Keymix Abdextra Key 94 Concentrated Soluble Multi-vitamins for Poultry

International Animal Health Products Pty Ltd

Chemwatch Hazard Alert Code: 3

Chemwatch: **36-3842**
Version No: **9.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Initial Date: **25/07/2013**
Revision Date: **21/05/2026**
Print Date: **21/10/2026**
S.GHS.AUS.EN.E

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

Product Identifier

Product name	Keymix Abdextra Key 94 Concentrated Soluble Multi-vitamins for Poultry
Chemical Name	Not Applicable
Synonyms	Abdextra
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	A concentrated soluble multi-vitamins for poultry. Add to drinking water. Medicated water should be consumed within 24 hours. Do not use disinfectants in the same water as Abdextra.

Details of the manufacturer or importer of the safety data sheet

Registered company name	**International Animal Health Products Pty Ltd**
Address	18 Healey Circuit Huntingwood NSW 2148 Australia
Telephone	+61 2 9672 7944
Fax	+61 2 9672 7988
Website	www.iahp.com.au
Email	info@iahp.com.au

Emergency telephone number

Association / Organisation	**Australian Poison Information Centre**
Emergency telephone number(s)	**13 11 26 (24 Hours)**
Other emergency telephone number(s)	**New Zealand: National Poisons Centre 0800 764 766 (24 hours)**

SECTION 2 Hazards identification

Classification of the substance or mixture

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

Poisons Schedule	Not Applicable
Classification [1]	Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 2A, Germ Cell Mutagenicity Category 2, Reproductive Toxicity Category 2
Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI***

Page 1 continued...

Label elements

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

Hazard statement(s)

H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H319	Causes serious eye irritation.
H341	Suspected of causing genetic defects.
H361fd	Suspected of damaging fertility. Suspected of damaging the unborn child.

Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

P202	Do not handle until all safety precautions have been read and understood.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing dust/fumes.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P302+P352	IF ON SKIN: Wash with plenty of water.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

P405	Store locked up.

Precautionary statement(s) Disposal

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

No further product hazard information.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
50-81-7	1-<10	ascorbic acid
98-92-0	1-<10	niacinamide
58-95-7	<5	D-alpha-tocopherol acetate
10101-89-0	<5	trisodium phosphate dodecahydrate
Not Available	balance	Ingredients determined not to be hazardous

Continued...

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

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	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 dust is inhaled, remove from contaminated area. ‣ Encourage patient to blow nose to ensure clear passage of breathing. ‣ If irritation or discomfort persists seek medical attention.
Ingestion	‣ Immediately give a glass of water. ‣ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 Firefighting measures

Extinguishing media

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

Special hazards arising from the substance or mixture

Continued...

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

Advice for firefighters

Fire Fighting	‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear breathing apparatus plus protective gloves. ‣ 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 solid which burns but propagates flame with difficulty; it is estimated that most organic dusts are combustible (circa 70%) - according to the circumstances under which the combustion process occurs, such materials may cause fires and / or dust explosions. ‣ Organic powders when finely divided over a range of concentrations regardless of particulate size or shape and suspended in air or some other oxidizing medium may form explosive dust-air mixtures and result in a fire or dust explosion (including secondary explosions). ‣ Avoid generating dust, particularly clouds of dust in a confined or unventilated space as dusts may form an explosive mixture with air, and any source of ignition, i.e. flame or spark, will cause fire or explosion. Dust clouds generated by the fine grinding of the solid are a particular hazard; accumulations of fine dust (420 micron or less) may burn rapidly and fiercely if ignited - particles exceeding this limit will generally not form flammable dust clouds; once initiated, however, larger particles up to 1400 microns diameter will contribute to the propagation of an explosion. ‣ In the same way as gases and vapours, dusts in the form of a cloud are only ignitable over a range of concentrations; in principle, the concepts of lower explosive limit (LEL) and upper explosive limit (UEL) are applicable to dust clouds but only the LEL is of practical use; - this is because of the inherent difficulty of achieving homogeneous dust clouds at high temperatures (for dusts the LEL is often called the "Minimum Explosible Concentration", MEC). ‣ When processed with flammable liquids/vapors/mists,ignitable (hybrid) mixtures may be formed with combustible dusts. Ignitable mixtures will increase the rate of explosion pressure rise and the Minimum Ignition Energy (the minimum amount of energy required to ignite dust clouds - MIE) will be lower than the pure dust in air mixture. The Lower Explosive Limit (LEL) of the vapour/dust mixture will be lower than the individual LELs for the vapors/mists or dusts. ‣ A dust explosion may release of large quantities of gaseous products; this in turn creates a subsequent pressure rise of explosive force capable of damaging plant and buildings and injuring people. ‣ Usually the initial or primary explosion takes place in a confined space such as plant or machinery, and can be of sufficient force to damage or rupture the plant. If the shock wave from the primary explosion enters the surrounding area, it will disturb any settled dust layers, forming a second dust cloud, and often initiate a much larger secondary explosion. All large scale explosions have resulted from chain reactions of this type. ‣ Dry dust can be charged electrostatically by turbulence, pneumatic transport, pouring, in exhaust ducts and during transport. ‣ Build-up of electrostatic charge may be prevented by bonding and grounding. ‣ Powder handling equipment such as dust collectors, dryers and mills may require additional protection measures such as explosion venting. ‣ All movable parts coming in contact with this material should have a speed of less than 1-meter/sec. ‣ A sudden release of statically charged materials from storage or process equipment, particularly at elevated temperatures and/ or pressure, may result in ignition especially in the absence of an apparent ignition source. ‣ One important effect of the particulate nature of powders is that the surface area and surface structure (and often moisture content) can vary widely from sample to sample, depending of how the powder was manufactured and handled; this means that it is virtually impossible to use flammability data published in the literature for dusts (in contrast to that published for gases and vapours). ‣ Autoignition temperatures are often quoted for dust clouds (minimum ignition temperature (MIT)) and dust layers (layer ignition temperature (LIT)); LIT generally falls as the thickness of the layer increases. Combustion products include: ‣ carbon monoxide (CO) ‣ carbon dioxide (CO2) nitrogen oxides (NOx) ‣ other pyrolysis products typical of burning organic material. **NOTE:** Burns with intense heat. Produces melting, flowing, burning liquid and dense acrid black smoke. May emit poisonous fumes. May emit corrosive fumes.
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Continued...

Minor Spills	‣ Clean up all spills immediately. ‣ Avoid breathing dust and contact with skin and eyes. ‣ Wear protective clothing, gloves, safety glasses and dust respirator. ‣ Use dry clean up procedures and avoid generating dust. ‣ Sweep up, shovel up or ‣ Vacuum up (consider explosion-proof machines designed to be grounded during storage and use). ‣ Place spilled material in clean, dry, sealable, labelled container.
Major Spills	Moderate hazard. ‣ **CAUTION**: Advise personnel in area. ‣ Alert Emergency Services and tell them location and nature of hazard. ‣ Control personal contact by wearing protective clothing. ‣ Prevent, by any means available, spillage from entering drains or water courses. ‣ Recover product wherever possible. ‣ **IF DRY:** Use dry clean up procedures and avoid generating dust. Collect residues and place in sealed plastic bags or other containers for disposal. **IF WET:** Vacuum/shovel up and place in labelled containers for disposal. ‣ **ALWAYS:** Wash area down with large amounts of water and prevent runoff into drains. ‣ If contamination of drains or waterways occurs, advise Emergency Services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	‣ Avoid skin contact, including inhalation. ‣ Wear protective clothing when risk of exposure occurs. ‣ Use in a well-ventilated area. ‣ Prevent concentration in hollows and sumps. ‣ **DO NOT** enter confined spaces until atmosphere has been checked. ‣ **DO NOT** allow material to come in direct contact with human skin or eyes. ‣ **DO NOT** allow material to come in contact with exposed food or food contact surfaces. ‣ Suitable PPE must be worn at all times. ‣ 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. ‣ Organic powders when finely divided over a range of concentrations regardless of particulate size or shape and suspended in air or some other oxidizing medium may form explosive dust-air mixtures and result in a fire or dust explosion (including secondary explosions) ‣ Minimise airborne dust and eliminate all ignition sources. Keep away from heat, hot surfaces, sparks, and flame. ‣ Establish good housekeeping practices. ‣ Remove dust accumulations on a regular basis by vacuuming or gentle sweeping to avoid creating dust clouds. ‣ Use continuous suction at points of dust generation to capture and minimise the accumulation of dusts. Particular attention should be given to overhead and hidden horizontal surfaces to minimise the probability of a "secondary" explosion. According to NFPA Standard 654, dust layers 1/32 in.(0.8 mm) thick can be sufficient to warrant immediate cleaning of the area. ‣ Do not use air hoses for cleaning. ‣ Minimise dry sweeping to avoid generation of dust clouds. Vacuum dust-accumulating surfaces and remove to a chemical disposal area. Vacuums with explosion-proof motors should be used. ‣ Control sources of static electricity. Dusts or their packages may accumulate static charges, and static discharge can be a source of ignition. ‣ Solids handling systems must be designed in accordance with applicable standards (e.g. NFPA including 654 and 77) and other national guidance. ‣ Do not empty directly into flammable solvents or in the presence of flammable vapors. ‣ The operator, the packaging container and all equipment must be grounded with electrical bonding and grounding systems. Plastic bags and plastics cannot be grounded, and antistatic bags do not completely protect against development of static charges. Empty containers may contain residual dust which has the potential to accumulate following settling. Such dusts may explode in the presence of an appropriate ignition source. ‣ Do NOT cut, drill, grind or weld such containers. ‣ In addition ensure such activity is not performed near full, partially empty or empty containers without appropriate workplace safety authorisation or permit.
Other information	‣ Store in original containers. ‣ Keep containers securely sealed. ‣ Store in a cool, dry area protected from environmental extremes.

Continued...

	‣ Store away from incompatible materials and foodstuff containers. ‣ Protect containers against physical damage and check regularly for leaks. ‣ Observe manufacturer's storage and handling recommendations contained within this SDS. For major quantities: ‣ Consider storage in bunded areas - ensure storage areas are isolated from sources of community water (including stormwater, ground water, lakes and streams}. ‣ Ensure that accidental discharge to air or water is the subject of a contingency disaster management plan; this may require consultation with local authorities.

Conditions for safe storage, including any incompatibilities

Suitable container	500g plastic container with water soluble bag. 25kg multiwalled paper bag with plastic liner. ‣ Polyethylene or polypropylene container. ‣ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	Reducing sugar-based material. Autooxidation of reducing sugars may produce up to 3000 ppm carbon monoxide under moderately alkaline conditions. High pH aqueous solutions of saccharides (aldoses, ketoses) or polysaccharides based on these sugars may generate hazardous atmospheres in confined spaces. Reducing sugars contain an aldehyde or free hemiacetal in the open-chain form. Sugars with ketone groups in their open chain form are capable of isomerising via a series of tautomeric shifts to produce an aldehyde group in solution. Therefore, ketone-bearing sugars like fructose are considered reducing sugars but it is the isomer containing an aldehyde group which is reducing since ketones cannot be oxidized without decomposition of the sugar. Many disaccharides, like lactose and maltose, also have a reducing form, as one of the two units may have an open-chain form with an aldehyde group. However, sucrose and trehalose, in which the anomeric carbons of the two units are linked together, are non-reducing disaccharides since neither of the rings is capable of opening. In glucose polymers such as starch and starch-derivatives like glucose syrup, maltodextrin and dextrin the macromolecule begins with a reducing sugar, a free aldehyde. More hydrolysed starch contains more reducing sugars. The percentage of reducing sugars present in these starch derivatives is called dextrose equivalent (DE). · Quinones may be converted to quinone methides by a number of mechanisms. · Quinone methides are structurally related to quinones with one of the carbonyl oxygens replaced by a methylene group. This structural change makes the molecule much more polarized and thus more reactive. · Simple quinone methides are short lived intermediates that are not stable enough to be isolated under normal circumstances but quickly react with nucleophiles and other reactants. · Quinone methides are electrophilic Michael acceptors that generally react quickly with nucleophiles, other reactants, and are readily reduced to hydroquinones. Quinone methides are conjugated but not aromatic. Conjugate addition usually breaks the conjugation. Reduction can either rearomatise the compound or break the conjugation · Unstable quinones may tautomerise to the methide · Nearly 200 naturally occurring quinones, many of them heterocyclic, have been shown to possess the structural features necessary for quinone methide formation · Photoreduction of benzylquinones, naphthaquinones and anthraquinones and their derivatives to dihydroquinones follows a common mechanism NOTE: Quinone methide derivatives form adducts with several proteins, including enzymes that protect cells from oxidative stress; this prooxidant state can also lead to cell oxidative damage. It must be noted that relationships between chronic oxidative stress and tumour promotion are well known . Dilute solutions of all sugars are subject to fermentation, either by yeast or by other microorganisms or enzymes derived from these, producing gases which can pressurise and burst sealed containers. Some microorganisms will produce hydrogen or methane, adding a fire and explosion hazard. Food grade materials must be protected from all possible contaminants ‣ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure. ‣ Local exhaust ventilation is required where solids are handled as powders or crystals; even when particulates are relatively large, a certain proportion will be powdered by mutual friction.

Continued...

- Exhaust ventilation should be designed to prevent accumulation and recirculation of particulates in the workplace.
- If in spite of local exhaust an adverse concentration of the substance in air could occur, respiratory protection should be considered. Such protection might consist of:

(a): particle dust respirators, if necessary, combined with an absorption cartridge;
(b): filter respirators with absorption cartridge or canister of the right type;
(c): fresh-air hoods or masks

- Build-up of electrostatic charge on the dust particle, may be prevented by bonding and grounding.
- Powder handling equipment such as dust collectors, dryers and mills may require additional protection measures such as explosion venting.

Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to efficiently remove the contaminant.

Type of Contaminant:	Air Speed:
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 ft/min)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 ft/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 4-10 m/s (800-2000 ft/min) for extraction of crusher dusts generated 2 metres distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

Individual protection measures, such as personal protective equipment



Eye and face protection

- Safety glasses with side shields.
- Chemical goggles.[AS/NZS 1337.1, EN166 or national equivalent]
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].

Skin protection

See Hand protection below

Hands/feet protection

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.

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:
· frequency and duration of contact,
· chemical resistance of glove material,
· glove thickness and
· dexterity
Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).
· When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
· When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
· Contaminated gloves should be replaced.
As defined in ASTM F-739-96 in any application, gloves are rated as:

Continued...

	· Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Experience indicates that the following polymers are suitable as glove materials for protection against undissolved, dry solids, where abrasive particles are not present. ‣ polychloroprene. ‣ nitrile rubber. ‣ butyl rubber. ‣ fluorocaoutchouc. ‣ polyvinyl chloride. Gloves should be examined for wear and/ or degradation constantly.
Body protection	See Other protection below
Other protection	‣ Overalls. ‣ P.V.C apron. ‣ Barrier cream. ‣ Skin cleansing cream. ‣ Eye wash unit.

Respiratory protection

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

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

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

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

· 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.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Continued...

Appearance	Dark yellow fine powder with characteristic odour; mixes with water. Bulk density: 1.0-1.3 g/mL

Physical state	Divided Solid	**Relative density (Water = 1)**	Not Available
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Applicable
pH (as supplied)	Not Available	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (mm2/s)**	Not Applicable
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Applicable	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Applicable
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Applicable
Vapour pressure (kPa)	Not Applicable	**Gas group**	Not Available
Solubility in water	Miscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Applicable	**VOC g/L**	Not Available
Heat of Combustion (kJ/g)	Not Available	**Ignition Distance (cm)**	Not Available
Flame Height (cm)	Not Available	**Flame Duration (s)**	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	**Enclosed Space Ignition Deflagration Density (g/m3)**	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

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

Continued...

Inhaled	The material is not classified as causing respiratory irritation under Regulation (EC) No 1272/2008 (CLP), as amended. Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be implemented in an occupational setting. Persons with impaired respiratory function, airway diseases and conditions such as emphysema or chronic bronchitis, may incur further disability if excessive concentrations of particulate are inhaled. If prior damage to the circulatory or nervous systems has occurred or if kidney damage has been sustained, proper screenings should be conducted on individuals who may be exposed to further risk if handling and use of the material result in excessive exposures.
Ingestion	Pantothenic Acid, also known as vitamin B5, is one of eight vitamins that comprise the B complex. Pantothenic acid is part of coenzyme A (CoA), an essential metabolite the body uses to produce energy from food (fats, carbohydrates and proteins). The FDA's reference daily intakes (RDI) for Pantothenic acid is 10 mg. Toxicity of pantothenic acid is unlikely. In fact, no Tolerable Upper Level Intake (UL) has been established for the vitamin. Large doses of the vitamin, when ingested, may only yield mild intestinal distress and diarrhea at worst. However, a very large amount of vitamin B5 (e.g: 5 - 9 gram) is widely known to cause nausea, headaches, diarrhea and a lack of energy. Other adverse effects include oedema, severe fatigue, joint pains, reduced protein metabolism, gastrointestinal symptoms, raised VLDL triglycerides, dehydration, depression The lack of energy is believed to be the depleted vitamin B12 (cobalamin), as massive amounts of vitamin B5 will deplete other vitamin B components. Restitution for this loss with an additional vitamin B complex may be necessary to compensate the lost vitamin B elements There are no adverse reactions known following parenteral or topical application of the vitamin. Reported overdose of 10 to 20 gm calcium pantothenic acid produce diarrhea and failure of the body to metabolise other B vitamins. . Use in food, and as food additive indicates high degree of tolerance Vitamin E, a fat-soluble, easily absorbable vitamin, stored in the liver, adipose tissue and muscle, as well as, acts as an antioxidant and free radical scavenger in lipophilic environments, may cause skin rashes and gastrointestinal irritation. It may also present with. Other nonspecific adverse effects such as fatigue, muscle weakness, delayed wound healing, headache and decreased levels of tri-iodothyronine and thyroxine.
Skin Contact	This material can cause inflammation of the skin on contact in some persons. The material may accentuate any pre-existing dermatitis condition Open cuts, abraded or irritated skin should not be exposed to this material
Eye	This material causes serious eye irritation.
Chronic	Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Strong evidence exists that this substance may cause irreversible mutations (though not lethal) even following a single exposure. Ample evidence from experiments exists that there is a suspicion this material directly reduces fertility. Based on experience with animal studies, exposure to the material may result in toxic effects to the development of the foetus, at levels which do not cause significant toxic effects to the mother. Laboratory (in vitro) and animal studies show, exposure to the material may result in a possible risk of irreversible effects, with the possibility of producing mutation. There is limited evidence that, skin contact with this product is more likely to cause a sensitisation reaction in some persons compared to the general population. Naphthoquinones are toxic to a variety of cell types in vitro.Naphthoquinone-based complexes with metal ions may present higher cytotoxic properties in comparison with naphthoquinone itself or with metal ions. Lawsone is a derivate of 1,4-napthoquinone with significant cytotoxic properties, demonstrated on different cell lines, due to its ability to induce production of reactive oxygen species (ROS). Data from experimental studies indicate that pyridines represent a potential cause of cancer in man. They have also been shown to cross the placental barrier in rats and cause premature delivery, miscarriages and stillbirths. Quinones may undergo a vicious cycle involving reduction-oxidation reaction and covalent bonding with the liberation of free radicals and reactive oxygen compounds. These can damage the DNA and other cellular macromolecules and activate signalling pathways which may lead to cancer. Oxygen activation and generation of a superoxide occurs in body metabolic reactions. However, when their rate of formation exceed the capacity of the body s defence mechanisms, it results in oxidative stress which is involved in some biological processes such as aging and inflammation reactions and in the pathogenesis of several diseases, including acute pancreatitis, post-ischaemic syndrome, tumour formation, hardening of the arteries, and diabetic angiopathy. Free radicals can readily react and damage cell membranes and genetic materials, sulphur containing amino acids, complex sugars and glycogen. It may induce allergic reaction and even cell death. Gene modification may result in tumour formation. Long term exposure to high dust concentrations may cause changes in lung function i.e. pneumoconiosis, caused by particles less than 0.5 micron penetrating and remaining in the lung. There has been some concern that this material can cause cancer or mutations but there is not enough data to make an assessment. Vitamin E has been shown to cause life-threatening adverse effects in premature infants, including sepsis and necrotizing enterocolitis (inflammation of the bowel with necrosis). Ascites (swelling in the abdomen), enlargement of the liver, and loss of platelets have also occurred, sometimes resulting in death. One study has shown that alpha-tocopherol at sufficient doses (50mg/d) can greatly increase the risk of subarachnoid haemorrhage in male smokers.

Keymix Abdextra Key 94 Concentrated Soluble Multi-vitamins for Poultry	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

Continued...

ascorbic acid	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: 11900 mg/kg[2]	Not Available
niacinamide	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[2]	Eye (Rodent - rabbit): 100mg
	Inhalation (Rat) LC50: >3.8 mg/l4h[1]	Eye (Rodent - rabbit): 100mg - Severe
	Oral (Rat) LD50: >2500 mg/kg[1]	Eye (Rodent - rabbit): 280mg/7D - Moderate
		Eye: adverse effect observed (irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
D-alpha-tocopherol acetate	**TOXICITY**	**IRRITATION**
	Oral (Mouse) LD50; 5000 mg/kg[2]	Not Available
trisodium phosphate dodecahydrate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 7940 mg/kg[2]	Not Available
	Oral (Rat) LD50: 6500 mg/kg[2]	

Legend:	***1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances***

Keymix Abdextra Key 94 Concentrated Soluble Multi-vitamins for Poultry	No significant acute toxicological data identified in literature search. The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.
ASCORBIC ACID	Laboratory (in vitro) and animal studies show, exposure to the material may result in a possible risk of irreversible effects, with the possibility of producing mutation. e acid sequestrants (BASs), including cholestyramine, colestipol, and colesevelam, are widely used in endocrine and gastrointestinal disorders. Their core mechanism involves promoting hepatic cholesterol catabolism into bile acids and reducing serum low-density lipoprotein cholesterol (LDL-C) concentrations, rendering them suitable for patients intolerant to statins or requiring intensified lipid-lowering therapy . Additionally, colesevelam has been applied in diabetes management due to its glucose-lowering effects via pathways such as stimulating glucagon-like peptide-1 (GLP-1) secretion [. In hepatobiliary diseases, BASs mitigate toxic bile acid accumulation, serving as therapeutic options for bile acid diarrhea, cholestatic pruritus, primary biliary cholangitis (PBC), and primary sclerosing cholangitis (PSC) [studies further suggest their potential roles in alleviating inflammatory bowel disease (IBD), irritable bowel syndrome (IBS), non-alcoholic fatty liver (NAFL), and microscopic colitis consistently high reporting rates of gastrointestinal disorders across all three BAS agents, establishing these as core adverse reactions mainly manifesting as diarrhea, constipation, flatulence, and abdominal pain, consistent with drug labeling information and existing clinical trial evidence . These gastrointestinal events are mechanistically linked to BAS pharmacological actions. By binding bile acids in the intestinal lumen, BAS disrupts enterohepatic circulation and stimulates compensatory bile acid synthesis, thereby reducing serum cholesterol levels [. However, this disruption depletes the bile acid pool necessary for lipid emulsification, leading to steatorrhea and subsequent osmotic diarrhea. Reduced luminal bile acid concentrations may attenuate their physiological stimulation of colonic motility via Takeda G protein-coupled receptor 5 (TGR5), potentially contributing to constipation he gut microbiota plays a pivotal role in BAS-induced dysbiosis [. Diminished secondary bile acids (e.g., deoxycholic acid) due to BAS sequestration alter the microbial composition, reducing beneficial taxa while enriching opportunistic pathogens potentially linked to hydrogen sulfide production and abdominal distension [39]. This dysbiotic shift may also impair gut barrier function, increasing intestinal permeability and systemic inflammation—a potential contributor to extraintestinal AEs. \Adverse effects of ion channel modulators range from common, mild issues like dizziness, fatigue, and rash to serious and potentially fatal complications such as hypotension, cardiac arrest, and severe neurological effects. Specific side effects depend on the type of ion channel targeted and the specific drug, but common issues include central and peripheral nervous system effects (dizziness, headache, drowsiness, ataxia) and cardiovascular effects (tachycardia, edema, hypotension). verse effects of ion channel inhibitors can include dizziness, fatigue, and gastrointestinal issues like nausea and constipation. More specific side effects depend on the type of channel and drug, such as headaches and edema with calcium channel blockers, or skin reactions and heart rhythm problems with other inhibitors. Severe effects can include serious heart problems, liver dysfunction, or severe allergic reactions, and may be more likely in patients with certain pre-existing conditions or when combined with other medications.
NIACINAMIDE	Mutation in microorganisms 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

Continued...

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.
The major components of the family of alkylpyridine derivatives, within the Chemical Abstracts Service definition,include discrete short chain alkylated pyridines, such as mono-, di-, tri-methylpyridines, ethylpyridines, methylethylpyridines and propylpyridines. They are not considered to be highly hazardous. It is likely that the hazard potential of this family. is due to the presence of by-products in commercial versions of the substances, such as non-alkylated derivatives of pyridine.
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.
effects of HDAC activators can include gastrointestinal issues like nausea and vomiting, decreased blood cell counts (neutropenia, thrombocytopenia), fatigue, and rashes or itching. They can also potentially cause more serious effects, such as cardiac arrhythmias, yellowing of the skin or eyes (jaundice), and cytotoxic effects like apoptosis. These agents are potent and can affect various cellular processes, leading to broad side effects.
Direct evidence suggests that HDAC activators, or inhibitors (a closely related class of drugs), can lead to cardiac arrhythmias by affecting ion channels
At higher concentrations, HDAC activators can induce cell death through apoptosis, which can be a detrimental effect with long-term use
Inhibition of several HDACs simultaneously confers greater toxicity and long term side effects. Therefore discovery of isoform-selective HDACs, improve therapeutic potential.
The use of histone deacetylase (HDAC) inhibitors (HDIs) as monotherapies for various solid malignancies demonstrate that they are well tolerated with good toxicity profiles compared to current standard cancer therapies. In general, the side effects of HDAC inhibitors are reversible with drug cessation and primarily include fatigue, nausea, dehydration, diarrhea, prolonged QT, thrombocytopenia, lymphopenia, and neutropenia. When used as monotherapies in solid cancers, HDAC inhibitors did not tend to significantly improve results compared to current standard therapies. Most preclinical studies, particularly in solid malignancies, have observed a cytostatic response to HDAC inhibitors when used as monotherapies, but when combined with radiation, chemotherapy, or other targeted agents, these drugs produce a more powerful cytotoxic response.
Although this family comprises chemical compounds from unrelated chemical classes that have different HDAC isoform specificities, they surprisingly have very similar toxicity profiles. In contrast, the observed toxicity profile is somewhat different from that of traditional cytotoxic chemotherapeutic agents and from other epigenetic agents. While some of the side effects may be familiar to the oncologist, others are less commonly seen.
Some side effects can be routinely managed (e.g., anti-emetics to alleviate associated nausea and vomiting).
Due to asymptomatic electrocardiogram (ECG) changes noted in early trials, patients receiving these agents have been closely monitored; however, HDIs do not appear to be associated with a greater incidence of cardiac adverse events than other chemotherapeutic agents.
Inhibition of NF-kB in vivo can be detrimental. NF-kB controls multiple functions in homeostasis including a functional immune response, cell cycle, and cell death. Genetic studies in mice and analysis of naturally occurring mutations in humans point to specific developmental and immune consequences due to altering NF-kB activity.
The same functions that make NF-kB attractive for developing inhibitors for treating disease also play a role in homeostasis,and disruption of the NF-kB pathway during development or in adults leads to unfavorable and potentially unhealthy consequences.
NF-kB plays a role in multiple homeostatic cellular processes including response to stimuli,cell proliferation, and death, regulating communication between cells, but is also tightly linked with other signaling pathways within the cell, such a p38 and JNK. In addition to mediating proinflammatory responses, NF-kB may regulate apoptotic and cell cycle changes induced by cellular stress, DNA damage or oncogenes by communication with the tumor suppressor p53. Disruption of normal cellular responses by inhibiting NF-kB can have adverse consequences such as immune suppression and tissue damage.
Understanding the consequences of lack of NF-kB activity in adult humans comes from observation of naturally occurring genetic deficiencies in this pathway. Mutations have been discovered in humans in signaling molecules upstream of NF-kB resulting in defects in development or immunity. Genetic defects have also been discovered in genes that immediately affect NF-kB activation including IKK gamma (NEMO), a subunit of the IKK complex, and IkBalpha.The IKK gamma mutations result in a defective IKK complex and the IkBalpha mutation results in an IkBalpha protein that cannot be phosphorylated and degraded. Both genetic defects result in suppressed NF-kB activation and ectodermal dysplasia with immunodeficiency.In general patients with these genetic defects have multiple immunological defects including impaired innate immunity,impaired antibody production, and ultimately severe bacterial infections. Understanding the immune defects and susceptibilities in patients with genetic defects in the NF-kB pathway will help prepare for potential adverse effects of pharmacologic NF-kB inhibitors
The requirement for NF-kB in the development and maintenance of the immune system is well documented. NF-kB is required for survival during fetal development and for normal lymphocyte generation in adult mice. Removal of the p65 (RelA) subunit of NF-kB or the IKKbeta gene results in death during fetal development primarily due to massive liver apoptosis
Fetal liver stem cells from p65 or IKKbeta deficient mice have been transplanted into irradiated hosts revealing a specific requirement of NF-kB for T-cells, B-cells, and common lymphoid progenitor development but not for myeloid cells or stem cells. The failure to produce lymphocytes is mediated through hypersensitivity to TNF due to lack of NF-kB activity. Lymphocyte depletion with chemical or genetic inhibition of NF-kB have implications for therapeutic potential use in humans. The double-sided nature of NF-kB inhibition is clear in this instance where chemical inhibition in vivo mimics genetic experiments inducing rapid TNF-dependent apoptosis. Rapid induction of apoptosis may be an advantage for treating some forms of cancer, but at the same time cause depletion of some lymphocyte populations.
In addition to controlling lymphocyte development, NF-kB plays a major role in both adaptive and innate immunity. Various signaling pathways responding to receptor recognition of immune challenge converge on NF-kB which then regulates genes that control the immune response. Both T-cell receptor and B-cell receptors activate NF-kB through phosphorylation of CARMA1 by

Continued...

PKC theta and PKC beta respectively, resulting in recruitment and activation of IKK and ultimately expression of genes that control cellular activation, proliferation, and survival. In addition, NF-kB plays a role in T-cell response to costimulatory signals.
Cells respond to pathogenic microorganisms in part through recognition by Toll-like receptors (TLRs).TLR-family members recognize different molecular structures present in microbes and respond by activating signaling pathways including NF-kB leading to expression of anti-microbial effector molecules, as well as molecules that help in development of the adaptive immune response. Inhibition of NF-kB during TLR stimulation can lead to macrophage apoptosis, a mechanism used by some pathogens to help evade immune response. NF-kB is clearly required for normal mature B-cell and T-cell maintenance and function, including regulatory, memory, and natural killer-like T cells. Inhibition of NF-kB activation in lymphocytes results in defects in growth, survival, and cytokine production and blocks multiple steps in germinal center formation.Given the diverse roles NF-kB plays in immune response to pathogens it is not surprising to find mice genetically deficient in components of the NF-kB pathway are susceptible to parasitic and bacterial infection.
The role of NF-kB in inhibition of apoptosis is one of the factors that make it a potential target for cancer therapy. NF-kB deficient mice die during embryogenesis in part due to TNF-mediated liver damage. Adult mice with impaired NF-kB targeted to the liver have normal liver function, but have severe liver damage after challenge with concanavalin A, a pan-T cell activator.Liver damage occurs due to sustained activation of JNK due to accumulation of reactive oxygen species (ROS) in the absence of normal NF-kB activation.
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 (CYP) enzyme modulators can cause adverse effects, primarily through drug-drug interactions. Specifically, CYP inhibitors can increase the concentration of other drugs in the body, leading to toxicity or adverse reactions, while CYP inducers can decrease drug concentrations, potentially causing therapeutic failure.
Sirtuins are evolutionarily conserved NAD+-dependent acetyl-lysine deacetylases that belong to class III type histone deacetylases. In humans, seven sirtuin isoforms (Sirt1 to Sirt7) have been identified.
otably, this protein family plays a variety of important roles in cellular biology such as inflammation, metabolism, oxidative stress, and apoptosis, etc., thus, it is considered a potential therapeutic target for different kinds of otably, this protein family plays a variety of important roles in cellular biology such as inflammation, metabolism, oxidative stress, and apoptosis, etc., thus, it is considered a potential therapeutic target for different kinds of patathologies including cancer, cardiovascular disease, respiratory disease, and other conditions
Sirtuins are involved in the regulation of metabolism and life span . They are also implicated in determining the balance between apoptosis, cell survival, and cell proliferation. In humans, seven sirtuin isoforms are localize either in the nucleus, cytoplasm, or mitochondria
Sirtuins are evolutionarily conserved NAD+-dependent acetyl-lysine deacetylases that belong to class III type histone deacetylases.
Certain research has implicated sirtuins as a central player in the determination of platelet aging.

Continued...

Sirtuin inhibitors induced apoptosis-like changes in blood platelets, associated with a rise in active Bax and a significant drop in platelet count.
Platelets, the enucleate blood cells derived from megakaryocytes, are discoid in shape, with size ranging between 2–4 µm. In response to vascular injury, platelets tether, adhere, aggregate, and finally form platelet plugs in injured vessel walls to arrest bleeding from blood vessels . HDAC inhibition has been reported previously to affect platelet function
Inhibition of sirtuins with sirtinol attenuated the activation phenotype of platelets, which included agonist-induced platelet aggregation, a rise in intracellular Ca2+, and the generation of thromboxane B2 (. However, it is not yet clear whether sirtuins have any role in platelet survival, as demonstrated for other cells
EAlier studies, including ours, have shown that delimitation of platelet life span involves balancing interactions between Bcl-XL, Bax/Bak, and the proteasome system . Here we asked whether sirtuins have a regulatory role in apoptosis-like events in platelets.
inhibitors of sirtuin deacetylases, sirtinol, EX-527, and AGK2, markedly stimulated apoptosis-like changes in platelets in a dose-dependent manner, as revealed by enhanced annexin V binding to the platelet surface, generation of reactive oxygen species (ROS), and disruption in mitochondrial transmembrane potential (??m). Apoptosis-like changes in platelets were associated with enhanced phagocytic clearance of cells by macrophages. The apoptosis-like phenotype in platelets induced by sirtuin inhibitors was attributable to p53-mediated transcription-independent induction of proapoptotic Bax and was calpain-dependent.
Administration of either sirtinol or EX-527 in mice resulted in a decrease i
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.
hepatitis B vaccine is a vaccine that prevents hepatitis B. The first dose is recommended within 24 hours of birth with either two or three more doses given after that.] This includes those with poor immune function such as from HIV/AIDS and those born premature. It is also recommended that health-care workers be vaccinated.] In healthy people, routine immunisation results in more than 95% of people being protected.
Serious side effects from the hepatitis B vaccine are very uncommon. Pain may occur at the site of injection.[13] It is safe for use during pregnancy or while breastfeeding.] It has not been linked to Guillain–Barré syndrome.[Hepatitis B vaccines are produced with recombinant DNA techniques and contain immunologic adjuvant.] They are available both by themselves and in combination with other vaccines
several studies have looked for an association between recombinant hepatitis B vaccine and multiple sclerosis (MS) in adults.[] Most studies do not support a causal relationship between hepatitis B vaccination and demyelinating diseases such as MS
2006 study concluded that evidence did not support an association between hepatitis B vaccination and sudden infant death syndrome, chronic fatigue syndrome, or multiple sclerosis. A 2007 study found that the vaccination does not seem to increase the risk of a first episode of MS in childhood.[Hepatitis B vaccination has not been linked to onset of autoimmune diseases in adulthood.
Studies have found that that immune memory against HepB is sustained for at least 30 years after vaccination, and protects against clinical disease and chronic HepB infection, even in cases where anti-hepatitis B surface antigen (anti-Hbs) levels decline below detectable levels.[Testing to confirm successful immunization or sustained immunity is not necessary or recommended for most people, but is recommended for infants born to a mother who tests positive for HBsAg or whose HBsAg status is not known; for healthcare and public safety workers; for immunocompromised people such as haemodialysis patients, HIV patients, haematopoietic stem cell transplant [HSCT] recipients, or people receiving chemotherapy; and for sexual partners of HBsAg-positive people
The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

Continued...

D-ALPHA-TOCOPHEROL ACETATE	for DL-form alpha-Tocopherol was non-mutagenic and non-carcinogenic, and the results of reproduction/ teratology studies did not indicate that alpha-tocopherol had adverse effects on reproductive function. However, in a long-term study in rats, a no-effect level could not be established with respect to effects on blood clotting and liver histology, and there was evidence from human studies that excessive intakes of alpha-tocopherol could cause haemorrhage. Other adverse effects noted in clinical studies at doses of > 720 mg alpha-tocopherol/day included weakness, fatigue, creatinuria and effects on steroid hormone metabolism. Clinical studies indicate that, generally, intakes of below about 720 mg/day are without adverse effects in man, but one investigation in elderly patients showed an increase in serum cholesterol at doses of 300 mg alpha-tocopherol daily. Incidences of allergic reactions seem to be very rare. alpha-Tocopherol may be an essential nutrient. The U.S. National Academy of Sciences/National Research Council has recommended a dietary allowance of 0.15 mg/kg b.w./day. However, excessive intakes of alpha-tocopherol produce adverse clinical and biochemical effects, and self-medication with large doses of vitamin E preparations could present a hazard. The previously-allocated ADI was amended to include a lower value, which reflects the fact that alpha-tocopherol may be an essential nutrient. The upper value, which represents the maximum value for the AID, is based on clinical experience in man. IPCS Inchem: https://www.inchem.org/documents/jecfa/jecmono/v21je05.htm
ASCORBIC ACID & NIACINAMIDE & TRISODIUM PHOSPHATE DODECAHYDRATE	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.

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

Keymix Abdextra Key 94 Concentrated Soluble Multi-vitamins for Poultry

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

ascorbic acid

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

D-alpha-tocopherol acetate

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

trisodium phosphate dodecahydrate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>100mg/l	2
EC50	48h	Crustacea	>100mg/l	2
NOEC(ECx)	48h	Crustacea	<52mg/L	4
LC50	96h	Fish	>100mg/l	2

Legend: ***Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. US EPA, Ecotox database - Aquatic Toxicity Data 4. ECETOC Aquatic Hazard Assessment Data 5. NITE (Japan) - Bioconcentration Data 6. METI (Japan) - Bioconcentration Data 7. Vendor Data 8. User defined data***

For Pyridine and its Derivatives:

Continued...

Environmental Fate: As molecular weight/substitution increase in the pyridine category, greater distribution to water and soil, and less to air, is predicted.
Atmospheric Fate: The lower weight pyridine, piperidine, is expected to be rapidly degraded by UV light in the atmosphere, with an estimated half-life of < 1 day. Higher molecular weight pyridines are expected to be broken down by sunlight, (photodegrades), more slowly, (half-lives ranging from 10-30 days). Lutidines and collidines are expected to photodegrade even more slowly. The nitrile derivatives of pyridine are also predicted to photodegrade slowly, with half-lives of 164 days; however, the nitrile derivatives of pyridine are predicted to partition to air much less favorably than to soil and water.
Terrestrial Fate: Depending upon the environmental conditions, different types of bacteria, fungi, and enzymes are involved in the breakdown of these substances.
Aquatic Fate: Pyridines are not expected to be broken down by water; however, breakdown by microbes is expected, if sufficient oxygen is available. These substances are expected to be stable in low oxygen/sterile conditions.
Ecotoxicity: Pyridine and its derivatives range from slightly to moderately toxic to fish, invertebrates and algae.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
ascorbic acid	LOW	LOW
niacinamide	HIGH	HIGH
trisodium phosphate dodecahydrate	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
ascorbic acid	LOW (LogKOW = -1.85)
niacinamide	LOW (LogKOW = 0.31)
trisodium phosphate dodecahydrate	LOW (LogKOW = -7.64)

Mobility in soil

Ingredient	Mobility
ascorbic acid	LOW (Log KOC = 10)
niacinamide	LOW (Log KOC = 10)
trisodium phosphate dodecahydrate	HIGH (Log KOC = 1)

SECTION 13 Disposal considerations

Waste treatment methods

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

SECTION 14 Transport information

Labels Required

Continued...

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

14.7. Maritime transport in bulk according to IMO instruments

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

Not Applicable

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

Product name	Group
ascorbic acid	Not Applicable
niacinamide	Not Applicable
D-alpha-tocopherol acetate	Not Applicable
trisodium phosphate dodecahydrate	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
ascorbic acid	Not Applicable
niacinamide	Not Applicable
D-alpha-tocopherol acetate	Not Applicable
trisodium phosphate dodecahydrate	Not Applicable

SECTION 15 Regulatory information

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

ascorbic acid is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

niacinamide is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 3
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australian Inventory of Industrial Chemicals (AIIC)

D-alpha-tocopherol acetate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

trisodium phosphate dodecahydrate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 3
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)

Additional Regulatory Information

Not Applicable

National Inventory Status

Continued...

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (ascorbic acid; D-alpha-tocopherol acetate; trisodium phosphate dodecahydrate)
China - IECSC	Yes
Colombia - Colombia National Industrial Chemicals Inventory	No (ascorbic acid; niacinamide; D-alpha-tocopherol acetate; trisodium phosphate dodecahydrate)
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory 'Active'
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	Yes
UAE - Control List (Banned/Restricted Substances)	No (ascorbic acid; niacinamide; D-alpha-tocopherol acetate; trisodium phosphate dodecahydrate)
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	21/05/2026
Initial Date	25/07/2013

Other information

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

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

Definitions and abbreviations

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

Continued...

- 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