Ranvet Pty Ltd

Chemwatch: 5555-76

Version No: 3.1 Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements Chemwatch Hazard Alert Code: 2

Issue Date: **09/06/2022** Print Date: **08/08/2024** L.GHS.AUS.EN.E

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

Product Identifier Product name Ranvet's Flexi Joint Plus 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

	Mix 30g/500kg body weight (One scoop) in feed daily.
Relevant identified uses	SDS are intended for use in the workplace ONLY. For domestic-use products, refer to consumer labels.
	Use according to manufacturer's directions.

Details of the manufacturer or supplier of the safety data sheet

Ranvet Pty Ltd
10-12 Green Street Banksmeadow NSW 2019 Australia
+61 2 9666 1744
+61 2 9666 1755
https://www.ranvet.com.au/other_msds.htm
info@ranvet.com.au

Emergency telephone number

Association / Organisation	Ranvet Pty Ltd	
Emergency telephone numbers	+61 417 580 980	
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	1	
Toxicity	1	0 = Minimum
Body Contact	1	1 = Low
Reactivity	1	2 = Moderate
Chronic	2	3 = High 4 = Extreme

Poisons Schedule	Not Applicable
Classification ^[1]	Germ Cell Mutagenicity Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements		
Hazard pictogram(s)		
Signal word	Warning	
Hazard statement(s)		
H341	Suspected of causing genetic defects.	
Precautionary statement(s) Prevention		

P280	Wear protective gloves and protective clothing.		
Precautionary statement(s) Response			
P308+P313	IF exposed or concerned: Get medical advice/ attention.		
Precautionary statement(s) Storage			
P405	Store locked up.		
Precautionary statement(s) Disposal			

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

SECTION 3 Composition / information on ingredients

P501

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name	
CAS NU	%[weight]	Name	
29031-19-4	<30	glucosamine sulfate	
Not Available	<10	polysaccharide, proprietary	
Not Available	<5	amino acid, proprietary	
50-81-7	<5	5 <u>ascorbic acid</u>	
Not Available	balance	carriers, binders proprietary	
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 If this product comes in contact with the eyes: Wash out immediately with fresh running water. • Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the Eye Contact upper and lower lids. Seek medical attention without delay; if pain persists or recurs seek medical attention. Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. If skin contact occurs: Immediately remove all contaminated clothing, including footwear. Flush skin and hair with running water (and soap if available). Seek medical attention in event of irritation. For thermal burns: Decontaminate area around burn. Consider the use of cold packs and topical antibiotics. For first-degree burns (affecting top layer of skin) Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides. Use compresses if running water is not available. Cover with sterile non-adhesive bandage or clean cloth. Do NOT apply butter or ointments; this may cause infection. Give over-the counter pain relievers if pain increases or swelling, redness, fever occur. For second-degree burns (affecting top two layers of skin) Cool the burn by immerse in cold running water for 10-15 minutes. Use compresses if running water is not available. Do NOT apply ice as this may lower body temperature and cause further damage. Do NOT break blisters or apply butter or ointments; this may cause infection. Skin Contact Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape. To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort): Lay the person flat. Elevate feet about 12 inches. Elevate burn area above heart level, if possible. Cover the person with coat or blanket. Seek medical assistance. For third-degree burns Seek immediate medical or emergency assistance In the mean time: Protect burn area cover loosely with sterile, nonstick bandage or, for large areas, a sheet or other material that will not leave lint in wound Separate burned toes and fingers with dry, sterile dressings. Do not soak burn in water or apply ointments or butter; this may cause infection. To prevent shock see above. For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway. Have a person with a facial burn sit up. Check pulse and breathing to monitor for shock until emergency help arrives 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. Inhalation Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. Transport to hospital, or doctor. Ingestion If swallowed do NOT induce vomiting If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration

Observe the patient carefully. Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. Seek medical advice.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

Both dermal and oral toxicity of manganese salts is low because of limited solubility of manganese. No known permanent pulmonary sequelae develop after acute manganese exposure. Treatment is supportive.

[Ellenhorn and Barceloux: Medical Toxicology]

In clinical trials with miners exposed to manganese-containing dusts, L-dopa relieved extrapyramidal symptoms of both hypo kinetic and dystonic patients. For short periods of time symptoms could also be controlled with scopolamine and amphetamine. BAL and calcium EDTA prove ineffective.

[Gosselin et al: Clinical Toxicology of Commercial Products.]

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

-p	
Fire Incompatibility	Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
Advice for firefighters	
Fire Fighting	 Alert Fire Brigade and tell them location and nature of hazard. Wear 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	 Combustible solid which burns but propagates flame with difficulty; it is estimated that most organic dusts are combustible (circa 70%) - according to the circumstances under which the combustion process occurs, such materials may cause fires and / or dust explosions. Organic powders when finely divide over a range of concentrations regardless of particulate size or shape and suspended in air or some other oxidizing medium may form explosive dust-air mixtures and result in a fire or dust explosion (including secondary explosions). Avoid generating dust, particularly clouds of dust in a confined or unventilated space as dusts may form an explosive mixture with air, and any source of giniton, i.e. flame or spark, will cause fire or explosion. Dust clouds generated by the fine grinding of the solid are a particular hazard; accumulations of fine dust (420 micron or less) may burn rapidly and fieredy if ginted - particles exceeding this limit will generally not form filmmable dust clouds; once initiated, however, larger particles up to 1400 microns diameter will contribute to the propagation of an explosive limit (UEL) and upper explosive limit (UEL) are applicable to dust clouds but only the LEL is of practical use; - this to because of the inherent difficulty of advinya proximist.ginitable (hybrid) mixtures may be formed with combustibe dusts. Ignitable mixtures will increase the mate of explosion pressure rise and the Minimum Ignition Energy (the minimum amount of energy required to ignite dust clouds - ME) will be lower than the prive dust in air mixture. The Lower Explosive limit (LEL) of the vapour/dust mixture will be lower than the individual LELs for the vapors/mists or dusts. A dust explosion may release of large quantilies of gaseous products; this in turn creates a subsequent pressure rise of explosive force capable of damaging plantant building and injuring people. Usually the initial or primary explosion takes place in a confined space su
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	 Remove all ignition sources. Clean up all spills immediately. Avoid contact with skin and eyes. Control personal contact with the substance, by using protective equipment. Use dry clean up procedures and avoid generating dust. Place in a suitable, labelled container for waste disposal.
Major Spills	 Moderate hazard. CAUTION: Advise personnel in area. Alert Emergency Services and tell them location and nature of hazard. Control personal contact by wearing protective clothing. Prevent, by any means available, spillage from entering drains or water courses. Recover product wherever possible. IF DRY: Use dry clean up procedures and avoid generating dust. Collect residues and place in sealed plastic bags or other containers for disposal. IF WET: Vacuum/shovel up and place in labelled containers for disposal. ALWAYS: Wash area down with large amounts of water and prevent runoff into drains. If contamination of drains or waterways occurs, advise Emergency Services.

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

SECTION 7 Handling and storage

Precautions for safe handling	
Safe handling	 Avoid all personal contact, including inhalation. Wear protective clothing when risk of exposure occurs. Use in a well-ventilated area. Prevent concentration in hollows and sumps. Do NOT enter confined spaces until atmosphere has been checked. Do NOT allow material to contact humans, exposed food or food utensils. Avoid contact with incompatible materials. When handing, 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. Organic powders when finely divided over a range of concentrations regardless of particulate size or shape and suspended in air or some other oxidizing medium may form explosive dust-air mixtures and result in a fire or dust explosion (including secondary explosions) Minimise airborne dust and eliminate all ignition sources. Keep away from heat, hot surfaces, sparks, and flame. Establish good housekeeping practices. Be econtinuous suction at points of dust generation to capture and minimise the accumulation of dusts. Particular attention should be given to overhead and hidden horizontal surfaces to minimise the probability of a "secondary" explosion. According to NFPA Standard 654, dust layers 1/32 in (0.8 mm) thick can be sufficient to warrant immediate cleaning of the area. Do not use air hoses for cleaning. Minimise dry sweeping to avoid generation of dust clouds. Vacuum dust-accumulating surfaces and remove to a chemical disposal area. Vacuums with explosi
Other information	 Store in original containers. Keep containers securely sealed. Store in a cool, dry area protected from environmental extremes. 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. For major quantities: Consider storage in bunded areas - ensure storage areas are isolated from sources of community water (including stormwater, ground water, lakes and streams}. Ensure that accidental discharge to air or water is the subject of a contingency disaster management plan; this may require consultation with local authorities.
Conditions for safe storage, in	cluding any incompatibilities
Suitable container	Polyethylene Pails with lids 1.5kg, 3kg 7kg. Polyethylene or polypropylene container. Check all containers are clearly labelled and free from leaks.

Avoid reaction with oxidising agents

Storage incompatibility

Control parameters

Occupational Exposure Limits (INGREDIENT DATA	OEL)				
Emergency Limits	TEEL-1	TEEL-2		TEEL-3	
Ranvet's Flexi Joint Plus	Not Available	Not Available		Not Available	
Ingredient	Original IDLH		Revised IDLH		
glucosamine sulfate	Not Available		Not Available		
ascorbic acid	Not Available		Not Available		
Occupational Exposure Banding					
Ingredient	Occupational Exposure Band Rating		Occupational Exposure Band Limit		
ascorbic acid	E		≤ 0.01 mg/m³		
Notes:	Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.				

MATERIAL DATA

Ceiling values were recommended for manganese and compounds in earlier publications. As manganese is a chronic toxin a TWA is considered more appropriate. Because workers exposed to fume exhibited manganism at air-borne concentrations below those that affect workers exposed to dust a lower value has been proposed to provide an extra margin of safety. This value is still above that experienced by two workers exposed to manganese fume in the course of one study.

Exposure controls

ca Th Pr En str de En Lo pro An	ingineering controls are used to remove a hazard or place a an be highly effective in protecting workers and will typically he basic types of engineering controls are: rocess controls which involve changing the way a job activity inclosure and/or isolation of emission source which keeps a trategically "adds" and "removes" air in the work environmen esign of a ventilation system must match the particular proce imployers may need to use multiple types of controls to preve ocal expansiventilation usually required. If risk of overexpose	be independent of worker interactions to provide this hig y or process is done to reduce the risk. selected hazard "physically" away from the worker and v t. Ventilation can remove or dilute an air contaminant if d ess and chemical or contaminant in use.	h level of protection. rentilation that		
ve	rotection. Supplied-air type respirator may be required in spe in approved self contained breathing apparatus (SCBA) may provide adequate ventilation in warehouse or closed storage elocities which, in turn, determine the "capture velocities" of the Type of Contaminant:	be required in some situations. area. Air contaminants generated in the workplace posse	equate protection. ess varying "escape" ntaminant. Air Speed:		
s	solvent, vapours, degreasing etc., evaporating from tank (in	ı 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)				
	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)				
	grinding, abrasive blasting, tumbling, high speed wheel gen of very high rapid air motion).	2.5-10 m/s (500- 2000 f/min.)			
Wi	Within each range the appropriate value depends on:				
1	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				
de ad a r me	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					
•	 Safety glasses with side shields. Chemical goggles. [AS/NZS 1337.1, EN166 or national ed Contact lenses may pose a special hazard; soft contact le describing the wearing of lenses or restrictions on use, sh lens absorption and adsorption for the class of chemicals should be trained in their removal and suitable equipment irrigation immediately and remove contact lens as soon as irritation - lens should be removed in a clean environment Intelligence Bulletin 59]. 	nses may absorb and concentrate irritants. A written pol nould be created for each workplace or task. This should in use and an account of injury experience. Medical and t should be readily available. In the event of chemical exp s practicable. Lens should be removed at the first signs of	include a review of first-aid personnel posure, begin eye of eye redness or		
Skin protection Se	ee Hand protection below				

Hands/feet protection Hands/feet protection For some and house and house the substances has to be obtained in the infantactule of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried throughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: interquency and duration of contact. interquency and duration of the protection application. When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time e zater than 20 minutes according to EN 374, ASIN25 2161.1 on rational equivalent) is recommended. When only brief contact is expected, a glove with a protection class of 5 or higher (breakthrough time e zater than 20 minutes according time > 480 min Goor when Breakthrough time > 20 min Fair when breakthrough time > 20 min Fair when glove matching and and the dependent on the exact composition of the glove with a specific chemical, as the peremetact effection of the glove with a contactes of vapro	Body protection Other protection	belyvinyl chloride. Gloves should be examined for wear and/ or degradation constantly. See Other protection below Voveralls. P.V.C apron. Barrier cream. Skin cleansing cream. Eye wash unit.
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.	Hands/feet protection	 manufacturer. Where the Chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: requency and duration of contact, chemical resistance of glove material, glove thickness and desterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. Contaminated gloves should be replaced. As defined in ASTM F-739-66 in any application, gloves are rated as: Excellent when breakthrough time > 480 min For when breakthrough time > 480 min For when breakthrough time > 20 min Fair when breakthrough time > 20 min<!--</td-->

Respiratory protection

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	P1 Air-line*	-	PAPR-P1 -
up to 50 x ES	Air-line**	P2	PAPR-P2
up to 100 x ES	-	P3	-
		Air-line*	-
100+ x ES	-	Air-line**	PAPR-P3

* - Negative pressure demand ** - Continuous flow

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)

· Respirators may be necessary when engineering and administrative controls do not adequately prevent exposures.

• The decision to use respiratory protection should be based on professional judgment that takes into account toxicity information, exposure measurement data, and frequency and likelihood of the worker's exposure - ensure users are not subject to high thermal loads which may result in heat stress or distress due to personal protective equipment (powered, positive flow, full face apparatus may be an option).

· Published occupational exposure limits, where they exist, will assist in determining the adequacy of the selected respiratory protection. These may be government mandated or vendor recommended.

· Certified respirators will be useful for protecting workers from inhalation of particulates when properly selected and fit tested as part of a complete respiratory protection program.

. Where protection from nuisance levels of dusts are desired, use type N95 (US) or type P1 (EN143) dust masks. Use respirators and components tested and approved under appropriate government standards such as NIOSH (US) or CEN (EU)

· Use approved positive flow mask if significant quantities of dust becomes airborne.

· Try to avoid creating dust conditions.

Class P2 particulate filters are used for protection against mechanically and thermally generated particulates or both.

P2 is a respiratory filter rating under various international standards, Filters at least 94% of airborne particles Suitable for:

Relatively small particles generated by mechanical processes eg. grinding, cutting, sanding, drilling, sawing.
 Sub-micron thermally generated particles e.g. welding fumes, fertilizer and bushfire smoke.

Biologically active airborne particles under specified infection control applications e.g. viruses, bacteria, COVID-19, SARS

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Bright yellow pellet with apple odour. Crystalline		
Physical state	Divided Solid	Relative density (Water = 1)	Not Available
Odour	Apple	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Applicable	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Applicable
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (1%)	Not Applicable
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Inhalation of dusts, generated by the material during the course of normal handling, may be damaging to the health of the individual. Persons with impaired respiratory function, airway diseases and conditions such as emphysema or chronic bronchitis, may incur further disability if excessive concentrations of particulate are inhaled. If prior damage to the circulatory or nervous systems has occurred or if kidney damage has been sustained, proper screenings should be conducted on individuals who may be exposed to further risk if handling and use of the material result in excessive exposures. Manganese fume is toxic and produces nervous system effects characterised by tiredness. Acute poisoning is rare although acute inflammation of the lungs may occur. A chemical pneumonia may also result from frequent exposure. 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.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual.
Skin Contact	Limited 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. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	Although the material is not thought to be an irritant (as classified by EC Directives), direct contact with the eye may cause transient discomfort characterised by tearing or conjunctival redness (as with windburn). Slight abrasive damage may also result. The material may produce foreign body irritation in certain individuals.
Chronic	Strong evidence exists that the substance may cause irreversible but non-lethal mutagenic effects following a single exposure. Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects. Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects.
	Continued

Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

	micron penetrating and remaining in the lung. A prime sy Repeated or prolonged exposure may also damage the from inhalation or chronic ingestion of manganese conta manganese poisoning if it is not treated, but it is not fata Chronic exposure has been associated with two major e "manganism", a neuropsychiatric disorder that may also accumulation of toxic concentrations in critical organs. T appear before any pathology is evident and may include dystonia (disordered muscle tone), fatigue, anorexia, as Insomnia may be an early finding. Chronic poisoning ma The onset of chronic manganese poisoning is insidious, follows with certain definitive features: unaccountable la aggressiveness and hallucinations. The final stage is ch system effects. Symptoms resemble those of Parkinson' produce lesions mimicking those found in Parkinsonism. from exposure, the course may be reversed. Inhalation of manganese fumes may cause 'metal fume aches. Manganese dust is no longer believed to be a ca aggravating factor to a preexisting condition. Prolonged or repeated eye contact may result in conjunc	ffects; bronchitis/pneumonitis following inhalation of manganese dusts and arise from inhalation exposures. Chronic exposure to low levels may result in the he brain in particular appears to sustain cellular damage to the ganglion. Symptoms a mask-like facial expression, spastic gait, tremors, slurred speech, sometimes thenia (loss of strength and energy), apathy and the inability to concentrate. by occur over a 6-24 month period depending on exposure levels. with apathy, anorexia weakness, headache and spasms. Manganese psychosis ughter, euphoria, impulsive acts, absentmindedness, mental confusion, aracterised by speech difficulties, muscular twitching, spastic gait and other nervous s disease. Rat studies indicate the gradual accumulation of brain manganese to If the disease is diagnosed whilst still in the early stages and the patient is removed fever' characterised by flu-like symptoms: fever, chill, nausea, weakness and body usative factor in pneumonia. If there is any relationship at all, it appears to be as an
Ranvet's Flexi Joint Plus	TOXICITY	IRRITATION
	Not Available	Not Available
	ΤΟΧΙΟΙΤΥ	IRRITATION
glucosamine sulfate	Not Available	for potassium salt Non-irritating - Skin Non-irritating - Eye
	ΤΟΧΙΟΙΤΥ	IRRITATION
ascorbic acid	Oral (Rat) LD50: 11900 mg/kg ^[2]	Not Available
Legend:	Value obtained from Europe ECHA Registered Substi specified data extracted from RTECS - Register of Toxic * Newsmart (Nantong) Chem-Spec Ind. MSDS	ances - Acute toxicity 2. Value obtained from manufacturer's SDS. Unless otherwise Effect of chemical Substances
	headache, insomnia, and mild and temporary digestive of diarrhea, and vomiting. In rare human cases, the combin blood pressure and heart rate and palpitations. There is some preliminary evidence suggesting that gluc insulin and/or haemoglobin A1C (a test that measures h with diabetes or insulin resistance. Another concern has been that the extra glucosamine con hexosamine biosynthesis pathway but several investigat Glucosamine sulfate may increase the risk of developing glucosamine and chondroitin sulfate are biochemically of glucose, so these compounds do not raise blood sugar 1 intolerance and has no documented effects on glucose in High dosages of glucosamine may cause gastric problet Special Precautions and warnings: <u>Pregnancy or breast-feeding.</u> There is nott enough reliat acetyl glucosamine is safe to use when pregnant or breas <u>Asthma</u> : There is one report linking an asthma attack wi asthma attack. <u>Diabetes</u> : Some early research suggested that glucosam reliable research now shows that glucosamine does not appears to be safe for most people with diabetes, but ble <u>Glaucoma</u> : Glucosamine does not seem to increase <u>High cholesterol</u> : Some early research suggested that glucosamine does not seem to increase <u>High blood pressur</u> : Some early research suggested that glucosamine shows that glucosamine does not seem to increase <u>High blood pressure</u> : Some early research suggested that glucosamine does not seem to increase <u>High blood pressure</u> : Some early research suggested that shows that glucosamine products might be contaminated with O-GlcNAcylation O -GlcNAcylation at the shell. But some people have do some glucosamine products might be contaminated with O-GlcNAcylation addition or removal of N-acetylglucosa O -GlcNAcylation, addition or removal of N-acetylglucosa O -GlcNAcylation, addition or removal of N-acetylglucosa O -GlcNAcylation at phosphorylation often compete for proteins, and is often seen as a response to stress. Hyperglycemia increases O-GlcNAcylation, leading to in dysfunctional form	g insulin resistance and could decrease the metabolic actions of insulin. Although lassed as carbohydrates (sugars), the body is not able to break them down into by providing an additional source of glucose. Glucosamine does not cause glucose netabolism. ms, nausea , diarrhea, indigestion, and heartburn. ble information to know if glucosamine sulfate, glucosamine hydrochloride, or N- ast-feeding. th taking glucosamine. It is not known for sure if glucosamine was the cause of the nine might raise blood sugar in people with diabetes. But more recent and more seem to affect blood sugar control in people with type 2 diabetes. Glucosamine bod sugar should be monitored closely. side the eye and could worsen glaucoma. lucosamine may increase cholesterol levels. But more recent and reliable research cholesterol levels. at glucosamine may increase insulin levels. But more recent and reliable research

The Norwegian Food Safety Authority calculated Margin of Safety (MoS) values for the use of 10% Glucosamine Sulfate in a body lotion, leg cream, face cream, and from overall exposure from cosmetics. The MoS for each of these formulation types were 35.0, 99.0, 178.0, and

29.2, respectively

Skin penetration

The penetration ability of acetyl glucosamine was evaluated in split-thickness Caucasian cadaver skin. Approximately 7% of the applied test substance (which contained 2% acetyl glucosamine) permeated the skin after 6 h. An in vitro permeation assay was also performed with glucosamine HCl in human epidermal membranes. Over a 48-h period, glucosamine HCl permeated through the skin with a flux of 1.497 ± 0.42 µg/cm2/h, a permeability coefficient of 5.66 ± 1.6 x 10-6 cm/h, and a lag time of 10.9 ± 4.6 h. In a different study, the skin permeation rate of glucosamine sulfate was determined to be 13.27 ug/cm2/h when evaluated in Sprague-

Dawley full-thickness rat skin. Female Beagle dogs were given a single dose of 450 mg glucosamine HCI, and a pharmacokinetic analysis was performed. Glucosamine was detected in the blood up to 8 h post-dose, with a Tmax of 2 h and a Cmax of 9.69 ug/ml. [14C] Glucosamine HCl diluted with unlabeled glucosamine sulfate was given to Sprague-Dawley rats to examine excretion patterns of radioactivity. Radioactivity analysis in tissues and organs revealed that [14C] glucosamine quickly entered into all tissues, included cartilage, reaching a maximum at 8 h.

Bioavailability was also evaluated in humans. Healthy, Chinese, adult males, under fasting conditions, were given a single oral dose of 480 mg glucosamine HCI in a dispersible tablet or capsule form. The mean Cmax, Tmax, and T1/2 values were reported to be 907.1 ng/ml, 3.03 h, and 1.10 h, respectively, for the dispersible tablet form, and 944.40 ng/ml, 3.30 h, and 1.50 h, respectively, for the capsule form. The pharmacokinetics of glucosamine after a single oral administration of glucosamine sulfate and glucosamine HCI were evaluated in 12 healthy volunteers. Glucosamine was determined at steady state in plasma collected up to 48 h after the last dose by a validated LC-MS/MS method. After glucosamine sulfate administration, peak concentrations and extent of exposure averaged 9.1 ± 6.3 µM and 76.5 ± 23.0 µM/h. respectively. Significantly lower plasma concentrations (p= 0.005) were determined after the administration of glucosamine HCI. Acute toxicity:

The lowest reported oral LD50s for glucosamine were reported to be >5000 mg/kg in mice, and >8000 mg/kg in rats and rabbits. In a 9-wk study, glucosamine (0.5%) was fed to male Sprague-Dawley and Spontaneously Hypertensive rats (SHR) rats. The systolic blood pressure in treated rats was statistically significantly lower than control animals. No statistically significant histological differences were found in the hearts, kidneys, and livers, among the treated and control groups. Acetyl glucosamine (up to 5%) was fed to F344 rats for 13 weeks. No obvious indications of toxicity were observed in any of the parameters evaluated. The NOAEL was determined to be > 5%. The effect of orally-ingested acetyl glucosamine (1000 mg) was evaluated in healthy Japanese adults. Volunteers ingested the dissolved acetyl glucosamine in water, once a day, for 16 weeks. A control group received green tea extract powder. Routine physical and cardiovascular characteristics, hematology, and blood chemistry, did not show any significant abnormalities between control and treated groups. The potential toxic effects of a tablet containing glucosamine HCI (1500 mg/d), chondroitin sulfate (1200 mg/d), and manganese ascorbate (228 mg/d) in degenerative disease patients was evaluated in a 16-week crossover study. No patients reported symptoms requiring termination of study and symptom frequency on medication was similar to that at baseline. Vital signs, occult blood testing, and hematologic parameters were similar among the placebo and medicated groups. The chronic toxicity potential of acetyl glucosamine (up to 5%) given in the diet for 52 weeks was evaluated in F344 rats. No toxic effects were observed in any parameter evaluated, however, slight suppression of body weight gain was observed in animals dosed with concentrations of greater than 2.5%.

Reproductive toxicity

The effects of glucosamine (20 mg) treatment via oral ingestion and peritoneal injection was evaluated in 8-week old and 16-week old adult female C57B1/6 mice. Mice were fed the test substance via diet for 3 week, and injected with glucosamine for 3 consecutive days. On the third day of injection, mice were mated. Pregnancy outcomes were assessed at day 18 of gestation. Fetal weight and length were reduced in glucosamine-treated 16-wk old mice, compared to control animals. In addition, a significantly higher number of abnormal fetuses was present in litters of 16-wk old glucosamine-treated mice compared with all other groups (p < 0.05). The effects of premating glucosamine supplementation via drinking water on Sprague-Dawley rat litter homogeneity, uterine receptivity, and maternal hormones levels were evaluated. Female rats were given 0.5 mK Glucosamine via drinking water for 2 wk, and then mated. Birth weights and absolute and relative ovary weights were statistically significantly greater in the glucosamine-treated group compared to the control group (P < 0.05). Maternal progesterone, estradiol, and insulin-like growth factor 1 (IGF-1) concentrations on day 19.5 of pregnancy were significantly increased in treated rats, while insulin and total cholesterol levels were significantly decreased compared with control rats. The effects of intrauterine glucosamine (up to 1500 µg) were evaluated in female ICR mice. Ten days after implantation of the glucosamine pellet, mice were mated. Mice that received glucosamine pellets delivered significantly fewer live pups/litter over a 60-d pellet active period than those that received placebo pellets. However, after the 60-day pellet active period, there was no statistically significant difference in litter sizes delivered by glucosamine-treated and placebo-treated mice, except at the highest dose level

Genotoxicity:

Acetyl glucosamine (up to 5000 µg/plate) was considered to be non-mutagenic in an Ames assay using S. typhimurium strains TA 1537, TA 1535, TA 98, TA 100, and TA 102, with and without metabolic activation. Similarly, an Ames assay was performed on glucosamine HCl derived from Aspergillus niger. Tester strains (S. typhimurium and E. coli WP2 uvrA) were exposed to up to 5000 ug/plate of the test substance, with and without metabolic activation. No mutagenicity was observed. In an in vivo micronucleus assay, mice (strain not reported) were administered Aspergillus niger-derived glucosamine HCI (up to 2000 mg/kg bw) in water, via gavage. There was no statistically significant decrease in the ratios of polychromatic erythrocytes (PCE) and normochromatic erythrocytes (NCE) at any dose level. In an in vitro anti-genotoxicity assay, human peripheral lymphocytes were exposed to glucosamine or acetyl glucosamine at concentrations up to 50 mM. DNA damage was induced with hydrogen peroxide. Glucosamine, at all concentrations, showed a significant protective activity (P < 0.001) against hydrogen peroxide-induced DNA damage. Acetyl glucosamine only indicated a slight DNA protection at the highest test concentration. The chemoprotective ability of glucosamine (diets containing up to 150 mg/kg glucosamine; 7 day exposure) against cisplatininduced genotoxicity was evaluated in male Wistar rats. The test substance was considered to be an effective chemoprotector against cisplatin-induced DNA damage

Carcinogenicity:

The carcinogenic potential of acetyl glucosamine (up to 5% in the diet; 104-week treatment) was evaluated in F344 rats. The test substance was considered to be non-carcinogenic. The anti-proliferative potential of glucosamine (10 mM) was evaluated in human renal cancer cell lines (786-O and caki-1) via an 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) and fluorescein isothiocyanate (FITC)annexin V/PI assay. The apoptosis rate of both cell lines was up-regulated by the high concentration of glucosamine (10 mM), but downregulated by low concentrations of glucosamine (1 and 5 mM), as compared with the control groups. The growth inhibitory effects of glucosamine, glucosamine HCl, and acetyl glucosamine on human haematoma SMMC-721 cells was

evaluated in vitro. Tumor cells were exposed to glucosamine, glucosamine HCI, or acetyl glucosamine, at concentrations of up to 1000 ug/ml. Results measured by an MTT assay showed that glucosamine HCI and glucosamine caused a concentration-dependent reduction in hepatoma cell growth.

In an in-vivo anti-carcinogenicity assay, Kunming male mice were inoculated with sarcoma 180 tumor cells. Mice were orally treated with up to 500 mg/kg glucosamine HCl dissolved in saline for 10 d. Glucosamine HCl, at the intermediate dose (250 mg/kg/d), had the highest inhibition ratio (34.02%) on sarcoma 180 tumor growth.

Melanin effects:

The effect of acetyl glucosamine on melanin production was evaluated in an in vitro assay. Reconstituted human tanned epidermis cells were exposed to up to 5% acetyl glucosamine in water for 10 days. Dose-dependent decreases in melanin content were observed. The whitening effect of acetyl glucosamine (5%) was evaluated in human and brown guinea pig skin subjected to UV-induced pigmentation. A visual reduction in hyperpigmentation was observed 2 week after treatment with the acetyl glucosamine solution, in humans, compared to the vehicle-treated group. Acetyl glucosamine-treated guinea pig skin had decreased levels of melanin without affecting the number of melanocytes, compared to vehicle-treated skin.

The reduction of facial hyperpigmentation after topical treatment on acetyl glucosamine was evaluated in a 10-week trial. Volunteers (101 women/group) were instructed to apply a facial lotion containing 4% niacinamide and 2% acetyl glucosamine twice a day for 8 weeks. A control group applied the lotion vehicle without 4% and 2% acetyl glucosamine. By all parameters measured, the niacinamide and acetyl glucosamine formulation regimen caused a significant reduction in the detectable area of facial spots and appearance of pigmentation compared to the controls (P < 0.05). In a similar study, healthy Japanese women (n = 25 women/group) were instructed to apply a facial lotion containing 2% acetyl glucosamine on the side of the face, twice daily, for 8 weeks. A control group applied the vehicle lotion that did not contain acetyl glucosamine. Topical 2% acetyl glucosamine reduced the appearance of facial hyperpigmentation, with an overall directional (p = 0.089) spot area fraction change across the entire study. The effects of a neck cream formulation containing 8% acetyl glucosamine was evaluated in 45 Caucasian women. Applications of the cream occurred once a day, for 16 week. The test cream was welltolerated with no signs of irritation. One subject experienced an adverse event of contact dermatitis on two separate occasions. No other adverse events were reported.

Allergenicity:

ASCORBIC ACID	The effect of glucosamine injections (concentrations u female BALB/c mice. Clinical dermatitis scores decrea IL-17 decreased after glucosamine administration (eac did not show differences across groups. The anti-alleg rhinitis was evaluated. OVA-specific IgE and eosinoph glucosamine treatment compared with the positive cor groups treated with glucosamine when compared to th The anti-allergic effects of orally-ingested acetyl gluco: in BALB/c mice with dinitrofluorobenzene (DNFB)-indu significantly inhibited DNFB-induced ear swelling in mi concentration of histamine in both the ear and plasma The tolerability of orally-ingested, shrimp-derived gluco 1500 mg of synthetically-derived or shrimp-derived gluco shrimp-derived and synthetic sources, without any inci Controlled, double-blind, clinical trial. Among the 16 pa atopic dermatitis symptoms. Three glucosamine (25 mg/ controlled, double-blind, clinical trial. Among the 16 pa atopic dermatitis symptoms. Three glucosamine was eval of cell viability was similar in the negative control and t A Direct Peptide Reactivity Assay (DPRA) was perform This assay is designed to mimic the covalent binding of reactivity of chemicals towards the model synthetic pe lysine was 1%, interpreted as minimal reactivity in the Ocular toxicity: An in vitro ocular irritation assay was performed in bow vitro irritancy scores for the test substance, negative of 105.42, respectively. Case Reports A 52-year old complained of exacerbation of underlyin containing 500 mg glucosamine. Within 24 h of discon completely resolved. A 67-year-old woman with arterial hypertension and os sulfate intake. After treatment with antihistamines and The association between glucosamine use and colorer log their glucosamine intake form 2001 - 2011. Curren lower risk of colon cancer (HR: 0.83, 95% Cl: 0.71 - 0. Similarly, the association between lung cancer risk (H Asthma-like symptoms may continue for months or evi- condition known as reactive airways dysfunction syndr compound. Main criteria for diagnosing	sed with increasing glucosamine do ch group: P = 0.002 and P < 0.001, r gic effect of glucosamine (concentra- lis in bronchoalveolar lavage (BAL) 1 htrol group. In addition, significant im re positive control group. samine and glucosamine HCI (up to icced skin sensitization. Oral administ ice at both 6 hours and 24 hours afte of DNFB-treated mice (P < 0.05). beamine was evaluated in 15 shrimp cosamine. All subjects tolerated the idences of hypersensitivity. kg) in the treatment of atopic dermat tients receiving glucosamine treatme ed patients reported adverse effects, uated in an in vitro assay using 3 red treated groups, therefore, the substa ned according to OECD TG 442C in of electrophilic chemicals to nucleoph ptides containing cysteine and lysine assay, and yielding a prediction of n rine comeas using a saline solution of ontrol (saline), and positive control (c g asthma after beginning treatment of thuing glucosamine for 3 week, GF steoarthritis was referred for evaluati corticosteroids, symptoms resolved tal cancer risk was examined amon t use of glucosamine, modeled using 97), compared to those who reporte (cosamine was evaluated in 76,904 y from the years 2000 - 2010. Compa IR: 0.80, 95% CI: 0.65 - 0.99) after n en years after exposure to the mater ome (RADS) which can occur after the absence of previous airways dis nours of a documented exposure to 1 sts, moderate to severe bronchial b duration of exposure to the irritating	se (P < 0.001). Concentrations of tissue IL-13 and espectively), but the concentrations of tissue IL-4 tions up to 5%) in female BALB/c mice with allergic fluid were significantly decreased after 5% oral provement of inflammation was apparent in 1 mg/mouse; 6 day treatment) was also evaluated tration of acetyl glucosamine or glucosamine HCI er DNFB challenge (P < 0.05), and reduced the allergic individuals. Subjects were given either 1500 mg glucosamine administration from the titis was evaluated in an 8-week placebo- ent, 15 patients reported clinical improvement of with abdominal pain being the most common constructed human epidermis samples. Reduction noce was considered to be non-irritating. order to evaluate the sensitization. hilic centers in skin proteins by quantifying the a. The mean percent depletion of cysteine and o sensitization containing 20% acetyl glucosamine. The mean in 20% imidazole in saline) were 0.42, 0.70, and with a glucosamine-chondroitin sulfate preparation treatment, the patient s asthma symptoms sufficiency and a reduction in GFR supposedly R increased from 47.5 to 60 ml/min. on after an episode of urticaria after glucosamine within 4 hours. og 113,067 volunteers. Participants were asked to g a time-varying exposure, was associate with a d no ingestion of glucosamine. volunteers with no prior history of lung cancer. The red to non-use, use of glucosamine was nultivariable adjustment rial ends. This may be due to a non-allergic exposure to high levels of highly irritating tease in a non-atopic individual, with sudden onset the irritant. Other criteria for diagnosis of RADS perreactivity on methacholine challenge testing, following an irritating inhalation is an infrequent substance. On the other hand, industrial bronchitis
	is a disorder that occurs as a result of exposure due to reversible after exposure ceases. The disorder is char	8	
Acute Toxicity	×	Carcinogenicity	×
Skin Irritation/Corrosion	×	Reproductivity	×
Serious Eye Damage/Irritation	×	STOT - Single Exposure	×
Respiratory or Skin sensitisation	×	STOT - Repeated Exposure	×

SECTION 12 Ecological information

Mutagenicity

~

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

Legend:

Aspiration Hazard

×

- Data available to make classification

🗙 – Data either not available or does not fill the criteria for classification

For manganese and its compounds:

Environmental fate:

It has been established that while lower organisms (e.g., plankton, aquatic plants, and some fish) can significantly bioconcentrate manganese, higher organisms (including humans) tend to maintain manganese homeostasis. This indicates that the potential for biomagnification of manganese from lower trophic levels to higher ones is low.

(Japan) - Bioconcentration Data 8. Vendor Data

There were two mechanisms involved in explaining the retention of manganese and other metals in the environment by soil. First, through cation exchange reactions, manganese ions and the charged surface of soil particles form manganese oxides, hydroxides, and oxyhydroxides which in turn form absorption sites for other metals. Secondly, manganese can be adsorbed to other oxides, hydroxides, and oxyhydroxides through ligand exchange reactions. When the soil solution becomes saturated, these manganese oxides, hydroxides, and oxyhydroxides can precipitate into a new mineral phase and act as a new surface to which other substances can absorb. The tendency of soluble manganese compounds to adsorb to soils and sediments depends mainly on the cation exchange capacity and the organic composition of the soil. The soil adsorption constants (the ratio of the concentration in soil to the concentration in water) for Mn(II) span five orders of mangitude, ranging from 0.2 to 10,000 mL/g, increasing as a function of the organic content and the ion exchange capacity of the soil; thus, adsorption may be highly variable. In some cases, adsorption of manganese to soils may not be a readily reversible process. At low concentrations, manganese may be "fixed" by clays and will not be released into solution readily. At higher concentrations, manganese may be desorbed by ion exchange mechanisms with other ions in solution. For example, the discharge of waste water effluent into estuarine environments resulted in the mobilization of manganese in soil adseiments. The metals in the effluent may have been preferentially adsorbed resulting in the release of manganese. The oxidation state of manganese in soil adseiments may be altered by microbial activity; oxidation may lead to the precipitation of manganese. Bacteria and microflora can increase the mobility of manganese.

The transport and partitioning of manganese in water is controlled by the solubility of the specific chemical form present, which in turn is determined by pH, Eh (oxidationreduction potential), and the characteristics of the available anions. The metal may exist in water in any of four oxidation states.

Manganese(II) predominates in most waters (pH 4-7) but may become oxidized at a pH >8 or 9. The principal anion associated with Mn(II) in water is usually carbonate (CO3.2), and the concentration of manganese is limited by the relatively low solubility (65 mg/L) of MnCO3. In relatively oxidized water, the solubility of Mn(II) may be controlled by manganese oxide equilibria, with manganese being converted to the Mn(II) or Mn(IV) oxidation states. In extremely reduced water, the fate of manganese tends to be controlled by formation of a poorly soluble sulfide. Manganese in water may undergo oxidation at high pH or Eh and is also subject to microbial activity. For example, Mn(II) in a lake was oxidized during the summer months, but this was inhibited by a microbial poison, indicating that the oxidation was mediated by bacteria . The microbial metabolism of manganese is presumed to be a function of pH, temperature, and other factors.

Manganese in water may be significantly bioconcentrated at lower trophic levels. A bioconcentration factor (BCF) relates the concentration of a chemical in plant and animal tissues to the concentration of the chemical in the water in which they live. The BCF of manganese was estimated as 2,500 - 6,300 for phytoplankton, 300 -5,500 for marine algae, 80 - 830 for intertidal mussels, and 35 - 930 for coastal fish. Similarly, the BCF of manganese was estimated to be 10,00 -20,000 for marine and freshwater plants, 10,000 - 40,000 for invertebrates, and 10 - 600 for fish. In general, these data indicate that lower organisms such as algae have larger BCFs than higher organisms. In order to protect consumers from the risk of manganese bioaccumulation in marine mollusks, the U.S. EPA has set a criterion for manganese at 0.1 mg/L for marine waters.

Elemental manganese and inorganic manganese compounds have negligible vapor pressures but may exist in air as suspended particulate matter derived from industrial emissions or the erosion of soils. Manganese-containing particles are mainly removed from the atmosphere by gravitational settling, with large particles tending to fall out faster than small particles. The half-life of airborne particles is usually on the order of days, depending on the size of the particle and atmospheric conditions. Some removal by washout mechanisms such as rain may also occur, although it is of minor significance in comparison to dry deposition.

Ecotoxicity:

Manganese ion is toxic to aqueous organisms Fish LC50 (28 d): orfe 2490 mg/l, trout 2.91 mg/l Daphnia magna LC50: 50 mg/l Pseudomonas putida LC50: 10.6 mg/l Photobacterium phosphoreum LC50: 14.7 mg/l Turbellarian worms (EC0): Polycelis nigra 660 mg/l (interference threshold); microregma 31 mg/l DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
ascorbic acid	LOW	LOW
Bioaccumulative potential		

Bioaccumulation
LOW (LogKOW = -1.85)
LOW (LogKOW = -1.85)

Mobility in soil

Ingredient	Mobility
ascorbic acid	LOW (Log KOC = 10)

SECTION 13 Disposal considerations

Waste treatment methods		
Product / Packaging disposal	 Containers may still present a chemical hazard/ danger when empty. Return to supplier for reuse/ recycling if possible. Otherwise: If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. Where possible retain label warnings and SDS and observe all notices pertaining to the product. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: Reduction Reuse Recycling Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. In most instances the supplier of the material should be consulted. D 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 sever may be subject to local laws and regulations and these should be considered first. Where in doubt contact the responsible authority. 	

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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		
glucosamine sulfate	Not Available		
ascorbic acid	Not Available		

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type		
glucosamine sulfate	Not Available		
ascorbic acid	Not Available		

SECTION 15 Regulatory information

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

glucosamine sulfate is found on the following regulatory lists

Not Applicable

ascorbic acid 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 (glucosamine sulfate)			
Canada - DSL	No (glucosamine sulfate)			
Canada - NDSL	No (glucosamine sulfate; ascorbic acid)			
China - IECSC	Yes			
Europe - EINEC / ELINCS / NLP	Yes			
Japan - ENCS	No (glucosamine sulfate)			
Korea - KECI	No (glucosamine sulfate)			
New Zealand - NZIoC	Yes			
Philippines - PICCS	No (glucosamine sulfate)			
USA - TSCA	No (glucosamine sulfate)			
Taiwan - TCSI	Yes			
Mexico - INSQ	Yes			
Vietnam - NCI	No (glucosamine sulfate)			
Russia - FBEPH	No (glucosamine sulfate)			
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	09/06/2022
Initial Date	09/05/2022

SDS Version Summary

Version	Date of Update	Sections Updated
2.1	09/05/2022	Toxicological information - Acute Health (swallowed), Physical and chemical properties - Appearance, Handling and storage - Storage (suitable container), Name
3.1	09/06/2022	Physical and chemical properties - Appearance, Composition / information on ingredients - Ingredients, Identification of the substance / mixture and of the company / undertaking - Use

Other information

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

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

Definitions and abbreviations

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