

Ranvet's Batphol Vitamin B Complex

Ranvet

Chemwatch Hazard Alert Code: 2

Chemwatch: 4614-30

Version No: 8.1

Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: 08/20/2021

Print Date: 07/18/2022

L.GHS.AUS.EN.E

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

Product Identifier

Product name	Ranvet's Batphol Vitamin B Complex
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	Prevention and treatment of Vitamin B Complex and Choline deficiencies in horses.
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Details of the supplier of the safety data sheet

Registered company name	Ranvet
Address	10-12 Green Street Banksmeadow NSW 2019 Australia
Telephone	+61 2 9666 1744
Fax	+61 2 9666 1755
Website	http://www.ranvet.com.au/other_msds.htm
Email	info@ranvet.com.au

Emergency telephone number

Association / Organisation	Ranvet
Emergency telephone numbers	+61 425 061 584
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	0		0 = Minimum
Body Contact	0		1 = Low
Reactivity	0		2 = Moderate
Chronic	2		3 = High
			4 = Extreme

Poisons Schedule	Not Applicable
Classification [1]	Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 2A
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)	
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Signal word	Warning
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Hazard statement(s)

H315	Causes skin irritation.
H317	May cause an allergic skin reaction.

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H319 Causes serious eye irritation.

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.
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.

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.

Not Applicable

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
67-03-8	4.5-6.5	<u>thiamine hydrochloride</u>
98-92-0	4.5-5.5	<u>niacinamide</u>
81-13-0	1.8-2.2	<u>d-panthenol</u>
58-56-0	0.9-1.1	<u>pyridoxine hydrochloride</u>
67-48-1	0.4-0.5	<u>choline chloride</u>
130-40-5	0.1-0.2	<u>riboflavin 5'-monophosphate sodium salt</u>
Not Available	<1	performance additives nonhazardous
7732-18-5	>60	<u>water</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 eyes:</p> <ul style="list-style-type: none"> ▶ Wash out immediately with water. ▶ If irritation continues, seek medical attention. ▶ 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.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▶ Other measures are usually unnecessary.
Ingestion	<ul style="list-style-type: none"> ▶ For advice, contact a Poisons Information Centre or a doctor.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 Firefighting measures

Extinguishing media

The product contains a substantial proportion of water, therefore there are no restrictions on the type of extinguishing media which may be used. Choice of extinguishing media should take into account surrounding areas.

Though the material is non-combustible, evaporation of water from the mixture, caused by the heat of nearby fire, may produce floating layers of combustible substances.

In such an event consider:

- ▶ foam.
- ▶ dry chemical powder.
- ▶ carbon dioxide.

Special hazards arising from the substrate or mixture

Fire Incompatibility	Avoid contamination with strong oxidising agents 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 to be a significant fire risk. ▶ Expansion or decomposition on heating may lead to violent rupture of containers. ▶ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO). ▶ May emit acrid smoke. <p>Decomposition may produce toxic fumes of: carbon dioxide (CO₂) nitrogen oxides (NO_x) other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.</p>
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	<p>Minor hazard.</p> <ul style="list-style-type: none"> ▶ Clear area of personnel. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Control personal contact with the substance, by using protective equipment as required. ▶ Prevent spillage from entering drains or water ways. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Absorb remaining product with sand, earth or vermiculite and place in appropriate containers for disposal. ▶ Wash area and prevent runoff into drains or waterways. ▶ 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"> ▶ Limit all unnecessary personal contact. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ When handling DO NOT eat, drink or smoke. ▶ Always wash hands with soap and water after handling. ▶ Avoid physical damage to containers. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.
Other information	<p>Store below 25 degC.</p> <ul style="list-style-type: none"> ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

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Conditions for safe storage, including any incompatibilities

Suitable container	Ampoule. Vial.
Storage incompatibility	▶ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
niacinamide	5.6 mg/m3	62 mg/m3	690 mg/m3

Ingredient	Original IDLH	Revised IDLH
thiamine hydrochloride	Not Available	Not Available
niacinamide	Not Available	Not Available
d-panthenol	Not Available	Not Available
pyridoxine hydrochloride	Not Available	Not Available
choline chloride	Not Available	Not Available
riboflavin 5'-monophosphate sodium salt	Not Available	Not Available
water	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
thiamine hydrochloride	E	≤ 0.01 mg/m³
niacinamide	E	≤ 0.01 mg/m³
pyridoxine hydrochloride	E	≤ 0.01 mg/m³
choline chloride	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

Exposure controls

Appropriate engineering controls	<p>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:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>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.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>General exhaust is adequate under normal operating conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.</p>											
	<table border="1"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>solvent, vapours, degreasing etc., evaporating from tank (in still air)</td> <td>0.25-0.5 m/s (50-100 f/min)</td> </tr> <tr> <td>aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)</td> <td>0.5-1 m/s (100-200 f/min.)</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min)</td> </tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td> <td>2.5-10 m/s (500-2000 f/min.)</td> </tr> </tbody> </table>	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.)	
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<p>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,</p>												

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	accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.
Personal protection	
Eye and face protection	<ul style="list-style-type: none"> ▶ Safety glasses with side shields ▶ Chemical goggles. ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	Wear general protective gloves, eg. light weight rubber gloves.
Body protection	See Other protection below
Other protection	No special equipment needed when handling small quantities. OTHERWISE: <ul style="list-style-type: none"> ▶ Overalls. ▶ Barrier cream. ▶ Eyewash 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
NEOPRENE	A
VITON	A
NATURAL RUBBER	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

Particulate. (AS/NZS 1716 & 1715, EN 143:2000 & 149:001, ANSI Z88 or national equivalent)

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

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

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

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

Appearance	Yellow / orange clear water-thin liquid; mixes with water.		
Physical state	Liquid	Relative density (Water = 1)	1.03-1.04
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Applicable
pH (as supplied)	Not Available	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available
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

Continued...

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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 (Not Available%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Not normally a hazard due to non-volatile nature of product
Ingestion	The material has NOT been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.
Skin Contact	The material is not thought to produce adverse health effects or skin irritation following contact (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting. 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 liquid is not thought to be an irritant (as classified by EC Directives), direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn).
Chronic	Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals. Exposure to small quantities may induce hypersensitivity reactions characterised by acute bronchospasm, hives (urticaria), deep dermal wheals (angioneurotic oedema), running nose (rhinitis) and blurred vision. Anaphylactic shock and skin rash (non-thrombocytopenic purpura) may occur. An individual may be predisposed to such antibody mediated reaction if other chemical agents have caused prior sensitisation (cross-sensitivity). Clinical symptoms and signs of intoxication following occupational exposure to pyridine, its homologues and derivatives include gastrointestinal disturbance with diarrhoea, abdominal pain and nausea, weakness, headache, insomnia and nervousness. Data indicate that piperidine, pyridine, methyl and alkyl derivatives of pyridine (picolines, lutidines collidines), nicotinonitrile and picolinonitrile are slightly to moderately toxic following acute exposures The available data support the conclusion that the pyridines possess similar human health-related data, and in particular, target organs appear to be the liver and the male reproductive tract. The weight-of-evidence suggests that Pyridine and Pyridine Derivatives Category chemicals are not mutagenic. This conclusion is supported by a number of in vivo mutagenicity assays and carcinogenicity studies with negative results for pyridine. Reproductive screening evaluations using several repeated dose toxicity studies indicates that piperidine, pyridine and nicotinonitrile may be male reproductive toxicants. Exposures less than those which produce overt clinical signs may produce varying levels of liver damage with central lobular fatty degeneration, congestion and cellular infiltration; repeated low level exposures may produce cirrhosis. The kidney is less sensitive to pyridine-induced damage than is the liver. Pyridine, like primidone, phenobarbital and oxazepam induces liver neoplasms in mice, but not in rats, even though in rats these chemicals cause a spectrum of toxic liver lesions. The mouse, an animal with a high background rate of liver neoplasms, is particularly sensitive to the development of malignant liver neoplasms following chemical exposure. There is equivocal evidence (1) that pyridine is carcinogenic in male F344/N rats (based on an increased incidence of renal tubule neoplasms), in female rats of the same species (based on increases in mononuclear cell leukaemia), in male Wistar rats (based on an increased incidence of mono- nuclear cell leukaemia), and clear evidence of carcinogenicity (1) in male and female B6C3F1 mice (based on increased incidences of malignant hepatocellular neoplasms). 1: National Toxicology Program Technical Report Series No. 470, March 2000

Ranvet's Batphol Vitamin B Complex	TOXICITY	IRRITATION
	Not Available	Not Available
thiamine hydrochloride	TOXICITY	IRRITATION
	Oral (Rat) LD50: 3710 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1] Skin: no adverse effect observed (not irritating) ^[1]
niacinamide	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	Not Available
	Inhalation(Rat) LC50: >3.8 mg/4h ^[1] Oral (Rat) LD50: >2500 mg/kg ^[1]	

d-panthenol	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 0.5 mg - mild
	Oral (Mouse) LD50; 15000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin (rabbit): 500 mg/4h - mild
		Skin: no adverse effect observed (not irritating) ^[1]
pyridoxine hydrochloride	TOXICITY	IRRITATION
	Oral (Rat) LD50; 4000 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1]
		Skin: no adverse effect observed (not irritating) ^[1]
choline chloride	TOXICITY	IRRITATION
	Oral (Rat) LD50; 3400 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin: no adverse effect observed (not irritating) ^[1]
riboflavin 5'-monophosphate sodium salt	TOXICITY	IRRITATION
	Not Available	Not Available
water	TOXICITY	IRRITATION
	Oral (Rat) LD50; >90000 mg/kg ^[2]	Not Available

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

NIACINAMIDE	<p>Mutation in microorganisms</p> <p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p> <p>The intestinal cytochrome P-450 3A4 system, is responsible for the first-pass metabolism of many medications. Through the inhibition of this enzyme system, inhibitors interact with a variety of medications, leading to elevation of their serum concentrations. Most notable are its effects on cyclosporine, some 1,4-dihydropyridine calcium antagonists, and some 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors. In the case of some drugs, these increased drug concentrations have been associated with an increased frequency of dose-dependent adverse effects. The P-glycoprotein pump, located in the brush border of the intestinal wall, also transports many cytochrome P-450 3A4 substrates, and this transporter also may be affected by CYP3A4 inhibitors..</p> <p>Most calcium channel blockers (CCBs) are metabolized by CYP3A4 and will be affected by strong inhibitors and inducers of CYP3A4. Grapefruit juice in sufficient quantities can block intestinal CYP3A4, which can lead to an enhancement of the effects of CCBs. This could affect the blood pressure response for all CCBs</p> <p>Common classes of drugs that are strong inhibitors of CYP3A4 include azole antifungals, macrolide antibiotics (except azithromycin), protease inhibitors used for HIV, amiodarone, diltiazem, and verapamil.</p> <p>Niacin (nicotinic acid, Vitamin B3, Vitamin PP) and nicotinamide are both converted into the coenzyme NAD. NAD converts to NADP by phosphorylation in the presence of the enzyme NAD+ kinase. NAD and NADP are coenzymes for many dehydrogenases, participating in many hydrogen transfer processes. NAD is important in catabolism of fat, carbohydrate, protein, and alcohol, as well as cell signaling and DNA repair, and NADP mostly in anabolism reactions such as fatty acid and cholesterol synthesis. High energy requirements (brain) or high turnover rate (gut, skin) organs are usually the most susceptible to their deficiency.</p> <p>Activating HCA2 has effects other than lowering serum cholesterol and triglyceride concentrations: antioxidative, anti-inflammatory, antithrombotic, improved endothelial function and plaque stability, all of which counter development and progression of atherosclerosis</p> <p>Niacin inhibits cytochrome P450 enzymes CYP2E1, CYP2D6 and CYP3A4. Niacin produces a rise in serum unconjugated bilirubin in normal individuals and in those with Gilbert's Syndrome. However, in the Gilbert's Syndrome, the rise in bilirubin is higher and clearance is delayed longer than in normal people</p> <p>In animal models and in vitro, niacin produces marked anti-inflammatory effects in a variety of tissues – including the brain, gastrointestinal tract, skin, and vascular tissue – through the activation of hydroxycarboxylic acid receptor 2 (HCA2), also known as niacin receptor 1 (NIACR1) Unlike niacin, nicotinamide does not activate NIACR1; however, both niacin and nicotinamide activate the G protein-coupled estrogen receptor (GPER) in vitro</p> <p>Niacin reduces synthesis of low-density lipoprotein cholesterol (LDL-C), very low-density lipoprotein cholesterol (VLDL-C), lipoprotein(a) and triglycerides, and increases high-density lipoprotein cholesterol (HDL-C) The lipid-therapeutic effects of niacin are partly mediated through the activation of G protein-coupled receptors, including hydroxycarboxylic acid receptor 2 (HCA2) and hydroxycarboxylic acid receptor 3 (HCA3), which are highly expressed in body fat HCA2 and HCA3 inhibit cyclic adenosine monophosphate (cAMP) production and thus suppress the release of free fatty acids (FFAs) from body fat, reducing their availability to the liver to synthesize the blood-circulating lipids in question. A decrease in free fatty acids also suppresses liver expression of apolipoprotein C3 and PPARGgamma coactivator-1b, thus increasing VLDL-C turnover and reducing its production Niacin also directly inhibits the action of diacylglycerol O-acyltransferase 2 (DGAT2) a key enzyme for triglyceride synthesis.</p> <p>The mechanism behind niacin increasing HDL-C is not totally understood, but seems to occur in various ways. Niacin increases apolipoprotein A1 levels by inhibiting the breakdown of this protein, which is a component of HDL-C. It also inhibits HDL-C hepatic uptake by suppressing production of the cholesterol ester transfer protein (CETP) gene. It stimulates the ABCA1 transporter in monocytes and macrophages and upregulates peroxisome proliferator-activated receptor gamma (PPARGgamma), resulting in reverse cholesterol transport.</p> <p>Severe deficiency of niacin in the diet causes the disease pellagra, characterized by diarrhea, sun-sensitive dermatitis involving hyperpigmentation and thickening of the skin, inflammation of the mouth and tongue, delirium, dementia, and if left untreated, death. Common psychiatric symptoms include irritability, poor concentration, anxiety, fatigue, loss of memory, restlessness, apathy, and depression. The biochemical mechanism(s) for the observed deficiency-caused neurodegeneration are not well understood, but may rest on: A) the requirement for nicotinamide adenine dinucleotide (NAD+) to suppress the creation of neurotoxic tryptophan metabolites, B) inhibition of mitochondrial ATP generation, resulting in cell damage; C), activation of the poly (ADP-ribose) polymerase (PARP) pathway, as PARP is a nuclear enzyme involved in DNA repair, but in the absence of NAD+ can lead to cell death; D) reduced synthesis of neuro-protective brain-derived neurotrophic factor or its receptor tropomyosin receptor kinase B; or E) changes to genome expression directly due to the niacin deficiency.</p> <p>Hartnup disease is a hereditary nutritional disorder resulting in niacin deficiency. It is caused by a genetic disorder that results in a failure to absorb the essential amino acid tryptophan, tryptophan being a precursor for niacin synthesis. The symptoms are similar to pellagra, including</p>
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red, scaly rash and sensitivity to sunlight. Oral niacin or niacinamide is given as a treatment for this condition in doses ranging from 50 to 100 mg twice a day, with a good prognosis if identified and treated early. Niacin synthesis is also deficient in carcinoid syndrome, because of metabolic diversion of its precursor tryptophan to form serotonin

Cytochrome P450 enzymes are essential for the metabolism of many medications. Although this class has more than 50 enzymes, six of them metabolize 90 percent of drugs, with the two most significant enzymes being CYP3A4 and CYP2D6. Genetic variability (polymorphism) in these enzymes may influence a patient's response to commonly prescribed drug classes, including beta blockers and antidepressants. Cytochrome P450 enzymes can be inhibited or induced by drugs, resulting in clinically significant drug-drug interactions that can cause unanticipated adverse reactions or therapeutic failures.

Drugs that inhibit CYP2D6 activity are likely to increase the plasma concentrations of certain medications, and, in some cases, adverse outcomes will occur. Some drugs, such as fluoxetine, paroxetine, and quinidine, are particularly potent inhibitors of CYP2D6; patients on these drugs may have almost no CYP2D6 activity.

Clinical results suggest that >30% of patients with a poor or ultrarapid CYP2D6 phenotype may experience an adverse outcome after being prescribed codeine, tramadol, oxycodone, or hydrocodone. These medications are frequently prescribed for pain relief, and ~39% of the US population is expected to carry one of these phenotypes, suggesting that the population-level impact of these gene-drug interactions could be substantial.

For drugs that are converted to active metabolites by CYP2D6, the addition of a CYP2D6 inhibitor will tend to inhibit the efficacy of the drug. Genetic variability in CYP2D6 activity also can affect the outcome of CYP2D6 drug interactions.

In patients genetically deficient in CYP2D6 and who are taking a CYP2D6 substrate, the addition of a CYP2D6 inhibitor will not result in any change in the plasma concentrations of the substrate.

CYP2D6 is highly polymorphic with single-nucleotide polymorphisms, small insertions/deletions and larger structural variants including duplications, deletions, tandem arrangements, and hybridisations with non-functional CYP2D7 pseudogenes. The frequency of these variants differs across populations, and they significantly influence the drug-metabolising enzymatic function of CYP2D6. Importantly, altered CYP2D6 function has been associated with both adverse drug reactions and reduced drug efficacy, and there is growing recognition of the clinical and economic burdens associated with suboptimal drug utilisation

The CYP2D6 genotype is associated with the occurrence of adverse effects and clinical nonresponse in psychiatric patients treated with CYP2D6-dependent antidepressants.

The cytochrome P450 isozymes, in particular CYP2D6, is responsible for the biotransformation of many psychopharmacological drugs . Substrates of CYP2D6 include first generation antipsychotics, selective serotonin receptor inhibitors and tricyclic antidepressants¹. Based on genetic variation, patients can be divided into poor metabolizers (PM), intermediate metabolizers (IM), extensive metabolizers (EM), and ultrarapid metabolizers (UM). The recommended dosages of psychopharmacological medication that are metabolized by this enzyme are based on the metabolism of the most common genotype, i.e., the EM type (i.e., a normal CYP2D6 function). However, because the plasma level of a drug is related to the genotype, the same dosage will probably lead to a higher plasma level in PMs and IMs, as compared to EMs, and to a lower plasma level in UMs as compared to EMs. The plasma level is often related to the effectiveness of the drug and the risk of dose-related side-effects . Also, when physicians prescribe a drug metabolized by CYP2D6 without taking into account the genotype, the hospital stay is longer (and the costs higher) in patients with a PM and UM profile.

D-PANTHENOL

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 epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

CHOLINE CHLORIDE

For choline:
In rabbits, choline chloride may lead to a slight irritation of the skin and eye, which is, however, not sufficient to warrant a classification of choline chloride as an irritant under GHS.

No data on sensitization in animals are available. The skin sensitisation potential of choline chloride for humans is regarded as negligible.

Repeated oral administration of 10g/day in patients with Alzheimer's disease produced a slight hypertensive effect, but no other adverse effects; this dose was regarded as a LOAEL;(it is equivalent to 7.5 mg choline per day). The tolerable upper intake level applied for chronic daily use for adults was set at 3.5 g/day. Inadequate dietary intake decreases choline liver stores and may produce liver abnormalities as indicated by elevated serum alanine aminotransferase levels. As adequate intake for chronic daily use for adult men 550 mg/day of choline is recommended. The adequate intake for adult women is 425 mg/day of choline, during pregnancy 450 mg/day and during lactation 550 mg/day, respectively.

Choline chloride did not produce gene mutations, clastogenicity or DNA damage in *in vitro* mutagenicity studies; furthermore it has no structural alerts. Choline chloride does not have any genotoxic potential.

In a rat repeated dose study, using a single dose level of approximately 500 mg/kg bw/day given over 72 weeks via feed, with a post-observation period of 30 weeks, no significant effects were observed relative to controls with respect to survival rates, body weights and relative liver weights. Only limited pathological investigations were carried out at autopsy (gross examination with histological investigation of only the liver and any tissues showing gross abnormalities). No adverse effects were observed.

Prolonged i.p. administration of choline chloride is toxic to the testes and causes damage to the seminiferous tubules. Under the testing protocol employed these lesions were reversible. However, this route of administration is not relevant for assessment of hazard to humans.

Developmental toxic effects have not been observed in the absence of maternal toxicity. Maternal and developmental toxicity started above the lowest dose which was already higher than the limit dose of 1000 mg/kg bw/day (NOAEL Maternal toxicity and developmental toxicity 1250 mg/kg bw/day). At the highest dose tested (20,000 mg/kg bw/day) 100% of the foetuses were resorbed. No developmental toxic effects were observed in mice after oral doses of 1250 mg/kg bw/day on gestation days 1 to 18. Higher doses, above the levels recommended currently and associated with maternal toxicity, did produce developmental toxic effects, but these were secondary to the maternal toxicity at the excessive doses used. The compound does not produce any significant developmental toxicity in the mouse.

Most undiluted cationic surfactants satisfy the criteria for classification as Harmful (Xn) with R22 and as Irritant (Xi) for skin and eyes with R38 and R41.

For quaternary ammonium compounds (QACs):
Quaternary ammonium compounds (QACs) are cationic surfactants. They are synthetic organically tetra-substituted ammonium compounds, where the R substituents are alkyl or heterocyclic radicals (where hydrogen atoms remain unsubstituted, the term "secondary- or "tertiary-ammonium compounds" is preferred) .

A common characteristic of these synthetic compounds is that one of the R's is a long-chain hydrophobic aliphatic residue

The cationic surface active compounds are in general more toxic than the anionic and non-ionic surfactants. The positively-charged cationic portion is the functional part of the molecule and the local irritation effects of QACs appear to result from the quaternary ammonium cation. Due to their relative ability to solubilise phospholipids and cholesterol in lipid membranes, QACs affect cell permeability which may lead to cell death. Further QACs denature proteins as cationic materials precipitate protein and are accompanied by generalised tissue irritation.

It has been suggested that the experimentally determined decrease in acute toxicity of QACs with chain lengths above C16 is due to decreased water solubility.

In general it appears that QACs with a single long-chain alkyl groups are more toxic and irritating than those with two such substitutions, The straight chain aliphatic QACs have been shown to release histamine from minced guinea pig lung tissue However, studies with benzalkonium chloride have shown that the effect on histamine release depends on the concentration of the solution. When cell suspensions (11% mast cells) from rats were exposed to low concentrations, a decrease in histamine release was seen. When exposed to high concentrations the opposite result was obtained.

In addition, QACs may show curare-like properties (specifically benzalkonium and cetylpyridinium derivatives, a muscular paralysis with no involvement of the central nervous system. This is most often associated with lethal doses Parenteral injections in rats, rabbits and dogs have resulted in prompt but transient limb paralysis and sometimes fatal paresis of the respiratory muscles. This effect seems to be transient.

From human testing of different QACs the generalised conclusion is obtained that all the compounds investigated to date exhibit similar toxicological properties.

Acute toxicity: Studies in rats have indicated poor intestinal absorption of QACs. Acute toxicity of QACs varies with the compound and, especially, the route of administration. For some substances the LD50 value is several hundreds times lower by the i.p. or i.v. than the oral route, whereas toxicities between the congeners only differ in the range of two to five times.

Ranvet's Batphol Vitamin B Complex

	<p>At least some QACs are significantly more toxic in 50% dimethyl sulfoxide than in plain water when given orally. Probably all common QAC derivatives produce similar toxic reactions, but as tested in laboratory animals the oral mean lethal dose varies with the compound.</p> <p>Oral toxicity: LD50 values for QACs have been reported within the range of 250-1000 mg/kg for rats, 150-1000 mg/kg for mice, 150-300 mg/kg for guinea pigs and about 500 mg/kg b.w. for rabbits and dogs. The ranges observed reflect differences in the study designs of these rather old experiments as well as differences between the various QACs.</p> <p>The oral route of administration was characterised by delayed deaths, gastrointestinal lesions and respiratory and central nervous system depression. It was also found that given into a full stomach, the QACs lead to lower mortality and fewer gastrointestinal symptoms. This supports the suggestion of an irritating effect.</p> <p>Dermal toxicity: It has been concluded that the maximum concentration that did not produce irritating effect on intact skin is 0.1%. Irritation became manifest in the 1-10% range. Concentrations below 0.1% have caused irritation in persons with contact dermatitis or broken skin. Although the absorption of QACs through normal skin probably is of less importance than by other routes, studies with excised guinea pig skin have shown that the permeability constants strongly depend on the exposure time and type of skin.</p> <p>Sensitisation: Topical mucosal application of QACs may produce sensitisation. Reports on case stories and patch test have shown that compounds such as benzalkonium chloride, cetalkonium chloride and cetrimide may possibly act as sensitisers. However, in general it is suggested that QACs have a low potential for sensitising man. It is difficult to distinguish between an allergic and an irritative skin reaction due to the inherent skin irritating effect of QACs.</p> <p>Long term/repeated exposure:</p> <p>Inhalation: A group of 196 farmers (with or without respiratory symptoms) were evaluated for the relationship between exposure to QACs (unspecified, exposure levels not given) and respiratory disorders by testing for lung function and bronchial responsiveness to histamine. After histamine provocation statistically significant associations were found between the prevalence of mild bronchial responsiveness (including asthma-like symptoms) and the use of QACs as disinfectant. The association seems even stronger in people without respiratory symptoms.</p> <p>Genetic toxicity: QACs have been investigated for mutagenicity in microbial test systems. In Ames tests using <i>Salmonella typhimurium</i> with and without metabolic activation no signs of mutagenicity have been observed. Negative results were also obtained in <i>E. coli</i> reversion and <i>B. subtilis</i> rec assays. However, for benzalkonium chloride also positive and equivocal results were seen in the <i>B. subtilis</i> rec assays.</p>
THIAMINE HYDROCHLORIDE & NIACINAMIDE & PYRIDOXINE HYDROCHLORIDE & CHOLINE CHLORIDE	<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>
NIACINAMIDE & D-PANTHENOL	<p>The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
RIBOFLAVIN 5'-MONOPHOSPHATE SODIUM SALT & WATER	<p>No significant acute toxicological data identified in literature search.</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

Ranvet's Batphol Vitamin B Complex	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
thiamine hydrochloride	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	NOEC(ECx)	48h	Crustacea	58mg/l	2
niacinamide	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	>1000mg/l	2
d-panthenol	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	72h	Algae or other aquatic plants	100mg/l	2
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	LC50	96h	Fish	>1000mg/l	2
pyridoxine hydrochloride	Endpoint	Test Duration (hr)	Species	Value	Source
	EC10(ECx)	72h	Algae or other aquatic plants	3.3mg/l	2

Continued...

Ranvet's Batphol Vitamin B Complex

	EC50	72h	Algae or other aquatic plants	72mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
	LC50	96h	Fish	>100mg/l	2
choline chloride	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>500mg/l	1
	EC100(ECx)	24h	Crustacea	>30mg/l	4
	EC50	48h	Crustacea	>320mg/l	4
	EC50	96h	Algae or other aquatic plants	>500mg/l	2
	LC50	96h	Fish	>10000mg/l	2
riboflavin 5'-monophosphate sodium salt	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
water	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					

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
thiamine hydrochloride	HIGH	HIGH
niacinamide	HIGH	HIGH
d-panthenol	LOW	LOW
pyridoxine hydrochloride	LOW	LOW
choline chloride	LOW	LOW
water	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
thiamine hydrochloride	LOW (LogKOW = -1.7773)
niacinamide	LOW (LogKOW = -0.37)
d-panthenol	LOW (LogKOW = -1.9222)
pyridoxine hydrochloride	LOW (LogKOW = -0.557)
choline chloride	LOW (LogKOW = -3.1555)

Mobility in soil

Ingredient	Mobility
thiamine hydrochloride	LOW (KOC = 87.51)
niacinamide	LOW (KOC = 51.56)
d-panthenol	LOW (KOC = 10)
pyridoxine hydrochloride	LOW (KOC = 10)
choline chloride	HIGH (KOC = 1.717)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ Recycle wherever possible or consult manufacturer for recycling options. ▶ Consult State Land Waste Management Authority for disposal. ▶ Bury residue in an authorised landfill. ▶ Recycle containers if possible, or dispose of in an authorised landfill.
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SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

Continued...

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

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

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

Not Applicable

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

Product name	Group
thiamine hydrochloride	Not Available
niacinamide	Not Available
d-panthenol	Not Available
pyridoxine hydrochloride	Not Available
choline chloride	Not Available
riboflavin 5'-monophosphate sodium salt	Not Available
water	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
thiamine hydrochloride	Not Available
niacinamide	Not Available
d-panthenol	Not Available
pyridoxine hydrochloride	Not Available
choline chloride	Not Available
riboflavin 5'-monophosphate sodium salt	Not Available
water	Not Available

SECTION 15 Regulatory information

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

thiamine hydrochloride is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

niacinamide is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 3

Australian Inventory of Industrial Chemicals (AIIC)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

d-panthenol is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

pyridoxine hydrochloride 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)

choline chloride is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

riboflavin 5'-monophosphate sodium salt is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

water is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (thiamine hydrochloride; niacinamide; d-panthenol; pyridoxine hydrochloride; choline chloride; riboflavin 5'-monophosphate sodium salt; water)
China - IECSC	No (thiamine hydrochloride)
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (thiamine hydrochloride)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes

Continued...

Ranvet's Batphol Vitamin B Complex

National Inventory	Status
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	No (riboflavin 5'-monophosphate sodium 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	08/20/2021
Initial Date	10/08/2004

SDS Version Summary

Version	Date of Update	Sections Updated
7.1	04/15/2021	Classification change due to full database hazard calculation/update.
8.1	08/20/2021	Classification change due to full database hazard calculation/update.

Other information

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

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

Definitions and abbreviations

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