

Multimin Chrome Injection for Cattle

Virbac (Australia) Pty Limited

Chemwatch: 5433-63

Version No: 9.1

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

Chemwatch Hazard Alert Code: 2

Issue Date: 12/10/2021

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L.GHS.AUS.EN.E

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

Product Identifier

Product name	Multimin Chrome Injection for Cattle
Chemical Name	Not Applicable
Synonyms	Not Available
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	An injectable formulation as a chelated trace mineral injection for beef and dairy cattle deficient in and/or responsive to manganese, zinc, selenium, chromium, and/or copper supplementation.
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Details of the manufacturer or supplier of the safety data sheet

Registered company name	Virbac (Australia) Pty Limited
Address	361 Horsley Road Milperra NSW 2214 Australia
Telephone	1800 242 100
Fax	+61 2 9772 9773
Website	au.virbac.com
Email	customercare@virbac.com.au

Emergency telephone number

Association / Organisation	Poisons Information Centre
Emergency telephone numbers	13 11 26
Other emergency telephone numbers	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	0	
Toxicity	2	
Body Contact	2	
Reactivity	1	
Chronic	2	

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

Poisons Schedule	S6
Classification [1]	Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 2A, Hazardous to the Aquatic Environment Long-Term Hazard Category 3
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	Warning

Hazard statement(s)

H302	Harmful if swallowed.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.

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H319	Causes serious eye irritation.
H412	Harmful to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing mist/vapours/spray.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P302+P352	IF ON SKIN: Wash with plenty of water.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.
P330	Rinse mouth.

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
14025-21-9	10-30	<u>EDTA disodium zinc salt</u>
39208-15-6	10-30	<u>EDTA copper disodium salt</u>
6381-92-6	1-10	<u>EDTA disodium salt</u>
15375-84-5	1-10	<u>EDTA, disodium manganese salt</u>
10025-73-7	<1	<u>chromic chloride</u>
10102-18-8	<1	<u>sodium selenite</u>
100-51-6	<1	<u>Benzenemethanol</u>
7732-18-5	balance	<u>Dwater</u>

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

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none">▶ 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	<p>If skin contact occurs:</p> <ul style="list-style-type: none">▶ Immediately remove all contaminated clothing, including footwear.▶ Flush skin and hair with running water (and soap if available).▶ Seek medical attention in event of irritation. <p>Accidental self injection may lead to an inflammatory response and medical advice should be sought on the management of deep injections, particularly those near a joint or at a point associated with bruising. If possible the application of gentle squeezing pressure with absorbent material (e.g. facial tissues) at the injection site will swab up unabsorbed product. CAUTION: Strong squeezing of the site should be avoided. The damaged area should be thoroughly cleansed and a topical antiseptic applied. Check your tetanus immunisation status. Consider surgical flushing if acute pain and inflammation within 24 hours.</p>
Inhalation	<ul style="list-style-type: none">▶ If fumes or combustion products are inhaled remove from contaminated area.▶ Lay patient down. Keep warm and rested.▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.▶ Transport to hospital, or doctor.
Ingestion	<ul style="list-style-type: none">▶ IF SWALLOWED, REFER FOR MEDICAL ATTENTION, WHERE POSSIBLE, WITHOUT DELAY.▶ For advice, contact a Poisons Information Centre or a doctor.▶ Urgent hospital treatment is likely to be needed.

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- ▶ In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition.
- ▶ If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS should be provided. Further action will be the responsibility of the medical specialist.
- ▶ If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS.

Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:

- ▶ **INDUCE** vomiting with fingers down the back of the throat, **ONLY IF CONSCIOUS**. Lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.

NOTE: Wear a protective glove when inducing vomiting by mechanical means.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.
for copper intoxication:

- ▶ Unless extensive vomiting has occurred empty the stomach by lavage with water, milk, sodium bicarbonate solution or a 0.1% solution of potassium ferrocyanide (the resulting copper ferrocyanide is insoluble).
- ▶ Administer egg white and other demulcents.
- ▶ Maintain electrolyte and fluid balances.
- ▶ Morphine or meperidine (Demerol) may be necessary for control of pain.
- ▶ If symptoms persist or intensify (especially circulatory collapse or cerebral disturbances, try BAL intramuscularly or penicillamine in accordance with the supplier's recommendations.
- ▶ Treat shock vigorously with blood transfusions and perhaps vasopressor amines.
- ▶ If intravascular haemolysis becomes evident protect the kidneys by maintaining a diuresis with mannitol and perhaps by alkalising the urine with sodium bicarbonate.
- ▶ It is unlikely that methylene blue would be effective against the occasional methaemoglobinemia and it might exacerbate the subsequent haemolytic episode.
- ▶ Institute measures for impending renal and hepatic failure.

[GOSSELIN, SMITH & HODGE: Commercial Toxicology of Commercial Products]

- ▶ A role for activated charcoals for emesis is, as yet, unproven.
- ▶ In severe poisoning CaNa2EDTA has been proposed.

[ELLENHORN & BARCELOUX: Medical Toxicology]

SECTION 5 Firefighting measures

Extinguishing media

- ▶ There is no restriction on the type of extinguisher which may be used.
- ▶ Use extinguishing media suitable for surrounding area.

Special hazards arising from the substrate or mixture

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

Fire Fighting	<ul style="list-style-type: none">▶ Alert Fire Brigade and tell them location and nature of hazard.▶ Wear breathing apparatus plus protective gloves in the event of a fire.▶ Prevent, by any means available, spillage from entering drains or water courses.▶ Use fire fighting procedures suitable for surrounding area.▶ DO NOT approach containers suspected to be hot.▶ Cool fire exposed containers with water spray from a protected location.▶ If safe to do so, remove containers from path of fire.▶ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	<ul style="list-style-type: none">▶ Non combustible.▶ Not considered a significant fire risk, however containers may burn. Decomposes on heating and produces: carbon dioxide (CO2) nitrogen oxides (NOx) metal oxides other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	<ul style="list-style-type: none">▶ Clean up all spills immediately.▶ Avoid breathing vapours and contact with skin and eyes.▶ 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	Moderate hazard. <ul style="list-style-type: none">▶ Clear area of personnel and move upwind.▶ Alert Fire Brigade and tell them location and nature of hazard.▶ Wear breathing apparatus plus protective gloves.▶ Prevent, by any means available, spillage from entering drains or water course.▶ Stop leak if safe to do so.▶ Contain spill with sand, earth or vermiculite.

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- ▶ Collect recoverable product into labelled containers for recycling.
- ▶ Neutralise/decontaminate residue (see Section 13 for specific agent).
- ▶ 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	<ul style="list-style-type: none">▶ DO NOT allow clothing wet with material to stay in contact with skin▶ Avoid all personal contact, including inhalation.▶ Wear protective clothing when risk of exposure occurs.▶ Use in a well-ventilated area.▶ Avoid contact with moisture.▶ 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. Launder contaminated clothing before re-use.▶ Use good occupational work practice.▶ Observe manufacturer's storage and handling recommendations contained within this SDS.▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	<ul style="list-style-type: none">▶ Store in original containers.▶ Keep containers securely sealed.▶ Store in a cool, dry, well-ventilated area.▶ Store away from incompatible materials and foodstuff containers.▶ Protect containers against physical damage and check regularly for leaks.▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none">▶ Polyethylene or polypropylene container.▶ Packing as recommended by manufacturer.▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<ul style="list-style-type: none">▶ Avoid reaction with oxidising agents▶ Avoid strong acids, bases.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	EDTA, disodium manganese salt	Manganese, dust & compounds (as Mn)	1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	chromic chloride	Chromium (III) compounds (as Cr)	0.5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	sodium selenite	Selenium compounds (as Se) excluding hydrogen selenide	0.1 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
EDTA disodium salt	11 mg/m3	120 mg/m3	730 mg/m3
EDTA disodium salt	30 mg/m3	330 mg/m3	2,000 mg/m3
chromic chloride	4.6 mg/m3	15 mg/m3	87 mg/m3
chromic chloride	7.7 mg/m3	43 mg/m3	260 mg/m3
sodium selenite	1.3 mg/m3	2.3 mg/m3	3.1 mg/m3
Benzenemethanol	30 ppm	52 ppm	740 ppm

Ingredient	Original IDLH	Revised IDLH
EDTA disodium zinc salt	Not Available	Not Available
EDTA copper disodium salt	Not Available	Not Available
EDTA disodium salt	Not Available	Not Available
EDTA, disodium manganese salt	500 mg/m3	Not Available
chromic chloride	25 mg/m3	Not Available
sodium selenite	1 mg/m3	Not Available
Benzenemethanol	Not Available	Not Available
Dwater	Not Available	Not Available


Occupational Exposure Banding

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Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
EDTA disodium zinc salt	E	≤ 0.01 mg/m³
EDTA copper disodium salt	E	≤ 0.01 mg/m³
EDTA disodium salt	E	≤ 0.01 mg/m³
Benzenemethanol	E	≤ 0.1 ppm
Notes:	Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.	

MATERIAL DATA

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.	
	Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. An approved self contained breathing apparatus (SCBA) may be required in some situations. Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.	
	Type of Contaminant:	Air Speed:
	solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
	aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
	grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)
	Within each range the appropriate value depends on:	
	Lower end of the range	Upper end of the range
	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity	
3: Intermittent, low production.	3: High production, heavy use	
4: Large hood or large air mass in motion	4: Small hood-local control only	
Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.		
Individual protection measures, such as personal protective equipment		
Eye and face protection	<ul style="list-style-type: none">▶ Safety glasses with side shields.▶ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent]▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].	
Skin protection	See Hand protection below	
Hands/feet protection	<ul style="list-style-type: none">▶ Wear chemical protective gloves, e.g. PVC.▶ Wear safety footwear or safety gumboots, e.g. Rubber <p>NOTE:</p> <ul style="list-style-type: none">▶ 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. <p>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.</p> <p>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.</p> <p>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.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none">· frequency and duration of contact,· chemical resistance of glove material,· glove thickness and· dexterity	

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	<p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none">· 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. <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none">· Excellent when breakthrough time > 480 min· Good when breakthrough time > 20 min· Fair when breakthrough time < 20 min· Poor when glove material degrades <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.</p> <p>Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none">· Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.· Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p>
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none">▶ Overalls.▶ P.V.C apron.▶ Barrier cream.▶ Skin cleansing cream.▶ Eye wash unit.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
"Forsberg Clothing Performance Index".
The effect(s) of the following substance(s) are taken into account in the *computer-generated* selection:
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Material	CPI
BUTYL	A
VITON	A
NATURAL RUBBER	C
NEOPRENE	C
PVA	C

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

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

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

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

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

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Clear dark blue liquid; mixes with water.		
Physical state	Liquid	Relative density (Water = 1)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Applicable
pH (as supplied)	6.5-7.5	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Applicable	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available

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Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Applicable	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	<p>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.</p> <p>Copper poisoning following exposure to copper dusts and fume may result in headache, cold sweat and weak pulse. Capillary, kidney, liver and brain damage are the longer term manifestations of such poisoning. Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in "metal fume fever". Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure.</p>
Ingestion	Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.
Skin Contact	<p>Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>
Eye	<p>Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p>
Chronic	<p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.</p> <p>Substances that can cause occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers. Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.</p> <p>Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>Chromium(III) is considered an essential trace nutrient serving as a component of the "glucose tolerance factor" and a cofactor for insulin action. High concentrations of chromium are also found in RNA. Trivalent chromium is the most common form found in nature.</p> <p>Chronic inhalation of trivalent chromium compounds produces irritation of the bronchus and lungs, dystrophic changes to the liver and kidney, pulmonary oedema, and adverse effects on macrophages. Intratracheal administration of chromium(III) oxide, in rats, increased the incidence of sarcomas, and tumors and reticulum cell sarcomas of the lung. There is inadequate evidence of carcinogenicity of chromium(III) compounds in experimental animals and humans (IARC).</p> <p>Chronic exposure to hexavalent chromium compounds reportedly produces skin, eye and respiratory tract irritation, yellowing of the eyes and skin, allergic skin and respiratory reactions, diminished sense of smell and taste, blood disorders, liver and kidney damage, digestive disorders and lung damage. There is sufficient evidence of carcinogenicity of chromium(VI) compounds in experimental animals and humans to confirm these as Class 1 carcinogens (IARC).</p> <p>Exposure to chromium during chrome production and in the chrome pigment industry is associated with cancer of the respiratory tract. A slight increase in gastrointestinal cancer following exposure to chromium compounds has also been reported. The greatest risk is attributed to exposure to acid-soluble, water-insoluble hexavalent chromium which occurs in roasting and refining processes. Animal studies support the idea that the most potent carcinogenic compounds are the slightly soluble hexavalent compounds. The cells are more active in the</p>

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uptake of the hexavalent forms compared to trivalent forms and this may explain the difference in occupational effect. It is the trivalent form, however, which is metabolically active and binds with nucleic acid within the cell suggesting that chromium mutagenesis first requires biotransformation of the hexavalent form by reduction.

Hexavalent chromes produce chronic ulceration of skin surfaces (quite independent of other hypersensitivity reactions exhibited by the skin). Water-soluble chromium(VI) compounds come close to the top of any published "hit list" of contact allergens (eczematogens) producing positive results in 4 to 10% of tested individuals. On the other hand only chromium(III) compounds can bind to high molecular weight carriers such as proteins to form a complete allergen (such as a hapten). Chromium(VI) compounds cannot. It is assumed that reduction must take place for such compounds to manifest any contact sensitivity. The apparent contradiction that chromium(VI) salts cause allergies to chromium(III) compounds but that allergy to chromium(III) compounds is difficult to demonstrate is accounted for by the different solubilities and skin penetration of these compounds. Water-soluble chromium(VI) salts penetrate the horny layer of the skin more readily than chromium(III) compounds which are bound by cross-linking in the horny layer ("tanning", as for leather) and therefore do not reach the cells involved in antigen processing.

Sequestering agents are occasionally used in therapies for various forms of poisoning and are normally injected intravenously. A systemic reaction known as the "excessive chelation syndrome" consists mainly of malaise, fatigue, thirst and parathesias, followed by chills and fever. Myalgia, headache, anorexia, nausea and occasionally marked urinary frequency and urgency may also occur. Minor reactions resembling those produced by histamine or the common cold are sometimes seen.

Parenteral administration of EDTA and its salts in high doses may produce severe renal lesions with tubular necrosis, internal haemorrhage, transient bone marrow depression and life-threatening hypocalcaemia. Prolonged parenteral exposures produce electrolyte imbalance and possible cardiac arrhythmias.

Prolonged or repeated skin contact may result in irritation. EDTA and its metal salts do not permeate the cellular membrane to a significant extent; they remain in the extracellular fluid until excreted. (ILO Encyclopaedia)

NOTE: Conflicting animal test data is available with regard to the teratogenic potential of EDTA sodium salts. Some data indicate that teratogenic effects may occur at extremely high maternal doses.

For copper and its compounds (typically copper chloride):

Acute toxicity: There are no reliable acute oral toxicity results available. Animal testing shows that skin in exposure to copper may lead to hardness of the skin, scar formation, exudation and reddish changes. Inflammation, irritation and injury of the skin were noted.

Repeat dose toxicity: Animal testing shows that very high levels of copper monochloride may cause anaemia.

Genetic toxicity: Copper monochloride does not appear to cause mutations in vivo, although chromosomal aberrations were seen at very high concentrations in vitro.

Cancer-causing potential: There was insufficient information to evaluate the cancer-causing activity of copper monochloride.

Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant 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.

Multimin Chrome Injection for Cattle	TOXICITY	IRRITATION
	Not Available	Not Available
EDTA disodium zinc salt	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation (Rat) LC50: >2.75 mg/l4h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
EDTA copper disodium salt	TOXICITY	IRRITATION
	Oral (Rat) LD50: 1750 mg/kg ^[2]	Not Available
EDTA disodium salt	TOXICITY	IRRITATION
	Oral (Rat) LD50: 2000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
EDTA, disodium manganese salt	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation (Rat) LC50: >5.16 mg/l4h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
chromic chloride	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
sodium selenite	TOXICITY	IRRITATION
	Inhalation (Rat) LC50: >0.052<=0.51 mg/l4h ^[1]	Not Available
Benzenemethanol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 2000 mg/kg ^[2]	Eye (rabbit): 0.75 mg open SEVERE
	Inhalation (Rat) LC50: >4.178 mg/l4h ^[2]	Eye: adverse effect observed (irritating) ^[1]
	Oral (Rat) LD50: 1230 mg/kg ^[2]	Skin (man): 16 mg/48h-mild
		Skin (rabbit):10 mg/24h open-mild
Dwater	TOXICITY	IRRITATION
	Oral (Rat) LD50: >90000 mg/kg ^[2]	Not Available

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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	
CHROMIC CHLORIDE	<p>for hexahydrate: for anhydrous form: Human cell mutagen Paternal effect, effects on fertility, effects on embryo (extra embryonic structures, foetotoxicity), specific developmental abnormalities (central nervous system, eye, ear) recorded.</p> <p>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).</p> <p>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.</p> <p>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.</p> <p>For chrome(III) and other valence states (except hexavalent):</p> <p>For inhalation exposure, all trivalent and other chromium compounds are treated as particulates, not gases.</p> <p>The mechanisms of chromium toxicity are very complex, and although many studies on chromium are available, there is a great deal of uncertainty about how chromium exerts its toxic influence. Much more is known about the mechanisms of hexavalent chromium toxicity than trivalent chromium toxicity. There is an abundance of information available on the carcinogenic potential of chromium compounds and on the genotoxicity and mutagenicity of chromium compounds in experimental systems. The consensus from various reviews and agencies is that evidence of carcinogenicity of elemental, divalent, or trivalent chromium compounds is lacking. Epidemiological studies of workers in a number of industries (chromate production, chromate pigment production and use, and chrome plating) conclude that while occupational exposure to hexavalent chromium compounds is associated with an increased risk of respiratory system cancers (primarily bronchogenic and nasal), results from occupational exposure studies to mixtures that were mainly elemental and trivalent (ferrochromium alloy worker) were inconclusive. Studies in leather tanners, who were exposed to trivalent chromium were consistently negative. In addition to the lack of direct evidence of carcinogenicity of trivalent or elemental chromium and its compounds, the genotoxic evidence is overwhelmingly negative. The lesser potency of trivalent chromium relative to hexavalent chromium is likely related to the higher redox potential of hexavalent chromium and its greater ability to enter cells. enter cells</p> <p>The general inability of trivalent chromium to traverse membranes and thus be absorbed or reach peripheral tissue in significant amounts is generally accepted as a probable explanation for the overall absence of systemic trivalent chromium toxicity. Elemental and divalent forms of chromium are not able to traverse membranes readily either. This is not to say that elemental, divalent, or trivalent chromium compounds cannot traverse membranes and reach peripheral tissue, the mechanism of absorption is simply less efficient in comparison to absorption of hexavalent chromium compounds. Hexavalent chromium compounds exist as tetrahedral chromate anions, resembling the forms of other natural anions like sulfate and phosphate which are permeable across nonselective membranes. Trivalent chromium forms octahedral complexes which cannot easily enter though these channels, instead being absorbed via passive diffusion and phagocytosis. Although trivalent chromium is less well absorbed than hexavalent chromium, workers exposed to trivalent compounds have had detectable levels of chromium in the urine at the end of a workday. Absorbed chromium is widely distributed throughout the body via the bloodstream, and can reach the foetus. Although there is ample in vivo evidence that hexavalent chromium is efficiently reduced to trivalent chromium in the gastrointestinal tract and can be reduced to the trivalent form by ascorbate and glutathione in the lungs, there is no evidence that trivalent chromium is converted to hexavalent chromium in biological systems. In general, trivalent chromium compounds are cleared rapidly from the blood and more slowly from the tissues. Although not fully characterized, the biologically active trivalent chromium molecule appears to be chromodulin, also referred to as (GTF). Chromodulin is an oligopeptide complex containing four chromic ions. Chromodulin may facilitate interactions of insulin with its receptor site, influencing protein, glucose, and lipid metabolism. Inorganic trivalent chromium compounds, which do not appear to have insulin-potentiating properties, are capable of being converted into biologically active forms by humans and animals</p> <p>Chromium can be a potent sensitiser in a small minority of humans, both from dermal and inhalation exposures.</p> <p>The most sensitive endpoint identified in animal studies of acute exposure to trivalent chromium appears to involve the respiratory system. Specifically, acute exposure to trivalent chromium is associated with impaired lung function and lung damage.</p> <p>Based on what is known about absorption of chromium in the human body, its potential mechanism of action in cells, and occupational data indicating that valence states other than hexavalent exhibit a relative lack of toxicity the toxicity of elemental and divalent chromium compounds is expected to be similar to or less than common trivalent forms.</p> <p>Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).</p> <p>The substance is classified by IARC as Group 3:</p> <p>NOT classifiable as to its carcinogenicity to humans.</p> <p>Evidence of carcinogenicity may be inadequate or limited in animal testing.</p>
SODIUM SELENITE	<p>Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.</p>
BENZENEMETHANOL	<p>Adverse reactions to fragrances in perfumes and in fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, photosensitivity, immediate contact reactions (contact urticaria), and pigmented contact dermatitis. Airborne and connubial contact dermatitis occur.</p> <p>Intolerance to perfumes, by inhalation, may occur if the perfume contains a sensitising principal. Symptoms may vary from general illness, coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea, acute respiratory illness, hayfever, and other respiratory diseases (including asthma). Perfumes can induce hyper-reactivity of the respiratory tract without producing an IgE-mediated allergy or demonstrable respiratory obstruction. This was shown by placebo-controlled challenges of nine patients to "perfume mix". The same patients were also subject to perfume provocation, with or without a carbon filter mask, to ascertain whether breathing through a filter with active carbon would prevent symptoms. The patients breathed through the mouth, during the provocations, as a nose clamp was used to prevent nasal inhalation. The patient's earlier symptoms were verified; breathing through the carbon filter had no protective effect. The symptoms were not transmitted via the olfactory nerve but they may have been induced by trigeminal reflex via the respiratory tract or by the eyes. Cases of occupational asthma induced by perfume substances such as isoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to give persistent symptoms even though the exposure is below occupational exposure limits.</p> <p>Inhalation intolerance has also been produced in animals. The emissions of five fragrance products, for one hour, produced various combinations of sensory irritation, pulmonary irritation, decreases in expiratory airflow velocity as well as alterations of the functional observational battery indicative of neurotoxicity in mice. Neurotoxicity was found to be more severe after mice were repeatedly exposed to the fragrance products, being four brands of cologne and one brand of toilet water.</p> <p>Contact allergy to fragrances is relatively common, affecting 1 to 3% of the general population, based on limited testing with eight common fragrance allergens and about 16 % of patients patch tested for suspected allergic contact dermatitis.</p> <p>Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a sufficient degree of fragrance contact allergens. Contact allergy is a life-long, specifically altered reactivity in the immune system. This means that once contact allergy is developed, cells in the immune system will be present which can recognise and react towards the allergen. As a consequence, symptoms, i.e. allergic contact dermatitis, may occur upon re-exposure to the fragrance allergen(s) in question. Allergic contact dermatitis is an inflammatory skin disease characterised by erythema, swelling and vesicles in the acute phase. If exposure continues it may develop into a chronic condition with scaling and painful fissures of the skin. Allergic contact dermatitis to fragrance ingredients is most often caused by cosmetic products and usually involves the face and/or hands. It may affect fitness for work and the quality of life of the individual. Fragrance contact allergy has long been recognised as a frequent and potentially disabling problem. Prevention is possible as it is an environmental disease and if the environment is modified (e.g. by reduced use concentrations of allergens), the disease frequency and severity will decrease Fragrance contact allergy is mostly non-occupational and related to the personal use of cosmetic products. Allergic contact dermatitis can be severe and widespread, with a significant impairment of quality of life and potential consequences for fitness for work.</p>

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Thus, prevention of contact sensitisation to fragrances, both in terms of primary prevention (avoiding sensitisation) and secondary prevention (avoiding relapses of allergic contact dermatitis in those already sensitised), is an important objective of public health risk management measure.

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

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

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

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

Pigmentary anomalies: The term "pigmented cosmetic dermatitis" was introduced in 1973 for what had previously been known as melanosis faciei feminae when the mechanism (type IV allergy) and causative allergens were clarified. It refers to increased pigmentation, usually on the face/neck, often following sub-clinical contact dermatitis. Many cosmetic ingredients were patch tested at non-irritant concentrations and statistical evaluation showed that a number of fragrance ingredients were associated: jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol, geranium oil.

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

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

Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A prohaptens is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems. A prohaptens is a chemical that itself is non- or low-sensitising but that is transformed into a hapten in the skin (bioactivation) usually via enzyme catalysis. It is not always possible to know whether a particular allergen that is not directly reactive acts as a prohaptens or as a hapten, or both, because air oxidation and bioactivation can often give the same product (geraniol is an example). Some chemicals might act by all three pathways.

Prohaptens

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

In the case of prohaptens, the possibility to become activated is inherent to the molecule and activation cannot be avoided by extrinsic measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Crossreactivity has been shown for certain alcohols and their corresponding aldehydes, i.e. between geraniol and geranial (citral) and between cinnamyl alcohol and cinnamal.

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

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

CYP1A2 is a member of the cytochrome P450 super family, is one of the best characterized. It is responsible for the metabolism of commonly drugs belonging to classes such as antidepressants, antipsychotics, mood stabilizers, beta blockers and sedative/hypnotics. CYP1A2 also metabolises a number of procarcinogens (such as those in cigarettes). Cigarette smoking may lead to three fold increase in 1A2 activity, which explains why smokers require higher doses of beta blockers than non-smokers.

Drugs that inhibit CYP1A2 will predictably increase the plasma concentrations of the medications or decrease in clearance of substrates.

Drugs such as ciprofloxacin, fluvoxamine, verapamil, cimetidine, caffeine and isoniazid are inhibitors of CYP1A2 enzyme. Vegetables such as grape fruit juice, cumic and turmeric are inhibitors of the CYP1A2 enzyme which may lead to increase plasma concentration of psychotropics.

Inhibition of NF- κ B in vivo can be detrimental. NF- κ B controls multiple functions in homeostasis including a functional immune response, cell cycle, and cell death. Genetic studies in mice and analysis of naturally occurring mutations in humans point to specific developmental and immune consequences due to altering NF- κ B activity.

The same functions that make NF- κ B attractive for developing inhibitors for treating disease also play a role in homeostasis, and disruption of the NF- κ B pathway during development or in adults leads to unfavorable and potentially unhealthy consequences.

NF- κ B plays a role in multiple homeostatic cellular processes including response to stimuli, cell proliferation, and death, regulating communication between cells, but is also tightly linked with other signaling pathways within the cell, such as p38 and JNK. In addition to mediating proinflammatory responses, NF- κ B may regulate apoptotic and cell cycle changes induced by cellular stress, DNA damage or oncogenes by communication with the tumor suppressor p53. Disruption of normal cellular responses by inhibiting NF- κ B can have adverse consequences such as immune suppression and tissue damage.

Understanding the consequences of lack of NF- κ B activity in adult humans comes from observation of naturally occurring genetic deficiencies in this pathway. Mutations have been discovered in humans in signaling molecules upstream of NF- κ B resulting in defects in development or immunity. Genetic defects have also been discovered in genes that immediately affect NF- κ B activation including IKK gamma (NEMO), a subunit of the IKK complex, and I κ Balpha. The IKK gamma mutations result in a defective IKK complex and the I κ Balpha mutation results in an I κ Balpha protein that cannot be phosphorylated and degraded. Both genetic defects result in suppressed NF- κ B.

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activation and ectodermal dysplasia with immunodeficiency. In general patients with these genetic defects have multiple immunological defects including impaired innate immunity, impaired antibody production, and ultimately severe bacterial infections. Understanding the immune defects and susceptibilities in patients with genetic defects in the NF- κ B pathway will help prepare for potential adverse effects of pharmacologic NF- κ B inhibitors.

The requirement for NF- κ B in the development and maintenance of the immune system is well documented. NF- κ B is required for survival during fetal development and for normal lymphocyte generation in adult mice. Removal of the p65 (RelA) subunit of NF- κ B or the IKK β gene results in death during fetal development primarily due to massive liver apoptosis.

Fetal liver stem cells from p65 or IKK β deficient mice have been transplanted into irradiated hosts revealing a specific requirement of NF- κ B for T-cells, B-cells, and common lymphoid progenitor development but not for myeloid cells or stem cells. The failure to produce lymphocytes is mediated through hypersensitivity to TNF due to lack of NF- κ B activity. Lymphocyte depletion with chemical or genetic inhibition of NF- κ B have implications for therapeutic potential use in humans. The double-sided nature of NF- κ B inhibition is clear in this instance where chemical inhibition in vivo mimics genetic experiments inducing rapid TNF-dependent apoptosis. Rapid induction of apoptosis may be an advantage for treating some forms of cancer, but at the same time cause depletion of some lymphocyte populations. In addition to controlling lymphocyte development, NF- κ B plays a major role in both adaptive and innate immunity. Various signaling pathways responding to receptor recognition of immune challenge converge on NF- κ B which then regulates genes that control the immune response. Both T-cell receptor and B-cell receptors activate NF- κ B through phosphorylation of CARMA1 by PKC θ and PKC β respectively, resulting in recruitment and activation of IKK and ultimately expression of genes that control cellular activation, proliferation, and survival. In addition, NF- κ B plays a role in T-cell response to costimulatory signals. Cells respond to pathogenic microorganisms in part through recognition by Toll-like receptors (TLRs). TLR-family members recognize different molecular structures present in microbes and respond by activating signaling pathways including NF- κ B leading to expression of anti-microbial effector molecules, as well as molecules that help in development of the adaptive immune response. Inhibition of NF- κ B during TLR stimulation can lead to macrophage apoptosis, a mechanism used by some pathogens to help evade immune response. NF- κ B is clearly required for normal mature B-cell and T-cell maintenance and function, including regulatory, memory, and natural killer-like T cells. Inhibition of NF- κ B activation in lymphocytes results in defects in growth, survival, and cytokine production and blocks multiple steps in germinal center formation. Given the diverse roles NF- κ B plays in immune response to pathogens it is not surprising to find mice genetically deficient in components of the NF- κ B pathway are susceptible to parasitic and bacterial infection.

The role of NF- κ B in inhibition of apoptosis is one of the factors that make it a potential target for cancer therapy. NF- κ B deficient mice die during embryogenesis in part due to TNF-mediated liver damage. Adult mice with impaired NF- κ B targeted to the liver have normal liver function, but have severe liver damage after challenge with concanavalin A, a pan-T cell activator. Liver damage occurs due to sustained activation of JNK due to accumulation of reactive oxygen species (ROS) in the absence of normal NF- κ B activation.

The aryl alkyl alcohol (AAA) fragrance ingredients are a diverse group of chemical structures with similar metabolic and toxicity profiles.

The AAA fragrances demonstrate low acute and subchronic dermal and oral toxicity.

At concentrations likely to be encountered by consumers, AAA fragrance ingredients are non-irritating to the skin.

The potential for eye irritation is minimal.

With the exception of benzyl alcohol and to a lesser extent phenethyl and 2-phenoxyethyl AAA alcohols, human sensitization studies, diagnostic patch tests and human induction studies, indicate that AAA fragrance ingredients generally have no or low sensitization potential. Available data indicate that the potential for photosensitization is low.

NOAELs for maternal and developmental toxicity are far in excess of current human exposure levels.

No carcinogenicity in rats or mice was observed in 2-year chronic testing of benzyl alcohol or a-methylbenzyl alcohol; the latter did induce species and gender-specific renal adenomas in male rats at the high dose. There was no to little genotoxicity, mutagenicity, or clastogenicity in the mutagenicity in vitro bacterial assays, and in vitro mammalian cell assays. All in vivo micronucleus assays were negative.

It is concluded that these materials would not present a safety concern at current levels of use as fragrance ingredients.

The Research Institute for Fragrance Materials (RIFM) Expert Panel

A member or analogue of a group of benzyl derivatives generally regarded as safe (GRAS) based in part on their self-limiting properties as flavouring substances in food; their rapid absorption, metabolic detoxification, and excretion in humans and other animals, their low level of flavour use, the wide margin of safety between the conservative estimates of intake and the no-observed-adverse effect levels determined from chronic and subchronic studies and the lack of significant genotoxic and mutagenic potential. This evidence of safety is supported by the fact that the intake of benzyl derivatives as natural components of traditional foods is greater than the intake as intentionally added flavouring substances.

All members of this group are aromatic primary alcohols, aldehydes, carboxylic acids or their corresponding esters or acetals. The substances in this group:

- contain a benzene ring substituted with a reactive primary oxygenated functional group or can be hydrolysed to such a functional group
- the major pathway of metabolic detoxification involves hydrolysis and oxidation to yield the corresponding benzoic acid derivative which is excreted either as the free acid or the glycine conjugate
- they show a consistent pattern of toxicity in both short- and long- term studies and
- they exhibit no evidence of genotoxicity in standardised batteries of in vitro and in vivo assays.

The benzyl derivatives are rapidly absorbed through the gut, metabolised primarily in the liver, and excreted in the urine as glycine conjugates of benzoic acid derivatives.

In general, aromatic esters are hydrolysed in vivo through the catalytic activity of carboxylesterases, the most important of which are the A-esterases. Hydrolysis of benzyl and benzoate esters to yield corresponding alcohols and carboxylic acids and hydrolysis of acetals to yield benzaldehyde and simple alcohols have been reported in several experiments.

The alcohols and aldehydes are rapidly oxidised to benzoic acid while benzoate esters are hydrolysed to benzoic acid.

Flavor and Extract Manufacturers Association (FEMA)

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

For benzyl alkyl alcohols:

Unlike benzylic alcohols, the beta-hydroxyl group of the members of this cluster is unlikely to undergo phase II metabolic activation. Instead, the beta-hydroxyl group is expected to contribute to detoxification via oxidation to hydrophilic acid. Despite structural similarity to carcinogenic ethyl benzene, only a marginal concern has been assigned to phenethyl alcohol due to limited mechanistic analogy.

For benzoates:

Acute toxicity: Benzyl alcohol, benzoic acid and its sodium and potassium salt can be considered as a single category regarding human health, as they are all rapidly metabolised and excreted via a common pathway within 24 hrs. Systemic toxic effects of similar nature (e.g. liver, kidney) were observed. However with benzoic acid and its salts toxic effects are seen at higher doses than with benzyl alcohol.

The compounds exhibit low acute toxicity as for the oral and dermal route. The LD50 values are > 2000 mg/kg bw except for benzyl alcohol which needs to be considered as harmful by the oral route in view of an oral LD50 of 1610 mg/kg bw. The 4 hrs inhalation exposure of benzyl alcohol or benzoic acid at 4 and 12 mg/l as aerosol/dust respectively gave no mortality, showing low acute toxicity by inhalation for these compounds.

Benzoic acid and benzyl alcohol are slightly irritating to the skin, while sodium benzoate was not skin irritating. No data are available for potassium benzoate but it is also expected not to be skin irritating. Benzoic acid and benzyl alcohol are irritating to the eye and sodium benzoate was only slightly irritating to the eye. No data are available for potassium benzoate but it is expected also to be only slightly irritating to the eye.

Sensitisation: The available studies for benzoic acid gave no indication for a sensitising effect in animals, however occasionally very low positive reactions were recorded with humans (dermatological patients) in patch tests. The same occurs for sodium benzoate. It has been suggested that the very low positive reactions are non-immunologic contact urticaria. Benzyl alcohol gave positive and negative results in animals. Benzyl alcohol also demonstrated a maximum incidence of sensitization of only 1% in human patch testing. Over several decades no sensitization with these compounds has been seen among workers.

Repeat dose toxicity: For benzoic acid repeated dose oral toxicity studies give a NOAEL of 800 mg/kg/day. For the salts values > 1000 mg/kg/day are obtained. At higher doses increased mortality, reduced weight gain, liver and kidney effects were observed.

For benzyl alcohol the long-term studies indicate a NOAEL > 400 mg/kg bw/d for rats and > 200 mg/kg bw/d for mice. At higher doses effects on bodyweights, lesions in the brains, thymus, skeletal muscle and kidney were observed. It should be taken into account that administration in these studies was by gavage route, at which saturation of metabolic pathways is likely to occur.

Multimin Chrome Injection for Cattle

	<p>Mutagenicity: All chemicals showed no mutagenic activity in <i>in vitro</i> Ames tests. Various results were obtained with other <i>in vitro</i> genotoxicity assays. Sodium benzoate and benzyl alcohol showed no genotoxicity <i>in vivo</i>. While some mixed and/or equivocal <i>in vitro</i> chromosomal/chromatid responses have been observed, no genotoxicity was observed in the <i>in vivo</i> cytogenetic, micronucleus, or other assays. The weight of the evidence of the <i>in vitro</i> and <i>in vivo</i> genotoxicity data indicates that these chemicals are not mutagenic or clastogenic. They also are not carcinogenic in long-term carcinogenicity studies.</p> <p>In a 4-generation study with benzoic acid no effects on reproduction were seen (NOAEL: 750 mg/kg). No compound related effects on reproductive organs (gross and histopathology examination) could be found in the (sub) chronic studies in rats and mice with benzyl acetate, benzyl alcohol, benzaldehyde, sodium benzoate and supports a non-reprotoxic potential of these compounds. In addition, data from reprotoxicity studies on benzyl acetate (NOAEL >2000 mg/kg bw/d; rats and mice) and benzaldehyde (tested only up to 5 mg/kg bw; rats) support the non-reprotoxicity of benzyl alcohol and benzoic acid and its salts.</p> <p>Developmental toxicity: In rats for sodium benzoate dosed via food during the entire gestation developmental effects occurred only in the presence of marked maternal toxicity (reduced food intake and decreased body weight) (NOAEL = 1400 mg/kg bw). For hamster (NOEL: 300 mg/kg bw), rabbit (NOEL: 250 mg/kg bw) and mice (CD-1 mice, NOEL: 175 mg/kg bw) no higher doses (all by gavage) were tested and no maternal toxicity was observed. For benzyl alcohol: NOAEL= 550 mg/kg bw (gavage; CD-1 mice). LOAEL = 750 mg/kg bw (gavage mice). In this study maternal toxicity was observed e.g. increased mortality, reduced body weight and clinical toxicology. Benzyl acetate: NOEL = 500 mg/kg bw (gavage rats). No maternal toxicity was observed.</p>		
EDTA DISODIUM ZINC SALT & EDTA COPPER DISODIUM SALT & EDTA DISODIUM SALT & EDTA, DISODIUM MANGANESE SALT & CHROMIC CHLORIDE & SODIUM SELENITE & BENZENEMETHANOL	<p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p>		
EDTA DISODIUM ZINC SALT & EDTA COPPER DISODIUM SALT & DWATER	No significant acute toxicological data identified in literature search.		
EDTA DISODIUM ZINC SALT & EDTA COPPER DISODIUM SALT & EDTA DISODIUM SALT & EDTA, DISODIUM MANGANESE SALT	<p>For ethylenediaminetetraacetic acid (EDTA) and its salts:</p> <p>EDTA is a strong organic acid (approximately 1000 times stronger than acetic acid). It has a high affinity for alkaline-earth ions (for example, calcium and magnesium) and heavy-metal ions (for example, lead and mercury). This affinity generally results in the formation of highly stable and soluble hexadentate chelate complexes. EDTA's ability to complex is used commercially to either promote or inhibit chemical reactions, depending on application.</p> <p>EDTA and its salts are expected to be absorbed by the lungs and gastrointestinal tract; absorption through the skin is unlikely.</p> <p>In general, EDTA and its salts are mild skin irritants but considered severe eye irritants. The greatest risk in the human body will occur when the EDTA attempts to scavenge the trace metals used and required by the body.</p> <p>The binding of divalent and trivalent cations by EDTA can cause mineral deficiencies, which seem to be responsible for all of the known pharmacological effects. Sensitivity to the toxic effects of EDTA is, at least in part, related to the deficiency of zinc.</p> <p>Several short term studies, reported no adverse effects from administering doses up to 5% of EDTA and its salts to lab rodents daily and for several weeks. Only diarrhoea and lowered food consumption were reported in animals given 5% disodium EDTA. However, abnormal effects were seen in animals that were fed mineral deficient diets. Abnormal symptoms were observed in male and female rats fed a low mineral diet (0.54% Ca and 0.013%Fe) with the addition of 0%, 0.5%, or 1% disodium EDTA for 205 days. Rats fed a low percent of disodium EDTA in the diet for short term studies with adequate minerals showed no signs of toxicity. Rats fed 0.5% disodium EDTA for 44-52 weeks were without deleterious effects on weight gain, appetite, activity and appearance. Rats fed 1% disodium EDTA with adequate mineral diet for 220 days showed no evidence of dental erosion.</p> <p>EDTA and its salts are eliminated from the body, 95% via the kidneys and 5% by the bile, along with the metals and free ionic calcium which was bound in transit through the circulatory system.</p> <p>Trisodium EDTA was tested in a bioassay for carcinogenicity by the National Cancer Institute. Trisodium EDTA administered to male and female rats at low (3,750 ppm) or high (7,500 ppm) concentrations for 103 weeks produced no compound-related signs of chemical toxicity, and tumor incidence was not related to treatment.</p> <p>EDTA and its salts should not pose a teratogenic concern based on previous studies in lab rodents. Study results indicate no teratogenic effects are likely in lab rodents at doses up to 1000 mg/kg. Adequate minerals in the diet and administration of tap water prevented possible teratogenic effects of EDTA during pregnancy. Teratogenic effects observed in lab rodents were likely due to animals maintained on deionised water and a semi-purified diet, and housed in nonmetallic caging. Infants and children will unlikely be exposed to high concentrations as in lab rodents.</p> <p>Rats given 1250 mg/kg or 1500 mg/kg by gavage exhibited more maternal toxicity than the diet group, but produced only 21% malformations in the offspring at the lower dose. The subcutaneously administration of 375 mg/kg was also maternally toxic, but did not result in malformations in the offspring. Differences in toxicity and teratogenicity are probably related to absorption differences and interaction with metals. Disodium EDTA ingested during pregnancy is teratogenic in rats at 2% in the diet and greater.</p> <p>The maximum human consumption of EDTA and its salts in foods was reported to be in the order of 0.4 mg/kg/day. Infants and children also generally drink tap water instead of deionised or distilled water. Even if young infants were to be fed some solid food, given the characteristics of EDTA and its salts, residues are not likely to be present at concentrations for potential sensitivity.</p>		
EDTA COPPER DISODIUM SALT & EDTA, DISODIUM MANGANESE SALT & CHROMIC CHLORIDE & SODIUM SELENITE	<p>Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.</p>		
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

Multimin Chrome Injection for Cattle	Endpoint	Test Duration (hr)	Species	Value	Source
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Continued...

Multimin Chrome Injection for Cattle

	Not Available	Not Available	Not Available	Not Available	Not Available
EDTA disodium zinc salt	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	72h	Algae or other aquatic plants	0.39mg/l	2
	EC50	72h	Algae or other aquatic plants	2.77mg/l	2
	EC50	48h	Crustacea	100.9mg/l	2
	LC50	96h	Fish	41mg/l	2
EDTA copper disodium salt	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
EDTA disodium salt	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>60mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
	LC50	96h	Fish	>100mg/l	2
	NOEC(ECx)	504h	Crustacea	25mg/l	2
EDTA, disodium manganese salt	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	649.3mg/l	2
	EC50	48h	Crustacea	100.9mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	1mg/l	2
chromic chloride	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	1.5h	Fish	0.004mg/L	4
	LC50	96h	Fish	0.39mg/L	4
	EC50	48h	Crustacea	3.01-3.49mg/l	4
	EC50	96h	Algae or other aquatic plants	0.397mg/L	2
sodium selenite	Endpoint	Test Duration (hr)	Species	Value	Source
	BCF	672h	Fish	<8.1-12	7
	NOEC(ECx)	840h	Fish	0.002mg/L	4
	LC50	96h	Fish	13-200mg/l	Not Available
	EC50	72h	Algae or other aquatic plants	0.032-0.1mg/L	4
	EC50	48h	Crustacea	0.47mg/l	4
	EC50	96h	Algae or other aquatic plants	0.032mg/L	2
Benzenemethanol	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	336h	Fish	5.1mg/l	2
	LC50	96h	Fish	10mg/l	2
	EC50	72h	Algae or other aquatic plants	500mg/l	2
	EC50	48h	Crustacea	230mg/l	2
	EC50	96h	Algae or other aquatic plants	76.828mg/l	2
Dwater	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available

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

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
EDTA disodium salt	LOW	LOW
EDTA, disodium manganese salt	HIGH	HIGH
Benzenemethanol	LOW	LOW
Dwater	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
EDTA disodium salt	LOW (LogKOW = -3.8573)

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Ingredient	Bioaccumulation
EDTA, disodium manganese salt	LOW (LogKOW = -9.4414)
sodium selenite	LOW (BCF = 85)
Benzenemethanol	LOW (LogKOW = 1.1)

Mobility in soil

Ingredient	Mobility
EDTA disodium salt	LOW (Log KOC = 1046)
EDTA, disodium manganese salt	LOW (Log KOC = 465.2)
Benzenemethanol	LOW (Log KOC = 15.66)

SECTION 13 Disposal considerations

Waste treatment methods	
Product / Packaging disposal	<ul style="list-style-type: none">Containers may still present a chemical hazard/ danger when empty.Return to supplier for reuse/ recycling if possible. Otherwise: <ul style="list-style-type: none">If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.Where possible retain label warnings and SDS and observe all notices pertaining to the product.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.Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified.Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or incineration in a licensed apparatus (after admixture with suitable combustible material).Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

SECTION 14 Transport information

Labels Required	
Marine Pollutant	NO
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

14.7.1. 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
EDTA disodium zinc salt	Not Available
EDTA copper disodium salt	Not Available
EDTA disodium salt	Not Available
EDTA, disodium manganese salt	Not Available
chromic chloride	Not Available
sodium selenite	Not Available
Benzenemethanol	Not Available
Dwater	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
EDTA disodium zinc salt	Not Available
EDTA copper disodium salt	Not Available
EDTA disodium salt	Not Available
EDTA, disodium manganese salt	Not Available
chromic chloride	Not Available
sodium selenite	Not Available
Benzenemethanol	Not Available
Dwater	Not Available

SECTION 15 Regulatory information

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

EDTA disodium zinc salt is found on the following regulatory lists
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australian Inventory of Industrial Chemicals (AIIC)
EDTA copper disodium salt is found on the following regulatory lists
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
EDTA disodium salt is found on the following regulatory lists
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australian Inventory of Industrial Chemicals (AIIC)
EDTA, disodium manganese salt is found on the following regulatory lists
Australian Inventory of Industrial Chemicals (AIIC)
chromic chloride is found on the following regulatory lists
Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic
sodium selenite is found on the following regulatory lists
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic
Benzenemethanol is found on the following regulatory lists
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Dwater is found on the following regulatory lists
Australian Inventory of Industrial Chemicals (AIIC)

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (EDTA copper disodium salt)
Canada - DSL	No (EDTA copper disodium salt)
Canada - NDSL	No (EDTA disodium zinc salt; EDTA copper disodium salt; EDTA disodium salt; EDTA, disodium manganese salt; chromic chloride; sodium selenite; Benzenemethanol; Dwater)
China - IECSC	No (EDTA copper disodium salt)
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (EDTA copper disodium salt)
Korea - KECI	No (EDTA disodium zinc salt; EDTA copper disodium salt)
New Zealand - NZIoC	Yes
Philippines - PICCS	No (EDTA disodium zinc salt; EDTA copper disodium salt; EDTA, disodium manganese salt)
USA - TSCA	No (EDTA copper disodium salt)
Taiwan - TCSI	Yes
Mexico - INSQ	No (EDTA disodium zinc salt; EDTA copper disodium salt; EDTA, disodium manganese salt)
Vietnam - NCI	Yes
Russia - FBEPH	No (EDTA disodium zinc salt; EDTA copper disodium salt; EDTA, disodium manganese salt)
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	12/10/2021
Initial Date	10/29/2020

SDS Version Summary

Version	Date of Update	Sections Updated
8.1	08/20/2021	Classification change due to full database hazard calculation/update.
9.1	12/10/2021	Classification change due to full database hazard calculation/update.

Other information

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

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

Definitions and abbreviations

- PC - TWA: Permissible Concentration-Time Weighted Average
- PC - STEL: Permissible Concentration-Short Term Exposure Limit
- IARC: International Agency for Research on Cancer
- ACGIH: American Conference of Governmental Industrial Hygienists
- STEL: Short Term Exposure Limit
- TEEL: Temporary Emergency Exposure Limit
- IDLH: Immediately Dangerous to Life or Health Concentrations
- ES: Exposure Standard
- OSF: Odour Safety Factor
- NOAEL: No Observed Adverse Effect Level
- LOAEL: Lowest Observed Adverse Effect Level
- TLV: Threshold Limit Value
- LOD: Limit Of Detection
- OTV: Odour Threshold Value
- BCF: BioConcentration Factors
- BEI: Biological Exposure Index
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration

- AIIC: Australian Inventory of Industrial Chemicals
- DSL: Domestic Substances List
- NDSL: Non-Domestic Substances List
- IECSC: Inventory of Existing Chemical Substance in China
- 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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TEL (+61 3) 9572 4700.