO are not expected to cause developmental toxicity or to demonstrate reproductive toxicity.
N-CYCLOHEXYL-2-BENZOTHIAZOLESULFENAMIDE	for N,N-dicylohexyl-2-benzothiazolesulfenamide Not mutagenic in Ames assay * Effects on the eye, somnolence, liver and blood tumours, foetotoxicity, foetolethality, extra-embryonic structures, effects on fertility recorded Equivocal tumourigen by RTECS criteria. for benzothiazole and morpholine-based thiazoles: All materials in this category contain a morpholine group attached to a benzothiazole group [benzene ring + thiazole ring] or to another morpholine group via a sulfur or sulfur-sulfur bond The data on acute mammalian toxicity indicate that the members of this category pose a low concern for acute toxicity. The most likely route of consumer exposure is skin contact from rubber or latex articles (containing these thiazoles as manufacturing aids). Skin irritation, or possibly an allergic skin reaction may occur, but only in sensitive individuals subjected to prolonged and repeated exposure, especially under moist conditions. **Mutagenicity:** Data from bacterial reverse mutation assays, ***in vitro*** and ***in vivo*** chromosome aberration studies, as well as additional supporting ***in vitro*** and ***in vivo*** genetic toxicity studies were reviewed, and the findings indicate a low concern for mutagenicity The predominant fate of the thiazole ring is oxidative ring scission catalysed by cytochrome P450 (CYP) and formation of the corresponding alpha-dicarbonyl metabolites and thioamide derivatives. The well-established toxicity associated with thioamides and thioureas has led to the speculation that thiazole toxicity is attributed to ring scission yielding the corresponding thioamide metabolite. Ring opening has also been observed in benzothiazoles. For instance, benzothiazole itself is converted to S-methylmercaptoaniline. **WARNING:** This substance has been classified by the IARC as Group 2A: Probably Carcinogenic to Humans. for 2-mercaptobenzothiazole (MBT) The sulfenamide group (-NH-C(=S)-S-) is the prime determinant of toxicity for all members of the Benzothiazole-based Thiazoles category. The acute and subchronic toxicity of MBT is relatively low. Skin and eye irritation effects are not present or are mild, but allergic skin reaction is possible in susceptible persons. Data from ***in vitro*** and ***in vivo*** studies indicate a low concern for mutagenicity caused by MBT. Mercaptobenzothiazole (MBT) is a potent skin sensitiser in man and cross reactions with other rubber chemicals can occur. It is also a skin irritant in man. A solution of the sodium salt caused irreversible damage to the rabbit eye. MBT and its sodium salt were of low acute general toxicity in laboratory animals treated by the oral and dermal routes. The target sites following oral administration included the central nervous system (CNS), lung, liver, kidney and gastro-intestinal tract, and rabbits treated dermally also suffered CNS effects. According to brief summaries, feeding MBT to rats produced no conclusive evidence of reproductive effects. Comprehensive oral studies generated some evidence of carcinogenicity in rats; results in mice were inconclusive. MBT and its sodium salt gave no evidence of mutagenicity in Ames bacterial assays, but MBT was mutagenic and induced chromosomal damage in mammalian cells in culture.
NAPHTHENIC DISTILLATE, HEAVY, HYDROTREATED (SEVERE)	Based on laboratory and animal testing, exposure to the material may result in irreversible effects and mutations in humans. Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length,with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent that iso- or cyclo-paraffins. The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption,is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver. The materials included in the Lubricating Base Oils category are related from both process and physical-chemical perspectives; The potential toxicity of a specific distillate base oil is inversely related to the severity or extent of processing the oil has undergone, since: · The adverse effects of these materials are associated with undesirable components, and · The levels of the undesirable components are inversely related to the degree of processing; · Distillate base oils receiving the same degree or extent of processing will have similar toxicities; · The potential toxicity of ***residual base oils*** is independent of the degree of processing the oil receives. · The reproductive and developmental toxicity of the distillate base oils is inversely related to the degree of processing. The degree of refining influences the carcinogenic potential of the oils. Whereas mild acid / earth refining processes are inadequate to substantially reduce the carcinogenic potential of lubricant base oils, hydrotreatment and / or solvent extraction methods can yield oils with no carcinogenic potential. Unrefined and mildly refined distillate base oils contain the highest levels of undesirable components, have the largest variation of hydrocarbon molecules and have shown the highest potential carcinogenic and mutagenic activities. Highly and severely refined distillate base oils are produced from unrefined and mildly refined oils by removing or transforming undesirable components. In comparison to unrefined and mildly refined base oils, the highly and severely refined distillate base oils have a smaller range of hydrocarbon molecules and have demonstrated very low mammalian toxicity. Mutagenicity and carcinogenicity testing of residual oils has been negative, supporting the belief that these materials lack biologically active components or the components are largely non-bioavailable due to their molecular size. Toxicity testing has consistently shown that lubricating base oils have low acute toxicities. Numerous tests have shown that a lubricating base oil s mutagenic and carcinogenic potential correlates with its 3-7 ring polycyclic aromatic compound (PAC) content, and the level of DMSO extractables (e.g. IP346 assay), both characteristics that are directly related to the degree/conditions of processing

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Skin irritating is not significant (CONCAWE) based on 14 tests on 10 CASs from the OLBO class (Other Lubricant Base Oils). Each study lasted for 24 hours, a period of time 6 times longer than the duration recommended by the OECD method).
Eye irritation is not significant according to experimental data (CONCAWE studies) based on 9 “in vivo” tests on 7 CASs from the OLBO class(Other Lubricant Base Oils).
Sensitisation: The substance does not cause the sensitization of the respiratory tract or of the skin. (CONCAWE studies based on 14 tests on 11 CASs from the OLBO class(Other Lubricant Base Oils))
Germ cell mutagenicity: The tests performed within the ‘in vivo” studies regarding gene mutation at mice micronuclei indicated negative results (CONCAWE studies. AMES tests had negative results in 7 studies performed on 4 CASs from the OLBO class(Other Lubricant Base Oils)).
Reproduction toxicity: Reproduction / development toxicity monitoring according to OECD 421 or 422 methods. CONCAWE tests gave negative results in oral gavage studies. Pre-birth studies regarding toxicity in the unborn foetus development process showed a maternal LOAEL (Lowest Observed Adverse Effect Level) of 125 mg/kg body/day, based on dermal irritation and a NOAEL (No Observable Adverse Effect Level) of 2000 mg/kg body/day, which shows that the substance is not toxic for reproduction.
STOT (toxicity on specific target organs) – repeated exposure: Studies with short term repeated doses (28-day test) on rabbit skin indicated the NOAEL value of 1000 mg/kg. NOAEL for inhalation, local effects > 280 mg/m3 and for systemic effects NOAEL > 980 mg/m3.
Sub-chronic toxicity
90-day study Dermal: NOAEL > 2000 mg/kg (CONCAWE studies).
Repeat dose toxicity:
Oral
NOAEL for heavy paraffinic distillate aromatic extract could not be identified and is less than 125 mg/kg/day when administered orally.
Inhalation
The NOAEL for lung changes associated with oil deposition in the lungs was 220 mg/m3. As no systemic toxicity was observed, the overall NOAEL for systemic effects was > 980 mg/m3.
Dermal
In a 90 day subchronic dermal study, the administration of Light paraffinic distillate solvent extract had an adverse effect on survivability, body weights, organ weights (particularly the liver and thymus), and variety of haematology and serum chemistry parameters in exposed animals. Histopathological changes which were treatment-related were most prominent in the adrenals, bone marrow, kidneys, liver, lymph nodes, skin, stomach, and thymus. Based on the results of this study, the NOAEL for the test material is less than 30 mg/kg/day.
Toxicity to reproduction:
Mineral oil (a white mineral oil) caused no reproductive or developmental toxicity with 1 mL/kg/day (i.e., 1000 mg/kg/day) in an OECD 421 guideline study, but did cause mild to moderate skin irritation. Therefore, the reproductive/developmental NOAEL for this study is =1000 mg/kg/day and no LOAEL was determined.
Developmental toxicity, teratogenicity:
Heavy paraffinic distillate furfural extract produced maternal, reproductive and foetal toxicity. Maternal toxicity was exhibited as vaginal discharge (dose-related), body weight decrease, reduction in thymus weight and increase in liver weight (125 mg/kg/day and higher) and aberrant haematology and serum chemistry (125 and/or 500 mg/kg/day). Evidence of potential reproductive effects was shown by an increased number of dams with resorptions and intrauterine death. Distillate aromatic extract (DAE) was developmentally toxic regardless of exposure duration as indicated by increased resorptions and decreased foetal body weights. Furthermore, when exposures were increased to 1000 mg/kg/day and given only during gestation days 10 through 12, cleft palate and ossification delays were observed. Cleft palate was considered to indicate a potential teratogenic effect of DAE.
The following Oil Industry Note (OIN) has been applied: OIN 8 - The classifications as a reproductive toxicant category 2; H361d (Suspected of damaging the unborn child) and specific target organ toxicant category 1; H372 (Causes damage to organs through prolonged or repeated exposure) need not apply if the substance is not classified as carcinogenic
Toxicokinetics of lubricant base oils has been examined in rodents. Absorption of other lubricant base oils across the small intestine is related to carbon chain length; hydrocarbons with smaller chain length are more readily absorbed than hydrocarbons with a longer chain length. The majority of an oral dose of mineral hydrocarbon is not absorbed and is excreted unchanged in the faeces. Distribution of mineral hydrocarbons following absorption has been observed in liver, fat, kidney, brain and spleen. Excretion of absorbed mineral hydrocarbons occurs via the faeces and urine. Based on the pharmacokinetic parameters and disposition profiles, the data indicate inherent strain differences in the total systemic exposure (~4 fold greater systemic dose in F344 vs SD rats), rate of metabolism, and hepatic and lymph node retention of C26H52, which may be associated with the different strain sensitivities to the formation of liver granulomas and MLN histiocytosis.
Highly and Severely Refined Distillate Base Oils
Acute toxicity: Multiple studies of the acute toxicity of highly & severely refined base oils have been reported. Irrespective of the crude source or the method or extent of processing, the oral LD50s have been observed to be >5 g/kg (bw) and the dermal LD50s have ranged from >2 to >5g/kg (bw). The LC50 for inhalation toxicity ranged from 2.18 mg/l to> 4 mg/l.
When tested for skin and eye irritation, the materials have been reported as “non-irritating” to “moderately irritating”
Testing in guinea pigs for sensitization has been negative
Repeat dose toxicity: . Several studies have been conducted with these oils**. T**he weight of evidence from all available data on highly & severely refined base oils support the presumption that a distillate base oil s toxicity is inversely related to the degree of processing it receives. Adverse effects have been reported with even the most severely refined white oils - these appear to depend on animal species and/ or the peculiarities of the study.

- The granulomatous lesions induced by the oral administration of white oils are essentially foreign body responses. The lesions occur only in rats, of which the Fischer 344 strain is particularly sensitive,
- The testicular effects seen in rabbits after dermal administration of a highly to severely refined base oil were unique to a single study and may have been related to stress induced by skin irritation, and
- The accumulation of foamy macrophages in the alveolar spaces of rats exposed repeatedly via inhalation to high levels of highly to severely refined base oils is not unique to these oils, but would be seen after exposure to many water insoluble materials.

Reproductive and developmental toxicity: A highly refined base oil was used as the vehicle control in a one-generation reproduction study. The study was conducted according to the OECD Test Guideline 421. There was no effect on fertility and mating indices in either males or females. At necropsy, there were no consistent findings and organ weights and histopathology were considered normal by the study s authors.
A single generation study in which a white mineral oil (a food/ drug grade severely refined base oil) was used as a vehicle control is reported. Two separate groups of pregnant rats were administered 5 ml/kg (bw)/day of the base oil via gavage, on days 6 through 19 of gestation. In one of the two base oil dose groups, three malformed foetuses were found among three litters The study authors considered these malformations to be minor and within the normal ranges for the strain of rat.
Genotoxicity:
In vitro (mutagenicity): Several studies have reported the results of testing different base oils for mutagenicity using a modified Ames assay Base oils with no or low concentrations of 3-7 ring PACs had low mutagenicity indices.
In vivo (chromosomal aberrations): A total of seven base stocks were tested in male and female Sprague-Dawley rats using a bone marrow cytogenetics assay. The test materials were administered via gavage at dose levels ranging from 500 to 5000 mg/kg (bw). Dosing occurred for either a single day or for five consecutive days. None of the base oils produced a significant increase in aberrant cells.
Carcinogenicity: Highly & severely refined base oils are not carcinogens, when given either orally or dermally.
NOTE: Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.

C.I. PIGMENT WHITE 6	Substance has been investigated as a mutagen, tumorigen and primary irritant.

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	For titanium dioxide: Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals. (General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) With regard to inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral ingestion of fine titanium dioxide showed particle size-dependent absorption by the gastrointestinal tract and large interindividual variations in blood levels of titanium dioxide. Studies on the application of sunscreens containing ultrafine titanium dioxide to healthy skin of human volunteers revealed that titanium dioxide particles only penetrate into the outermost layers of the stratum corneum, suggesting that healthy skin is an effective barrier to titanium dioxide. There are no studies on penetration of titanium dioxide in compromised skin. Respiratory effects that have been observed among groups of titanium dioxide-exposed workers include decline in lung function, pleural disease with plaques and pleural thickening, and mild fibrotic changes. However, the workers in these studies were also exposed to asbestos and/or silica. No data were available on genotoxic effects in titanium dioxide-exposed humans. Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain. Clearance of titanium dioxide is also affected by pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols. Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide are more slowly cleared than their fine counterparts. Titanium dioxide causes varying degrees of inflammation and associated pulmonary effects including lung epithelial cell injury, cholesterol granulomas and fibrosis. Rodents experience stronger pulmonary effects after exposure to ultrafine titanium dioxide particles compared with fine particles on a mass basis. These differences are related to lung burden in terms of particle surface area, and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium. Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the generation of reactive oxygen species by both particle types. This effect is stronger for ultrafine than for fine titanium oxide, and is markedly enhanced by exposure to simulated sunlight/ultraviolet light. **Animal carcinogenicity data** Pigmentary and ultrafine titanium dioxide were tested for carcinogenicity by oral administration in mice and rats, by inhalation in rats and female mice, by intratracheal administration in hamsters and female rats and mice, by subcutaneous injection in rats and by intraperitoneal administration in male mice and female rats. In one inhalation study, the incidence of benign and malignant lung tumours was increased in female rats. In another inhalation study, the incidences of lung adenomas were increased in the high-dose groups of male and female rats. Cystic keratinizing lesions that were diagnosed as squamous-cell carcinomas but re-evaluated as non-neoplastic pulmonary keratinizing cysts were also observed in the high-dose groups of female rats. Two inhalation studies in rats and one in female mice were negative. Intratracheally instilled female rats showed an increased incidence of both benign and malignant lung tumours following treatment with two types of titanium dioxide. Tumour incidence was not increased in intratracheally instilled hamsters and female mice. In-vivo studies have shown enhanced micronucleus formation in bone marrow and peripheral blood lymphocytes of intraperitoneally instilled mice. Increased Hprt mutations were seen in lung epithelial cells isolated from titanium dioxide-instilled rats. In another study, no enhanced oxidative DNA damage was observed in lung tissues of rats that were intratracheally instilled with titanium dioxide. The results of most in-vitro genotoxicity studies with titanium dioxide were negative.
HYDROCARBON RESIN, POSTPOLYMERISED WITH MALEIC ANHYDRIDE	Oral (-) LD50: 7000-10000 mg/kg Nil reported. [Manufacturer]
PENTAERYTHRITOL TETRABUTYLHYDROXYHYDROCINNAMATE	Non-sensitising in Guinea pig skin assay ** * CG ** Clariant For hindered phenols: Available data shows that acute toxicity of these substances is low. **Mutagenicity.** Data from bacterial reverse mutation assays and ***in vitro*** and ***in vivo*** chromosome aberration studies were reviewed. All assays, with and without metabolic activation, were negative. The weight of evidence for mutagenic potential for this category indicates these substances are not mutagenic. **In Vitro Chromosome Aberration Studies.** In vitro chromosome aberration studies are available for several members All except 2,6-di-tert-butyl-p-cresol were negative **In Vivo Chromosome Aberration Studies.** In vivo studies evaluating chromosome damage are available for six of the hindered phenols. All in vivo evaluations were negative. **Repeated Dose Toxicity.** Repeated dose toxicity data of approximately three months (90-day, 12- and 13-week) are available for some of the substances in this group. The liver was the target organ in rats for almost all of the substances with subchronic toxicity data in that species. Other target organs included thyroid and kidney and mesenteric lymph nodes. NOAELs in rats ranged from 100 ppm (approximately 5 mg/kg/day) to 10,000 ppm (500 mg/kg/day **Carcinogenicity:** Data is available for 2,6-di-tert-butyl-p-cresol (128-37-0); and 4,4'-thiobis-6-(t-butyl-m-cresol) (96-69-5). Liver adenomas were reported for 2,6-di-tert-butyl-p-cresol (128-37-0) and a NOAEL was established for the study at 25 mg/kg/day. 4,4'-Thiobis-6-(t-butyl-m-cresol) (96-69-5) was not carcinogenic in rats or mice, but the kidney was identified as a target organ in female rats
TRIS(2,4-DI-TERT-BUTYLPHENYL)PHOSPHITE	Sensitization: (Guinea Pig) No sensitization observed. Teratogenicity: (Rabbits) No evidence of Teratogenicity. Mutagenicity: AMES Test: Negative Chromosome study spermatogonia/spermatocytes (Mouse): No evidence of mutagenicity. Sister chromatid exchange study (Chinese Hamster): Negative. Saccharomyces cerevisiae MP-1: Negative. Chromosome study in somatic cells (Chinese Hamster): Negative. Neurotoxicity: White leghorn strain: No signs of neurotoxicity. The Hansen Group MSDS * Ciba ** Cytec
STYRENE/ BUTADIENE RUBBER & CALCIUM CARBONATE & SOYBEAN OIL, EPOXIDISED	The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.
NATURAL RUBBER & N-CYCLOHEXYL-2-BENZOTHIAZOLESULFENAMIDE	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.

Continued...

N-CYCLOHEXYL-2-BENZOTHIAZOLESULFENAMIDE & NAPHTHENIC DISTILLATE, HEAVY, HYDROTREATED (SEVERE) & C.I. PIGMENT WHITE 6	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.
NAPHTHENIC DISTILLATE, HEAVY, HYDROTREATED (SEVERE) & ISOBUTYLENE HOMOPOLYMER & STYRENE/ ISOPRENE COPOLYMER	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

FORSPEC PRESWELL

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

sodium polyacrylate

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

sodium carboxymethylcellulose

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	46.04-165.37mg/l	4
EC50(ECx)	48h	Crustacea	46.04-165.37mg/l	4
LC50	96h	Fish	>20000mg/L	4

styrene/ butadiene rubber

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

natural rubber

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

calcium carbonate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>14mg/l	2
NOEC(ECx)	1h	Fish	4-320mg/l	4
LC50	96h	Fish	>165200mg/L	4

soybean oil, epoxidised

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	8mg/l	1
NOEC(ECx)	72h	Algae or other aquatic plants	0.7mg/l	1

N-cyclohexyl-2-benzothiazolesulfenamide

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	0.15mg/l	2
EC50	48h	Crustacea	18mg/l	Not Available
EC50	96h	Algae or other aquatic plants	0.9mg/l	1
EC50(ECx)	96h	Algae or other aquatic plants	0.9mg/l	1
LC50	96h	Fish	>1000mg/l	Not Available

sulfur

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	>0.005mg/L	2
NOEC(ECx)	504h	Crustacea	>0.003mg/L	2
LC50	96h	Fish	>207mg/L	4

Continued...

naphthenic distillate, heavy, hydrotreated (severe)	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	48h	Crustacea	>1000mg/l	1
	EC50	96h	Algae or other aquatic plants	>1000mg/l	1
	NOEC(ECx)	504h	Crustacea	>1mg/l	1
	ErC50	72h	Algae or other aquatic plants	>1000mg/l	1
isobutylene homopolymer	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	LC50	96h	Fish	>5600mg/L	4
C.I. Pigment White 6	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	BCF	1008h	Fish	<1.1-9.6	7
	EC50	72h	Algae or other aquatic plants	3.75-7.58mg/l	4
	EC50	48h	Crustacea	1.9mg/l	2
	NOEC(ECx)	672h	Fish	>=0.004mg/L	2
	EC50	96h	Algae or other aquatic plants	179.05mg/l	2
	LC50	96h	Fish	1.85-3.06mg/l	4
styrene/ isoprene copolymer	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available
hydrocarbon resin, postpolymerised with maleic anhydride	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	<1mg/l	2
hydrocarbons, C6-20, hydrogenated, polymers	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available
pentaerythritol tetrabutylhydroxyhydrocinnamate	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	BCF	1008h	Fish	<0.2	7
	EC50	72h	Algae or other aquatic plants	>100mg/l	1
	EC50(ECx)	24h	Crustacea	>86mg/l	1
	LC50	96h	Fish	>100mg/l	2
tris(2,4-di-tert-butylphenyl)phosphite	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	>75.2mg/l	1
	NOEC(ECx)	72h	Algae or other aquatic plants	75.2mg/l	1

Legend: ***Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. US EPA, Ecotox database - Aquatic Toxicity Data 4. ECETOC Aquatic Hazard Assessment Data 5. NITE (Japan) - Bioconcentration Data 6. METI (Japan) - Bioconcentration Data 7. Vendor Data***

Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
Toxic to soil organisms.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
N-cyclohexyl-2-benzothiazolesulfenamide	HIGH	HIGH
sulfur	LOW	LOW
isobutylene homopolymer	LOW	LOW
C.I. Pigment White 6	HIGH	HIGH
pentaerythritol tetrabutylhydroxyhydrocinnamate	HIGH	HIGH
tris(2,4-di-tert-butylphenyl)phosphite	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
soybean oil, epoxidised	LOW (LogKOW = 14.84)
N-cyclohexyl-2-benzothiazolesulfenamide	LOW (LogKOW = 3.47)
sulfur	LOW (LogKOW = -1.38)

Continued...

Ingredient	Bioaccumulation
isobutylene homopolymer	LOW (LogKOW = 17.14)
C.I. Pigment White 6	LOW (BCF = 10)
pentaerythritol tetrabutylhydroxyhydrocinnamate	LOW (BCF = 2.3)
tris(2,4-di-tert-butylphenyl)phosphite	LOW (BCF = 4.66)

Mobility in soil

Ingredient	Mobility
N-cyclohexyl-2-benzothiazolesulfenamide	LOW (Log KOC = 18890)
sulfur	LOW (Log KOC = 14.3)
isobutylene homopolymer	LOW (Log KOC = 35.04)
C.I. Pigment White 6	LOW (Log KOC = 23.74)
tris(2,4-di-tert-butylphenyl)phosphite	LOW (Log KOC = 10000000000)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information

Labels Required

Marine Pollutant	
HAZCHEM	Not Applicable

Land transport (ADG): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Air transport (ICAO-IATA / DGR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Sea transport (IMDG-Code / GGVSee): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

14.7. Maritime transport in bulk according to IMO instruments

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

Not Applicable

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

Product name	Group
sodium polyacrylate	Not Applicable
sodium carboxymethylcellulose	Not Applicable
styrene/ butadiene rubber	Not Applicable
natural rubber	Not Applicable
calcium carbonate	Not Applicable
soybean oil, epoxidised	Not Applicable

Continued...

Product name	Group
N-cyclohexyl-2-benzothiazolesulfenamide	Not Applicable
sulfur	Not Applicable
naphthenic distillate, heavy, hydrotreated (severe)	Not Applicable
isobutylene homopolymer	Not Applicable
C.I. Pigment White 6	Not Applicable
styrene/ isoprene copolymer	Not Applicable
hydrocarbon resin, postpolymerised with maleic anhydride	Not Applicable
hydrocarbons, C6-20, hydrogenated, polymers	Not Applicable
pentaerythritol tetrabutylhydroxyhydrocinnamate	Not Applicable
tris(2,4-di-tert-butylphenyl)phosphite	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
sodium polyacrylate	Not Applicable
sodium carboxymethylcellulose	Not Applicable
styrene/ butadiene rubber	Not Applicable
natural rubber	Not Applicable
calcium carbonate	Not Applicable
soybean oil, epoxidised	Not Applicable
N-cyclohexyl-2-benzothiazolesulfenamide	Not Applicable
sulfur	Not Applicable
naphthenic distillate, heavy, hydrotreated (severe)	Not Applicable
isobutylene homopolymer	Not Applicable
C.I. Pigment White 6	Not Applicable
styrene/ isoprene copolymer	Not Applicable
hydrocarbon resin, postpolymerised with maleic anhydride	Not Applicable
hydrocarbons, C6-20, hydrogenated, polymers	Not Applicable
pentaerythritol tetrabutylhydroxyhydrocinnamate	Not Applicable
tris(2,4-di-tert-butylphenyl)phosphite	Not Applicable

SECTION 15 Regulatory information

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

sodium polyacrylate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

sodium carboxymethylcellulose is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

styrene/ butadiene rubber is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

natural rubber is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

calcium carbonate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

soybean oil, epoxidised is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

N-cyclohexyl-2-benzothiazolesulfenamide is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Continued...

Australian Inventory of Industrial Chemicals (AIIC)

sulfur is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

naphthenic distillate, heavy, hydrotreated (severe) is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List

isobutylene homopolymer is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

C.I. Pigment White 6 is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

styrene/ isoprene copolymer is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

hydrocarbon resin, postpolymerised with maleic anhydride is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

hydrocarbons, C6-20, hydrogenated, polymers is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

pentaerythritol tetrabutylhydroxyhydrocinnamate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

tris(2,4-di-tert-butylphenyl)phosphite is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (sodium polyacrylate; sodium carboxymethylcellulose; styrene/ butadiene rubber; natural rubber; soybean oil, epoxidised; N-cyclohexyl-2-benzothiazolesulfenamide; sulfur; naphthenic distillate, heavy, hydrotreated (severe); isobutylene homopolymer; C.I. Pigment White 6; styrene/ isoprene copolymer; hydrocarbon resin, postpolymerised with maleic anhydride; pentaerythritol tetrabutylhydroxyhydrocinnamate; tris(2,4-di-tert-butylphenyl)phosphite)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (sodium polyacrylate; sodium carboxymethylcellulose; styrene/ butadiene rubber; styrene/ isoprene copolymer; hydrocarbons, C6-20, hydrogenated, polymers)
Japan - ENCS	No (natural rubber; soybean oil, epoxidised; sulfur)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	TSCA Inventory 'Active' substance(s) (sodium polyacrylate; sodium carboxymethylcellulose; styrene/ butadiene rubber; calcium carbonate; soybean oil, epoxidised; N-cyclohexyl-2-benzothiazolesulfenamide; sulfur; naphthenic distillate, heavy, hydrotreated (severe); isobutylene homopolymer; C.I. Pigment White 6; styrene/ isoprene copolymer; hydrocarbon resin, postpolymerised with maleic anhydride; hydrocarbons, C6-20, hydrogenated, polymers; pentaerythritol tetrabutylhydroxyhydrocinnamate; tris(2,4-di-tert-butylphenyl)phosphite); No (natural rubber)
Taiwan - TCSI	Yes
Mexico - INSQ	No (natural rubber; styrene/ isoprene copolymer; hydrocarbons, C6-20, hydrogenated, polymers)
Vietnam - NCI	Yes
Russia - FBEPH	No (hydrocarbons, C6-20, hydrogenated, polymers)
UAE - Control List (Banned/Restricted Substances)	No (sodium polyacrylate; sodium carboxymethylcellulose; styrene/ butadiene rubber; natural rubber; calcium carbonate; soybean oil, epoxidised; N-cyclohexyl-2-benzothiazolesulfenamide; naphthenic distillate, heavy, hydrotreated (severe); isobutylene homopolymer; C.I. Pigment White 6; styrene/ isoprene copolymer; hydrocarbon resin, postpolymerised with maleic anhydride; hydrocarbons, C6-20, hydrogenated, polymers; pentaerythritol tetrabutylhydroxyhydrocinnamate; tris(2,4-di-tert-butylphenyl)phosphite)
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	12/03/2026
Initial Date	12/03/2026

Continued...

SDS Version Summary

Version	Date of Update	Sections Updated
2.1	12/03/2026	Physical and chemical properties - Appearance, Ecological Information - Environmental, Exposure controls / personal protection - Exposure Standard, Composition / information on ingredients - Ingredients, Handling and storage - Storage (suitable container), Toxicological information - Toxicity and Irritation (Other), Identification of the substance / mixture and of the company / undertaking - Use

Other information

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

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

Definitions and abbreviations

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

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

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