Avian B

Animal Science Products, Inc.

Part Number: **22290044**
Version No: **2.3**
Safety Data Sheet according to OSHA HazCom Standard (2012) requirements

Issue Date: **14/06/2023**
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L.GHS.USA.EN

SECTION 1 Identification

Product Identifier

Product name	Avian B
Synonyms	Not Available
Other means of identification	22290044

Recommended use of the chemical and restrictions on use

Relevant identified uses	Not Available

Name, address, and telephone number of the chemical manufacturer, importer, or other responsible party

Registered company name	**Animal Science Products, Inc.**
Address	3418 Rayburn Drive, Nacogdoches TX 75961 United States
Telephone	936-560-0003
Fax	Not Available
Website	www.asp-inc.com
Email	Not Available

Emergency phone number

Association / Organisation	**Infotrac**
Emergency telephone numbers	**800-535-5053**
Other emergency telephone numbers	**Not Available**

SECTION 2 Hazard(s) identification

Classification of the substance or mixture

NFPA 704 diamond

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

Classification	Serious Eye Damage/Eye Irritation Category 2A, Specific Target Organ Toxicity - Repeated Exposure Category 2, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Skin Corrosion/Irritation Category 2, Reproductive Toxicity Category 2, Sensitisation (Skin) Category 1, Germ Cell Mutagenicity Category 2

Label elements

Hazard pictogram(s)	

Signal word	**Warning**

Hazard statement(s)

H319	Causes serious eye irritation.
H373	May cause damage to organs through prolonged or repeated exposure.
H335	May cause respiratory irritation.
H315	Causes skin irritation.
H361	Suspected of damaging fertility or the unborn child.
H317	May cause an allergic skin reaction.
H341	Suspected of causing genetic defects.

Hazard(s) not otherwise classified

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe dust/fume.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing dust/fumes.
P202	Do not handle until all safety precautions have been read and understood.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing must not be allowed out of the workplace.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P314	Get medical advice/attention if you feel unwell.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P302+P352	IF ON SKIN: Wash with plenty of water.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

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

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
98-92-0	29	niacinamide
7647-14-5	25	sodium chloride
7447-40-7	15	potassium chloride

CAS No	%[weight]	Name
50-81-7	9	ascorbic acid
58-56-0	1	pyridoxine hydrochloride
59-30-3	.04	folic acid
68-19-9	.008	cyanocobalamin
83-88-5	4	riboflavin

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

SECTION 4 First-aid measures

Description of first aid measures

Eye Contact	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 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.
Skin Contact	If skin contact occurs: ‣ Immediately remove all contaminated clothing, including footwear. ‣ Flush skin and hair with running water (and soap if available). ‣ Seek medical attention in event of irritation. For thermal burns: ‣ Decontaminate area around burn. ‣ Consider the use of cold packs and topical antibiotics. For first-degree burns (affecting top layer of skin) ‣ Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides. ‣ Use compresses if running water is not available. ‣ Cover with sterile non-adhesive bandage or clean cloth. ‣ Do NOT apply butter or ointments; this may cause infection. ‣ Give over-the counter pain relievers if pain increases or swelling, redness, fever occur. For second-degree burns (affecting top two layers of skin) ‣ Cool the burn by immerse in cold running water for 10-15 minutes. ‣ Use compresses if running water is not available. ‣ Do NOT apply ice as this may lower body temperature and cause further damage. ‣ Do NOT break blisters or apply butter or ointments; this may cause infection. ‣ Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape. To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort): ‣ Lay the person flat. ‣ Elevate feet about 12 inches. ‣ Elevate burn area above heart level, if possible. ‣ Cover the person with coat or blanket. ‣ Seek medical assistance. For third-degree burns Seek immediate medical or emergency assistance. In the mean time: ‣ Protect burn area cover loosely with sterile, nonstick bandage or, for large areas, a sheet or other material that will not leave lint in wound. ‣ Separate burned toes and fingers with dry, sterile dressings. ‣ Do not soak burn in water or apply ointments or butter; this may cause infection. ‣ To prevent shock see above. ‣ For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway. ‣ Have a person with a facial burn sit up. ‣ Check pulse and breathing to monitor for shock until emergency help arrives.
Inhalation	‣ If fumes or combustion products are inhaled remove from contaminated area. ‣ Lay patient down. Keep warm and rested. ‣ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ‣ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ‣ Transport to hospital, or doctor, without delay.
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.

Most important symptoms and effects, both acute and delayed

See Section 11

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 Fire-fighting measures

Extinguishing media

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

Special hazards arising from the substrate or mixture

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

Special protective equipment and precautions for fire-fighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear breathing apparatus plus protective gloves. ▸ Prevent, by any means available, spillage from entering drains or water courses. ▸ Use water delivered as a fine spray to control fire and cool adjacent 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. Will burn if ignited. Combustion products include: carbon monoxide (CO) carbon dioxide (CO2) hydrogen chloride phosgene nitrogen oxides (NOx) metal oxides other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.

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	▸ Clean up all spills immediately. ▸ Avoid breathing vapours/ aerosols/ or dusts and avoid 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. ▸ Place in a suitable, labelled container for waste disposal.
Major Spills	▸ Clear area of personnel and move upwind. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear breathing apparatus plus protective gloves.

	‣ Prevent, by any means available, spillage from entering drains or water course. ‣ Stop leak if safe to do so. ‣ Contain spill with sand, earth or vermiculite. ‣ Collect recoverable product into labelled containers for recycling. ‣ Neutralise/decontaminate residue (see Section 13 for specific agent). ‣ Collect solid residues and seal in labelled drums for disposal. ‣ Wash area and prevent runoff into drains. ‣ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ‣ If contamination of drains or waterways occurs, advise emergency services.

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

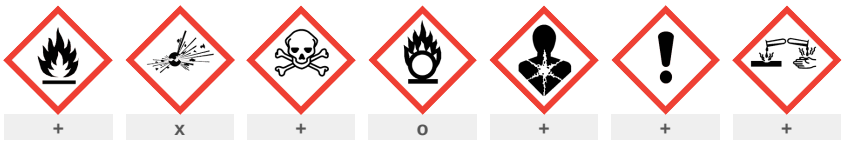
SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	‣ 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 handling, **DO NOT** eat, drink or smoke. ‣ Keep containers securely sealed when not in use. ‣ Avoid physical damage to containers. ‣ Always wash hands with soap and water after handling. ‣ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ‣ Use good occupational work practice. ‣ Observe manufacturer's storage and handling recommendations contained within this SDS. ‣ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	

Conditions for safe storage, including any incompatibilities

Suitable container	‣ Glass container is suitable for laboratory quantities ‣ Polyethylene or polypropylene container. ‣ Packing as recommended by manufacturer. ‣ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	Contains a six-membered heterocyclic ring. Six-membered heterocycles can be described as pi--deficient. Substitution by electronegative groups or additional nitrogen atoms in the ring significantly increase the pi-deficiency. These effects also decrease the basicity. Electrophilic aromatic substitution is more difficult while nucleophilic aromatic substitution is facilitated. for pyridines: · Because of the electronegative nitrogen in the pyridine ring, the molecule is relatively electron deficient. It, therefore, enters less readily electrophilic aromatic substitution reactions, which are characteristic of benzene derivatives; even more so if the reaction mix doesn't scavenge protons released by the reaction (protonated pyridine is even more electron-deficient). However, unlike benzene and its derivatives, pyridine is more prone to nucleophilic substitution and metalation of the ring by strong organometallic bases. · The nitrogen center of pyridine features a basic lone pair of electrons. Because this lone pair is not part of the aromatic ring, pyridine is a base, having chemical properties similar to those of tertiary amines. Pyridine can act as Lewis base, donating its pair of electrons to a Lewis acid. · Pyridine is protonated by reaction with acids and forms a positively charged aromatic polyatomic ion called pyridinium The reactivity of pyridine can be distinguished for three chemical groups. · With electrophiles, electrophilic substitution takes place where pyridine expresses aromatic properties. · With nucleophiles, pyridine reacts at positions 2 and 4 and thus behaves similar to imines and carbonyls. · The reaction with many Lewis acids results in the addition to the nitrogen atom of pyridine, which is similar to the reactivity of tertiary amines. The ability of pyridine and its derivatives to oxidize, forming amine oxides (N-oxides), is also a feature of tertiary amines Secondary amines form salts with strong acids and can be oxidized to the corresponding nitrone using hydrogen peroxide, catalyzed by selenium dioxide ‣ Avoid reaction with oxidising agents



+	x	+	o	+	+	+

X — *Must not be stored together*
0 — *May be stored together with specific preventions*
+ — *May be stored together*

Note: Depending on other risk factors, compatibility assessment based on the table above may not be relevant to storage situations, particularly where large volumes of dangerous goods are stored and handled. Reference should be made to the Safety Data Sheets for each substance or article and risks assessed accordingly.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US OSHA Permissible Exposure Limits (PELs) Table Z-1	folic acid	Particulates Not Otherwise Regulated (PNOR)- Respirable fraction	5 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	folic acid	Particulates Not Otherwise Regulated (PNOR)- Total dust	15 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	folic acid	Inert or Nuisance Dust: Respirable fraction	5 mg/m3 / 15 mppcf	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	folic acid	Inert or Nuisance Dust: Total Dust	15 mg/m3 / 50 mppcf	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	folic acid	Particulates not otherwise regulated	Not Available	Not Available	Not Available	See Appendix D
US OSHA Permissible Exposure Limits (PELs) Table Z-1	riboflavin	Particulates Not Otherwise Regulated (PNOR)- Total dust	15 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	riboflavin	Particulates Not Otherwise Regulated (PNOR)- Respirable fraction	5 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	riboflavin	Inert or Nuisance Dust: Total Dust	15 mg/m3 / 50 mppcf	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	riboflavin	Inert or Nuisance Dust: Respirable fraction	5 mg/m3 / 15 mppcf	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	riboflavin	Particulates not otherwise regulated	Not Available	Not Available	Not Available	See Appendix D

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
niacinamide	5.6 mg/m3	62 mg/m3	690 mg/m3
sodium chloride	0.5 ppm	2 ppm	20 ppm

Ingredient	Original IDLH	Revised IDLH
niacinamide	Not Available	Not Available
sodium chloride	Not Available	Not Available
potassium chloride	Not Available	Not Available
ascorbic acid	Not Available	Not Available
pyridoxine hydrochloride	Not Available	Not Available
folic acid	Not Available	Not Available
cyanocobalamin	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
riboflavin	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
niacinamide	E	≤ 0.01 mg/m³
sodium chloride	E	≤ 0.01 mg/m³
ascorbic acid	E	≤ 0.01 mg/m³
pyridoxine hydrochloride	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

Airborne particulate or vapour must be kept to levels as low as is practicably achievable given access to modern engineering controls and monitoring hardware. Biologically active compounds may produce idiosyncratic effects which are entirely unpredictable on the basis of literature searches and prior clinical experience (both recent and past).
for cobalt:
In view of the serious effects seen in experimental animals after a relatively short exposure period at 0.1 mg/m3 the recommended TLV-TWA is thought to reduce the significant risk of material impairment of health posed by respiratory disease and pulmonary sensitisation which have been shown to occur at higher levels of exposure. The value does not apply generally to cobalt compounds.

A significant increase in the risk of lung cancer was reported among workers involved in cobalt production (with concomitant exposure to nickel and arsenic) and hard-metal workers with documented exposure to cobalt-containing dusts. A significant increase in lung cancer risk has been observed in workers whose exposure began more than 20 years previously. A number of single cases of malignant tumours, mostly sarcomas, have been reported at the site, following implant of cobalt-containing orthopedic implants.

Exposure controls

Appropriate engineering controls

Enclosed local exhaust ventilation is required at points of dust, fume or vapour generation.
HEPA terminated local exhaust ventilation should be considered at point of generation of dust, fumes or vapours.
Barrier protection or laminar flow cabinets should be considered for laboratory scale handling.
A fume hood or vented balance enclosure is recommended for weighing/ transferring quantities exceeding 500 mg.
When handling quantities up to 500 gram in either a standard laboratory with general dilution ventilation (e.g. 6-12 air changes per hour) is preferred. Quantities up to 1 kilogram may require a designated laboratory using fume hood, biological safety cabinet, or approved vented enclosures. Quantities exceeding 1 kilogram should be handled in a designated laboratory or containment laboratory using appropriate barrier/ containment technology.
Manufacturing and pilot plant operations require barrier/ containment and direct coupling technologies.
Barrier/ containment technology and direct coupling (totally enclosed processes that create a barrier between the equipment and the room) typically use double or split butterfly valves and hybrid unidirectional airflow/ local exhaust ventilation solutions (e.g. powder containment booths). Glove bags, isolator glove box systems are optional. HEPA filtration of exhaust from dry product handling areas is required.
Fume-hoods and other open-face containment devices are acceptable when face velocities of at least 1 m/s (200 feet/minute) are achieved. Partitions, barriers, and other partial containment technologies are required to prevent migration of the material to uncontrolled areas. For non-routine emergencies maximum local and general exhaust are necessary. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Air Speed:
solvent, vapours, 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 (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, 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.)

Within each range the appropriate value depends on:

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

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the

	extraction fan, for example, should be a minimum of 1-2.5 m/s (200-500 f/min.) for extraction of gases discharged 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. The need for respiratory protection should also be assessed where incidental or accidental exposure is anticipated: Dependent on levels of contamination, PAPR, full face air purifying devices with P2 or P3 filters or air supplied respirators should be evaluated. The following protective devices are recommended where exposures exceed the recommended exposure control guidelines by factors of: 10; high efficiency particulate (HEPA) filters or cartridges 10-25; loose-fitting (Tyvek or helmet type) HEPA powered-air purifying respirator. 25-50; a full face-piece negative pressure respirator with HEPA filters 50-100; tight-fitting, full face-piece HEPA PAPR 100-1000; a hood-shroud HEPA PAPR or full face-piece supplied air respirator operated in pressure demand or other positive pressure mode.
Individual protection measures, such as personal protective equipment	
Eye and face protection	When handling very small quantities of the material eye protection may not be required. For laboratory, larger scale or bulk handling or where regular exposure in an occupational setting occurs: ‣ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent] ‣ Face shield. Full face shield may be required for supplementary but never for primary protection of eyes. ‣ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].
Skin protection	See Hand protection below
Hands/feet protection	**NOTE:** ‣ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ‣ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. ‣ Rubber gloves (nitrile or low-protein, powder-free latex, latex/ nitrile). Employees allergic to latex gloves should use nitrile gloves in preference. ‣ Double gloving should be considered. ‣ PVC gloves. ‣ Change gloves frequently and when contaminated, punctured or torn. ‣ Wash hands immediately after removing gloves. ‣ Protective shoe covers. [AS/NZS 2210] ‣ Head covering.
Body protection	See Other protection below
Other protection	‣ For quantities up to 500 grams a laboratory coat may be suitable. ‣ For quantities up to 1 kilogram a disposable laboratory coat or coverall of low permeability is recommended. Coveralls should be buttoned at collar and cuffs. ‣ For quantities over 1 kilogram and manufacturing operations, wear disposable coverall of low permeability and disposable shoe covers. ‣ For manufacturing operations, air-supplied full body suits may be required for the provision of advanced respiratory protection. ‣ Eye wash unit. ‣ Ensure there is ready access to an emergency shower. ‣ For Emergencies: Vinyl suit

Respiratory protection

Type -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	Powder

Physical state	Powder	**Relative density (Water = 1)**	Not Available
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature (°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 Available
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Miscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	Product is considered stable and 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	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Side effects of the inhalation of cobalt and its compounds may include flushing of the face and ringing in the ears (tinnitus). Cobalt inhalation can be lethal in animals if exposure is sufficiently high or prolonged. The acute LC50 for a 30-minute inhalation exposure in rats was 165 mg cobalt/m3 as cobalt hydrocarbonyl. Exposure to 9 mg cobalt/m3 as cobalt hydrocarbonyl for 6 hours/day, 5 days/week for 3 months resulted in 16 deaths out of 75 rats. Death was reported in rats and mice exposed to 19 mg cobalt/m3 (but not 1.9 mg cobalt/m3) as cobalt sulfate over 16 days, but exposure to 11.4 mg cobalt/m3 over 13 weeks was lethal only to mice and not to rats. Exposure to 1.14 mg cobalt/m3 as cobalt sulfate for 104 weeks resulted in no increase in mortality in rats and mice of either sex. Inhalation of stable cobalt by humans and/or animals resulted in respiratory, cardiovascular, hematological, hepatic, renal, endocrine, ocular, and body weight effects. As with exposures in humans, exposures of animals to cobalt-containing aerosols have resulted in pronounced respiratory effects. Animals exposed to aerosols of cobalt oxides and cobalt sulfate developed respiratory effects that varied in severity with exposure level and duration. A single 30-minute exposure of rats to relatively high levels (26-236 mg cobalt/m3 as cobalt hydrocarbonyl) resulted in congestion, edema, and hemorrhage of the lung. Prolonged exposure (3-4 months) of rats and rabbits to mixed cobalt oxides (0.4-9 mg cobalt/m3) resulted in lesions in the alveolar region of the respiratory tract characterised histologically by nodular accumulation of Type II epithelial cells, accumulations of enlarged highly vacuolated macrophages, interstitial inflammation, and fibrosis In at least one instance, the lesions appeared to regress when exposure was terminated . Guinea pigs sensitized to cobalt by repeated dermal application and then exposed to 2.4 mg cobalt/m3 as cobalt chloride showed pulmonary inflammatory changes (altered BAL fluid recovery, increased neutrophils and eosinophils in the recovered BAL fluid) that were different than those in exposed animals not sensitised to cobalt. Decreased lung compliance was found in pigs exposed to 0.1 mg cobalt/m3 as cobalt dust for 3 months. Lifetime exposure of hamsters to 7.9 mg cobalt/m3 as cobalt oxide resulted in emphysema. Necrosis and inflammation of the respiratory tract epithelium (nasal turbinates, larynx, trachea, bronchioles) were reported in rats exposed to 19 mg cobalt/m3 and mice exposed to 1.9 mg cobalt/m3 or greater as cobalt sulfate over 16 days. Exposure of rats and mice to cobalt as cobalt sulfate for 13 weeks resulted in adverse effects on all parts of the respiratory tract, with the larynx being the most sensitive part
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. Cobalamins are absorbed from the gastrointestinal tract but may be irregularly absorbed when given in large therapeutic doses. Absorption is impaired in the absence of Castles Intrinsic Factor. Cobalamins are stored in the liver, excreted in the bile and undergo some hepatoenteric recirculation; part of the dose is excreted in the urine. Studies have shown that soluble cobalt compounds are generally more acutely toxic than insoluble cobalt compounds. When expressed in terms of the cobalt ion for the sake of comparison, however, the differences in lethality values from the available studies are within an order of magnitude Animal test indicate an increase in red blood cells (polycythaemia) following the absorption of cobalt salts. [ICI] In toxic doses soluble cobalt salts act locally on the gastro-intestinal tract to produce pain and vomiting. Systemic effects in man include a peculiar vasodilation (flushing) of the face and ears, mild hypotension, rash, tinnitus (ringing in the ears) and nerve deafness. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products] Nicotinic acid and several of its derivatives, have a vasodilatory action. When given by mouth or by injection, in therapeutic doses, it may produce transient flushing of the face, a sensation of heat and a pounding in the head. High doses may cause flushing and dryness of the skin, skin lesions, abdominal cramps, diarrhoea, nausea, vomiting, malaise, anorexia, activation of peptic ulcer, jaundice and impairment of liver function, decrease in glucose tolerance, mild diabetes and hyperuricaemia. Most of these effects subside with withdrawal of the drug.
Skin Contact	The material may accentuate any pre-existing dermatitis condition Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions. Irritation and skin reactions are possible with sensitive skin Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.

	The material produces mild skin irritation; evidence exists, or practical experience predicts, that the material either ▸ produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or ▸ produces significant, but mild, 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.
Eye	Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Strong evidence exists that the substance may cause irreversible but non-lethal mutagenic effects following a single exposure. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. 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. For niacin (Vitamin B3, Vitamin PP) and its derivatives (including nicotinates and nicotinamides): Prescription niacin was shown to cause hepatotoxicity and increase risk of type 2 diabetes. Niacin is contraindicated for people with either active or a past history of liver disease because is has been associated with instances of serious, on occasion fatal, liver failure. Niacin is contraindicated for people with existing peptic ulcer disease, or other bleeding problems because niacin lowers platelet count and interferes with blood clotting. Niacin is excreted into human milk, but the amount and potential for adverse effects in the nursing infant are not known. As the precursor for nicotinamide adenine dinucleotide (NAD) and nicotinamide adenine dinucleotide phosphate (NADP), niacin is involved in DNA repair. Systematic reviews found no effect of prescription niacin on all-cause mortality, cardiovascular mortality, myocardial infarctions, nor fatal or non-fatal strokes despite raising HDL cholesterol. Reported side effects include an increased risk of new-onset type 2 diabetes The most common adverse effects of medicinal niacin (500-3000 mg) are flushing (e.g., warmth, redness, itching or tingling) of the face, neck and chest, headache, abdominal pain, diarrhea, dyspepsia, nausea, vomiting, rhinitis, pruritus and rash.These can be minimized by initiating therapy at low dosages, increasing dosage gradually, and avoiding administration on an empty stomach. The acute adverse effects of high-dose niacin therapy (1-3 grams per day) – which is commonly used in the treatment of hyperlipidemias – can further include hypotension, fatigue, glucose intolerance and insulin resistance, heartburn, blurred or impaired vision, and macular edema. With long-term use, the adverse effects of high-dose niacin therapy (750 mg per day) also include liver failure (associated with fatigue, nausea, and loss of appetite), hepatitis, and acute liver failure these hepatotoxic effects of niacin occur more often when extended-release dosage forms are used. The long-term use of niacin at greater than or equal to 2 grams per day also significantly increases the risk of cerebral hemorrhage, ischemic stroke, gastrointestinal ulceration and bleeding, diabetes, dyspepsia, and diarrhea. Flushing – a short-term dilatation of skin arterioles, causing reddish skin colour – usually lasts for about 15 to 30 minutes, although sometimes can persist for weeks. Typically, the face is affected, but the reaction can extend to neck and upper chest. The cause is blood vessel dilation] due to elevation in prostaglandin GD2 (PGD2) and serotonin. Flushing is sometimes accompanied by a prickly or itching sensation, in particular, in areas covered by clothing. Prevention of flushing requires altering or blocking the prostaglandin-mediated pathway. Aspirin taken half an hour before the niacin prevents flushing, as does ibuprofen. Taking niacin with meals also helps reduce this side effect. Acquired tolerance will also help reduce flushing; after several weeks of a consistent dose, most people no longer experience flushing. Slow- or "sustained"-release forms of niacin have been developed to lessen these side effects. Niacin in medicinal doses can cause modest elevations in serum transaminase and unconjugated bilirubin, both biomarkers of

liver injury. The increases usually resolves even when drug intake is continued. However, less commonly, the sustained release form of the drug can lead to serious hepatotoxicity, with onset in days to weeks. Early symptoms of serious liver damage include nausea, vomiting and abdominal pain, followed by jaundice and pruritus. The mechanism is thought to be a direct toxicity of elevated serum niacin. Lowering dose or switching to the immediate release form can resolve symptoms. In rare instances the injury is severe, and progresses to liver failure.
The high doses of niacin used to treat hyperlipidemia have been shown to elevate fasting blood glucose in people with type 2 diabetes. Long-term niacin therapy was also associated with an increase in the risk of new-onset type 2 diabetes.
High doses of niacin can also cause niacin maculopathy, a thickening of the macula and retina, which leads to blurred vision and blindness. This maculopathy is reversible after niacin intake ceases. Niaspan, the slow-release product, has been associated with a reduction in platelet content and a modest increase in prothrombin time.
In general, available cohort studies in humans have not reported a significant increase in total mortality as a result of cobalt exposure. Several studies have noted increased mortality rates resulting from lung cancer following occupational exposure to cobalt, either as a mixture of cobalt compounds or as hard metal, a metal alloy with a tungsten carbide and cobalt matrix. Fatal cases of hard metal disease and cardiomyopathy believed to have resulted from occupational cobalt exposure have also been reported. However, in the majority of these and other reported occupational studies, co-exposure to other substances was common, and was unable to be corrected for in the analysis.
The effects of chronic occupational exposure to cobalt and cobalt compounds on the respiratory system in humans are well-documented. These effects include respiratory irritation, diminished pulmonary function, wheezing, asthma, pneumonia, and fibrosis and occurred at exposure levels ranging from 0.007 to 0.893 mg cobalt/m3 (exposure from 2 to 17 years). These effects have been observed in workers employed in cobalt refineries, as well as hard metal workers, diamond polishers, and ceramic dish painters (painting with cobalt blue dye).
Occupational asthma attributed to the inhalation of cobalt powder has been confirmed following bronchial challenge tests. Chest tightness and chronic bronchitis have been recorded in hard-metal workers exposed to cobalt. Cobalt is known to function as a hapten, resulting in the generation of antibodies against cobalt-protein complexes. Although the minimum exposure level associated with cobalt sensitisation has not been determined, sensitisation has been demonstrated in hard metal workers with work-related asthma who have experienced prolonged occupational exposure (>3 years) to levels ranging from 0.007 to 0.893 mg cobalt/m3. The sensitisation phenomenon includes the production of IgE and IgA antibodies to cobalt. Exposure to inhaled cobalt chloride aerosols can precipitate an asthmatic attack in sensitised individuals believed to be the result of an allergic reaction within the lungs.
Allergic dermatitis of an erythematous papular type may also occur following occupational exposure. Dermatitis is a common result of dermal exposure to cobalt in humans that has been verified in a large number of studies. Using patch tests and intradermal injections, it has been demonstrated that the dermatitis is probably caused by an allergic reaction to cobalt. Contact allergy was reported in 22 of 223 (9.9%) nurses who were tested with a patch test of 1.0% cobalt chloride as well as 16 of 79 (20.3%) of examined dentists. Persons with body piercings showed an increased prevalence of allergy to cobalt, with the incidence of contact allergy being proportional to number of piercings The prevalence of sensitivity to cobalt following exposure to cobalt as a component of metal implants is low, with only 3.8% of patients developing a new sensitivity to cobalt following insertion of the implant
Exposure levels associated with the development of dermatitis have not been identified. It appears that the allergic properties of cobalt result mainly from exposure to the metal itself, rather than a salt, as it has been demonstrated that daily repeated exposure to aqueous cobalt salts did not result in hand eczema in patients known to have cobalt allergy.
Occupational exposure to cobalt in humans has been reported to cause several effects on the nervous system, including memory loss , nerve deafness, and a decreased visual acuity. It should be noted though, that both of the studies reporting on these findings, had small numbers of subjects, and exposure characterization was not reported.
Chronic exposure to cobalt produces polycythaemia (increase in blood haemoglobin), increased production of cells of the bone marrow and thyroid gland, pericardial effusion and damage to the alpha cells of the pancreas. Chronic exposure to cobalt compounds may result in pericardial effusion, polycardial effusion, cardiac failure, vomiting, convulsions and thyroid enlargement.
Chronic administration of cobaltous chloride has produced goiter, reduced thyroid activity and lowered synthesis rates and levels of cytochrome P-450, an enzymatic system responsible for chemical detoxification, in the liver. A toxic nephritis (kidney disease) may also develop.
Epidemic cardiomyopathy (heart disease) among heavy beer drinkers in the 1960's in Canada, the USA and Belgium has been attributed to the addition of up to 1.5 ppm of cobalt as a foam restorative and stabiliser. Other factors are probably implicated as therapeutic doses of cobalt, up to 50 mg/day (in the treatment of refractory anaemias) do not produce this effect. Inadequate protein or vitamin intake amongst heavy drinkers, or the effects of alcohol in rendering the heart more susceptible to disease may be important.
Single and repeated subcutaneous or intramuscular injection of cobalt powder and salts to rats may cause sarcoma at the injection site but evidence for carcinogenicity by any other route of exposure does not exist. A number of single cases of malignant tumours, mostly sarcomas, have been reported at the site of orthopedic implants containing cobalt.
Animals, exposed to cobalt compounds also exhibit an increase in respiration, as well as tremor and convulsion. Exposure of rats and mice to aerosols of cobalt (as cobalt sulfate) at concentrations from 0.11 to 1.14 mg cobalt/m3 for 2 years resulted in a spectrum of inflammatory, fibrotic, and proliferative lesions in the respiratory tract of male and female rats and mice. Squamous metaplasia of the larynx occurred in rats and mice at exposure concentrations of .0.11 mg cobalt/m3, with severity of the lesion increasing with increased cobalt concentration. Hyperplastic lesions of the nasal epithelium occurred in rats at concentrations of .0.11 mg cobalt/3, and in mice at concentrations of .0.38 mg cobalt/m3. Both sexes of rats had greatly increased incidences (>90% incidence) of alveolar lesions at all exposure levels, including inflammatory changes, fibrosis, and metaplasia. Similar changes were seen in mice at all exposure levels, though the changes in mice were less severe.
Cobalt metal dust inhalations by miniature swine resulted in early marked decrease in lung compliance and increases in septal collagen. After a one-week "sensitising period", followed by a 10-day lapse period, further exposures resulted in wheezing produced by hypersensitivity reactions.
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, phenobarbitol 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 On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Though the cobalamins are generally well tolerated, allergic hypersensitivity reactions have followed the administration of the Vitamin B12 factors, cyanocobalamin and hydroxocobalamin. Vitamin B12 rapidly increases the rate of cell maturation, in vivo, and as a consequence increases the rate of nucleic acid degradation which in turn increases blood uric acid levels; this may produce gout in susceptible individuals. 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). Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.

Avian B	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
niacinamide	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[2]	Not Available
	Inhalation(Rat) LC50: >3.8 mg/l4h[1]	
	Oral (Rat) LD50: >2500 mg/kg[1]	
sodium chloride	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >10000 mg/kg[1]	Eye (rabbit): 10 mg - moderate
	Inhalation(Rat) LC50: >10.5 mg/l4h[1]	Eye (rabbit):100 mg/24h - moderate
	Oral (Rat) LD50: 3000 mg/kg[2]	Skin (rabbit): 500 mg/24h - mild
potassium chloride	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: 2600 mg/kg[2]	Eye (rabbit): 500 mg/24h - mild
ascorbic acid	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: 11900 mg/kg[2]	Not Available
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]
folic acid	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >8000 mg/kg[2]	Not Available
cyanocobalamin	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
riboflavin	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >10000 mg/kg[2]	Eye (rabbit): non-irritant (Draize) *BASF
		Skin (rabit): non-irritant (Draize) *
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

Avian B	Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.
NIACINAMIDE	Mutation in microorganisms 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.. 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 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.
SODIUM CHLORIDE	The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. 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.
FOLIC ACID	The risk of toxicity from folic acid is low, because folate is a water-soluble vitamin and is regularly removed from the body through urine. One potential issue associated with high doses of folic acid is that it has a masking effect on the diagnosis of pernicious anaemia due to vitamin B12 deficiency, and may even precipitate or exacerbate neuropathy in vitamin B12-deficient individuals. This evidence justified development of a Tolerable Upper Intake Level(UL) for folate. In general, ULs are set for vitamins and minerals when evidence is sufficient. The adult UL of 1,000 µg for folate (and lower for children) refers specifically to folic acid used as a supplement, as no health risks have been associated with high intake of folate from food sources. The EFSA reviewed the safety question and agreed with United States that the UL be set at 1,000 ug.] The Japan National Institute of Health and Nutrition set the adult UL at 1,300 or 1,400 ug depending on age. Reviews of clinical trials that called for long-term consumption of folic acid in amounts exceeding the UL have raised concerns. One theory is that consumption of large amounts of folic acid leads to detectable amounts of unmetabolized folic acid circulating in blood because the enzyme dihydrofolate reductase that converts folic acid to the biologically active forms is rate limiting. Evidence of a negative health effect of folic acid in blood is not consistent, and folic acid has no known cofactor function that would increase the likelihood of a causal role for free FA in disease development. However, low vitamin B12 status in combination with high folic acid intake, in addition to the previously mentioned neuropathy risk, appeared to increase the risk of cognitive impairment in the elderly. Long-term use of folic acid dietary supplements in excess of 1,000 ug/day has been linked to an increase in prostate cancer risk. An excess of dietary folate may interfere with neurodevelopmental metabolism, increasing the risk of adverse outcomes, including autism spectrum disorder (ASD). It has been suggested that folate affects connectivity among neurons as the brain develops. Glutamate is important in the regulation of neural tissue development, as it is a common excitatory neurotransmitter that binds to synaptic membranes. Because it is so structurally similar to folic acid, it may compete for binding sites on neurons within developing tissues, affecting connectivity of neurons during embryonic brain development. In vitro data suggests that excess glutamate can overcome effects of excess folate, which authenticates a mechanism of inhibition of neural development by excess folate. . Folate deficiency can be caused by unhealthy diets that do not include enough vegetables and other folate-rich foods; diseases in which folates are not well absorbed in the digestive system (such as Crohn's disease or celiac disease); some genetic disorders that affect levels of folate; and certain medicines (such as phenytoin, sulfasalazine, or trimethoprim-sulfamethoxazole). Folate deficiency is accelerated by alcohol consumption, possibly by interference with folate transport. Folate deficiency may lead to glossitis, diarrhea, depression, confusion, anemia, and fetal neural tube and brain defects. Other symptoms include fatigue, gray hair, mouth sores, poor growth, and swollen tongue. Folate deficiency is diagnosed by analyzing a complete blood count (CBC) and plasma vitamin B12 and folate levels. A serum folate of 3 µg/L or lower indicates deficiency. Serum folate level reflects folate status, but erythrocyte folate level better reflects tissue stores after intake. An erythrocyte folate level of 140 µg/L or lower indicates inadequate folate status. Serum folate reacts more rapidly to folate intake than erythrocyte folate Since folate deficiency limits cell division, erythropoiesis (production of red blood cells) is hindered. This leads to megaloblastic anemia, which is characterized by large, immature red blood cells. This pathology results from persistently thwarted attempts at normal DNA replication, DNA repair, and cell division, and produces abnormally large red cells called megaloblasts (and hypersegmented neutrophils) with abundant cytoplasm capable of RNA and protein synthesis, but with clumping and fragmentation of nuclear chromatin. Some of these large cells, although immature (reticulocytes), are released early from the marrow in an attempt to compensate for the anemia Both adults and children need folate to make normal red and white blood cells and prevent anemia, which causes fatigue, weakness, and inability to concentrateIncreased homocysteine levels suggest tissue folate deficiency, but homocysteine is also affected by vitamin B12 and vitamin B6, renal function, and genetics. One way to differentiate between folate deficiency and vitamin B12 deficiency is by testing for methylmalonic acid (MMA) levels. Normal MMA levels indicate folate deficiency and elevated MMA levels indicate vitamin B12 deficiency. Folate deficiency is treated with supplemental oral folic acid of 400 to 1000 µg per day. This treatment is very successful in replenishing tissues, even if deficiency was caused by malabsorption. People with megaloblastic anemia need to be tested for vitamin B12 deficiency before treatment with folic acid, because if the person has vitamin B12 deficiency, folic acid supplementation can remove the anemia, but can also

	worsen neurologic problems. Cobalamin deficiency may lead to folate deficiency, which, in turn, increases homocysteine levels and may result in the development of cardiovascular disease or birth defects.
CYANOCOBALAMIN	Oral (several) species: LD50 >5000 mg/kg* Nil reported Reproductive effector in rats
RIBOFLAVIN	Hamster cell mutagen. * BASF MSDS
Avian B & NIACINAMIDE & SODIUM CHLORIDE & ASCORBIC ACID & PYRIDOXINE HYDROCHLORIDE & RIBOFLAVIN	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.
Avian B & NIACINAMIDE	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested. 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. 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 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 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 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 PPARgamma 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. 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 (PPARgamma), resulting in reverse cholesterol transport. 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. 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 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 multiplications, 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.
NIACINAMIDE & POTASSIUM CHLORIDE	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

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

Avian B

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

niacinamide

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	72h	Algae or other aquatic plants	560mg/l	1
LC50	96h	Fish	>1000mg/l	2

sodium chloride

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	6h	Fish	0.001mg/l	4
EC50	96h	Algae or other aquatic plants	1110.36mg/L	4
EC50	72h	Algae or other aquatic plants	20.76-36.17mg/L	4
LC50	96h	Fish	1000mg/l	4
EC50	48h	Crustacea	0.00439-0.00565mg/l	4

potassium chloride

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	25h	Fish	9.319mg/L	4
EC50	96h	Algae or other aquatic plants	894.6mg/L	4
EC50	72h	Algae or other aquatic plants	>100mg/l	2

	LC50	96h	Fish	390mg/l	4
	EC50	48h	Crustacea	93mg/l	4

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

pyridoxine hydrochloride	Endpoint	Test Duration (hr)	Species	Value	Source
	EC10(ECx)	72h	Algae or other aquatic plants	3.3mg/l	2
	EC50	72h	Algae or other aquatic plants	72mg/l	2
	LC50	96h	Fish	>100mg/l	2
	EC50	48h	Crustacea	>100mg/l	2

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

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

riboflavin	Endpoint	Test Duration (hr)	Species	Value	Source
	EC10(ECx)	48h	Algae or other aquatic plants	3.3-9mg/l	4
	LC50	96h	Fish	10000mg/l	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*

for cobalt compounds:

Environmental Fate:

Cobalt strongly binds to humic substances naturally present in aquatic environments. Humic acids can be modified by UV light and bacterial decomposition, which may change their binding characteristics over time. The lability of the complexes is strongly influenced by pH, the nature of the humic material, and the metal-to-humic substance ratio. The lability of cobalt-humate complexes decreases in time (“aging effect”). The “aging effect” indicates that after a period of time (~12 hours), complexes that were initially formed are transformed into stronger ones from which the metal ion is less readily dislodged.

Between 45 and 100% of dissolved cobalt was found to occur in very strong complexes. The distribution coefficient of cobalt may vary considerably in the same sediment in response to conditions affecting the pH, redox conditions, ionic strength, and amount of dissolved organic matter. Uptake of 60Co from the water by sediment increased rapidly as the pH was increased from 5 to 7-7.5 and then slightly decrease. Therefore, pH would be an important factor affecting the migration of cobalt in surface water. Uptake was little affected by changes in liquid-to-solids ratio and ionic strength. 60Co is more mobile in anaerobic marine aquatic environments than in freshwater aerobic ones. In seawater sediment systems under anaerobic conditions 60Co was 250 times more mobile than 60Co in freshwater sediment systems under aerobic conditions. Under anaerobic conditions, 30% of the 60Co added to a sediment-freshwater system was "exchangeable" and therefore potentially mobile, while under aerobic conditions, 98% of the 60Co was permanently fixed. Most of the mobile 60Co produced under anaerobic conditions in seawater consisted of nonionic cobalt associated with low molecular weight organic substances that were stable to changes in pH; the exchangeable 60Co appeared to be mostly ionic.

The mobility of cobalt in soil is inversely related to how strongly it is adsorbed by soil constituents. Cobalt may be retained by mineral oxides such as iron and manganese oxide, crystalline materials such as aluminosilicate and goethite, and natural organic substances in soil. Sorption of cobalt to soil occurs rapidly (within 1-2 hours). Soil-derived oxide materials were found to adsorb greater amounts of cobalt than other materials examined, although substantial amounts were also adsorbed by organic materials.

Clay minerals sorbed relatively smaller amounts of cobalt. In addition, little cobalt was desorbed from soil oxides while substantial amounts desorbed from humic acids and montorillonite. In clay soil, adsorption may be due to ion exchange at the cationic sites on clay with either simple ionic cobalt or hydrolysed ionic species such as CoOH+. Adsorption of cobalt onto iron and manganese increases with pH. In addition, as pH increases, insoluble hydroxides or carbonates may form, which would also reduce cobalt mobility. Conversely, sorption onto mobile colloids would enhance its mobility. In most soils, cobalt is more mobile than lead, chromium (II), zinc, and nickel, but less mobile than cadmium. In several studies, the Kd of cobalt in a variety of soils ranged from 0.2 to 3,800. The soil properties showing the highest correlation with Kd were exchangeable calcium, pH, water content, and cation exchange capacity. Organic complexing agents such as ethylenediaminetetraacetic acid (EDTA), which are used for decontamination operations at nuclear facilities, greatly enhance the mobility of cobalt in soil. Other organic complexing agents, such as those obtained from plant decay, may also increase cobalt mobility in soil. However, both types of complexes decrease cobalt uptake by plants. Addition of sewage sludge to soil also increases the mobility of cobalt, perhaps due to organic complexation of cobalt.

Cobalt may be taken up from soil by plants. Surface deposition of cobalt on leaves of plants from airborne particles may also occur. Elevated levels of cobalt have been found in the roots of sugar beets and potato tubers in soils with high cobalt concentrations (e.g., fly ash-amended soil) due to absorption of cobalt from soil. However, the translocation of cobalt from roots to above-ground parts of plants is not significant in most soils, as indicated by the lack of cobalt in seeds of barley, oats, and wheat grown in high-cobalt soil. However, in highly acidic soil (pH as low as 3.3), significantly higher than normal concentrations of cobalt were found in rye grass foliage, oats, and barley. For example, cobalt concentrations in rye grass grown in unlimed soil (pH<5.0) was 19.7 mg/kg compared with 1.1 mg/kg in rye grass grown in limed soil (pH>5.0). Soil and plant samples taken in the 30-km zone around Chernobyl indicated that 60Co was not accumulated by plants and mushrooms. Studies investigating the uptake of 60Co by tomato plants watered with 60Co contaminated water showed that tomato plants absorbed <2% of the activity available from the soil.

60Co is taken up by phytoplankton and unicellular algae (Senenastrum capricornutum) with concentration factors (dry weight) ranging from 15,000 to 40,000 and

2,300 to 18,000, respectively. Elimination experiments with the algae indicate a two component biological half-life, 1 hour and 11 days, respectively, and suggest that the cobalt might be absorbed not only on the surface, but also intracellularly. Since these organisms are at the bottom of the food chain, they could play an important role in the trophic transfer of 60Co released into waterways by nuclear facilities. However, cobalt levels generally diminish with increasing trophic levels in a food chain. The low levels of cobalt in fish may also reflect cobalt's strong binding to particles and sediment. The bioaccumulation factors (dry weight basis) for cobalt in marine and freshwater fish are ~100-4,000 and <10-1,000, respectively; accumulation in the muscle of marine fish is 5- 500.
Cobalt largely accumulates in the viscera and on the skin, as opposed to the edible parts of the fish. In carp, accumulation from water accounted for 75% of 60Co accumulated from both water and food; accumulation from water and food was additive. Depuration half-lives were 53 and 87 days for fish contaminated from food and water, respectively. In the case of an accidental release of 60Co into waterways, the implication is that effects would manifest themselves rapidly since the primary route of exposure is from water rather than food. Uptake of 60Co was very low in whitefish, with concentrations being highest in kidney and undetectable in muscle. Similarly, while accumulation of 60Co by carp from food was dependent on food type, the transfer factor was very low, approximately 0.01, and no long-term bioaccumulation of the radionuclide occurred.
Concentration factors have also been reported for various other aquatic organisms. Freshwater mollusks have concentration factors of 100-14,000 (~1-300 in soft tissue). Much of the cobalt taken up by mollusks and crustacae from water or sediment is adsorbed to the shell or exoskeleton; very little cobalt is generally accumulated in the edible parts. A concentration factor for 60Co of 265 mL/g (wet weight) was determined for Daphnia magna in laboratory studies. The rapid decrease in radioactivity during the depuration phase indicated that adsorption to the surface was the major contamination process. However, the digestive glands of crustaceans, which are sometimes eaten by humans, may accumulate high levels of 60Co. The shell accounted for more than half of the body burden. Among the soft tissue, the gills and viscera had the highest concentrations factors and the muscle had the lowest.
In mussels, higher absorption efficiencies and lower efflux rates were obtained for cobalamins than for inorganic cobalt, suggesting that it is a more bioavailable form of cobalt.
Vitamin B12, which contains cobalt, is synthesized by 58 species of seven genuses of bacteria as well as blue-green algae and actinomycetes (mold-like bacteria). Consequently, vitamin B12 levels in marine water range from very low levels in some open ocean water to much higher levels in some coastal waters. Freshwater environments have comparable levels of vitamin B12. The high level of cobalamins in coastal water appears to be elated to the occurrence of macrophytes in these areas with their high concentrations of vitamin B12. Cobalamins are released into the water when the organisms die.
Some female birds sequester metals into their eggs under certain conditions, a phenomenon that may jeopardize the developing embryos.
Pyridine and its derivatives:
Environmental fate:
The atmospheric photodegradation estimates for the Pyridine and Pyridine Derivatives Category chemicals indicate that piperidine which is the lower molecular weight, non-aromatic and unsubstituted chemical in the category, would be expected to degrade rapidly (t1/2 < 1 day) when exposed to UV light in the atmosphere. Pyridine and the three methyl derivatives of pyridine (picolines) which are the higher molecular weight, aromatic and substituted chemicals in the category, would be expected to photodegrade more slowly (t1/2 . 30 or 10 days, respectively). , Lutidines, and collidines are expected to photodegrade even more slowly. The nitriles derivatives of pyridine are also predicted to photodegrade more slowly (t1/2 . 164 days). However, the nitrile derivatives of pyridine were predicted to partition to air much less favorably than to soil and water. As molecular weight and substitution increase in the category, greater distribution to water and soil and less to air is predicted. This trend is consistent with the vapor pressure data.
Pyridines are not expected to hydrolysis in the environment because they lack a potentially hydrolysable functional group.
There are adequate measured data across the pyridine group to allow the conclusion that these chemicals are biodegradable in the presence of adequate oxygen and bacteria; however, they are relatively stable under anaerobic and/or sterile environments**.**
Depending upon the environmental conditions, different types of bacteria, fungi, and enzymes are involved in the degradation process of these compounds .
Different organisms are using different pathways to biotransform a substrate The transformation rate of the pyridine derivatives is dependent on the substituents .
For example, pyridine carboxylic acids have the highest transformation rate followed by mono-hydroxypyridines, methylpyridines, aminopyridines, and halogenated pyridines
Ecotoxicity:
Measured values for acute aquatic toxicity indicate that the Pyridine and Pyridine Derivatives Category chemicals are slightly to moderately toxic to fish, invertebrates and algae. Modeled data for acute aquatic toxicity were generally consistent with the reliable measured values in cases for which both existed.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
niacinamide	HIGH	HIGH
sodium chloride	LOW	LOW
potassium chloride	HIGH	HIGH
ascorbic acid	LOW	LOW
pyridoxine hydrochloride	LOW	LOW
folic acid	HIGH	HIGH
cyanocobalamin	HIGH	HIGH
riboflavin	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
niacinamide	LOW (LogKOW = -0.37)
sodium chloride	LOW (LogKOW = 0.5392)
potassium chloride	LOW (LogKOW = -0.4608)
ascorbic acid	LOW (LogKOW = -1.85)
pyridoxine hydrochloride	LOW (LogKOW = -0.557)
folic acid	LOW (LogKOW = -1.9983)

Ingredient	Bioaccumulation
cyanocobalamin	LOW (LogKOW = -12.1962)
riboflavin	LOW (LogKOW = -1.46)

Mobility in soil

Ingredient	Mobility
niacinamide	LOW (KOC = 51.56)
sodium chloride	LOW (KOC = 14.3)
potassium chloride	LOW (KOC = 14.3)
ascorbic acid	LOW (KOC = 10)
pyridoxine hydrochloride	LOW (KOC = 10)
folic acid	LOW (KOC = 647.4)
cyanocobalamin	LOW (KOC = 10000000000)
riboflavin	LOW (KOC = 325.8)

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. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO

Shipping container and transport vehicle placarding and labeling may vary from the below information. Products that are regulated for transport will be packaged and marked as Dangerous Goods in Excepted Quantities according to US DOT, IATA and IMDG regulations. In case of reshipment, it is the responsibility of the shipper to determine the appropriate labels and markings in accordance with applicable transport regulations.

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

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

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

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
niacinamide	Not Available
sodium chloride	Not Available
potassium chloride	Not Available
ascorbic acid	Not Available
pyridoxine hydrochloride	Not Available

Product name	Group
folic acid	Not Available
cyanocobalamin	Not Available
riboflavin	Not Available

Transport in bulk in accordance with the IGC Code

Product name	Ship Type
niacinamide	Not Available
sodium chloride	Not Available
potassium chloride	Not Available
ascorbic acid	Not Available
pyridoxine hydrochloride	Not Available
folic acid	Not Available
cyanocobalamin	Not Available
riboflavin	Not Available

SECTION 15 Regulatory information

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

niacinamide is found on the following regulatory lists

US DOE Temporary Emergency Exposure Limits (TEELs)
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

sodium chloride is found on the following regulatory lists

US DOE Temporary Emergency Exposure Limits (TEELs)
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

potassium chloride is found on the following regulatory lists

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

ascorbic acid is found on the following regulatory lists

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

pyridoxine hydrochloride is found on the following regulatory lists

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US Toxicology Excellence for Risk Assessment (TERA) Workplace Environmental Exposure Levels (WEEL)

folic acid is found on the following regulatory lists

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
US - Alaska Air Quality Control - Concentrations Triggering an Air Quality Episode for Air Pollutants Other Than PM-2.5
US NIOSH Recommended Exposure Limits (RELs)
US OSHA Permissible Exposure Limits (PELs) Table Z-1
US OSHA Permissible Exposure Limits (PELs) Table Z-3
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

cyanocobalamin is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
US - California Hazardous Air Pollutants Identified as Toxic Air Contaminants
US Clean Air Act - Hazardous Air Pollutants
US EPA Integrated Risk Information System (IRIS)
US EPCRA Section 313 Chemical List
US National Toxicology Program (NTP) 15th Report Part B. Reasonably Anticipated to be a Human Carcinogen
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

riboflavin is found on the following regulatory lists

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
US - Alaska Air Quality Control - Concentrations Triggering an Air Quality Episode for Air Pollutants Other Than PM-2.5
US Clean Air Act - Hazardous Air Pollutants
US NIOSH Recommended Exposure Limits (RELs)
US OSHA Permissible Exposure Limits (PELs) Table Z-1
US OSHA Permissible Exposure Limits (PELs) Table Z-3
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

Federal Regulations

Superfund Amendments and Reauthorization Act of 1986 (SARA)

Section 311/312 hazard categories

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

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

None Reported

State Regulations

US. California Proposition 65

None listed

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (niacinamide; sodium chloride; potassium chloride; ascorbic acid; pyridoxine hydrochloride; folic acid; cyanocobalamin; riboflavin)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (cyanocobalamin)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	No (folic acid; cyanocobalamin)
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	14/06/2023
Initial Date	15/06/2023

Other information

Ingredients with multiple cas numbers

Name	CAS No
sodium chloride	7647-14-5, 14762-51-7, 16887-00-6, 8028-77-1
ascorbic acid	50-81-7, 129940-97-2, 14536-17-5, 154170-90-8, 259133-78-3, 30208-61-8, 50976-75-5, 56172-55-5, 56533-05-2, 57304-74-2, 57606-40-3, 623158-95-2, 882690-91-7, 884381-69-5, 885512-24-3, 88845-26-5, 89924-69-6
folic acid	59-30-3, 75708-92-8

Classification of the preparation and its individual components has drawn on official and authoritative sources 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