NIACINAMIDE

Ingredients Plus Pty Limited

Chemwatch Hazard Alert Code: **2**

Chemwatch: **17476**
Version No: **5.1**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **22/03/2021**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	NIACINAMIDE
Chemical Name	niacinamide
Chemical formula	C6H6N2O
Other means of identification	Not Available
CAS number	98-92-0

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

Relevant identified uses	Used as alternative to nicotinic acid for the prevention and treatment of pellagra; dietary supplement. Therapeutic or pharmacologically-active agent. Niacin, also known as nicotinic acid, is an organic compound and a form of vitamin B3, an essential human nutrient It can be manufactured by plants and animals from the amino acid tryptophan. Prescription niacin, in immediate-release and slow-release forms, is used to treat primary hyperlipidemia and hypertriglyceridemia. It is used either as a monotherapy or in combination with other lipid-modifying drugs. Niacin as a dietary supplement is used to treat pellagra, a disease caused by niacin deficiency. Signs and symptoms of pellagra include skin and mouth lesions, anemia, headaches, and tiredness Many countries mandate its addition to wheat flour or other food grains, thereby reducing the risk of pellagra. The amide derivative nicotinamide (niacinamide) is a component of the coenzymes nicotinamide adenine dinucleotide (NAD) and nicotinamide adenine dinucleotide phosphate (NADP+). Although niacin and nicotinamide (niacinamide) are identical in their vitamin activity, nicotinamide does not have the same pharmacological, lipid-modifying effects or side effects as niacin, i.e., when niacin takes on the -amide group, it does not reduce cholesterol nor cause flushing. Nicotinamide is recommended as a treatment for niacin deficiency because it can be administered in remedial amounts without causing the flushing, considered an adverse effect. NAD and NADP compounds 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. Vitamin intake recommendations made by several countries are that intakes of 14-18 mg/day are sufficient to meet the needs of healthy adults. Niacin or nicotinamide (niacinamide) are used for prevention and treatment of pellagra, a disease caused by lack of the vitamin. When niacin is used as a medicine to treat elevated cholesterol and triglycerides, daily doses range from 500 to 3,000 mg/day. High-dose nicotinamide does not have this medicinal effect Niacin is also a prescription medication. Amounts far in excess of the recommended dietary intake for vitamin functions will lower blood triglycerides and low density lipoprotein cholesterol (LDL-C), and raise blood high density lipoprotein cholesterol (HDL-C, often referred to as "good" cholesterol). Prescription niacin, while affecting lipid levels, did not reduce all-cause mortality, cardiovascular mortality, myocardial infarctions, nor fatal or non-fatal strokes Acyltransferase inhibitor. Acyltransferase (AT) are involved in the synthesis of tri- and di- acylglycerides. The pathway for triacylglycerol synthesis has provided several attractive targets for drug discovery in the treatment of metabolic diseases. Marketed drugs that inhibit enzymes in this pathway include orlistat (pancreatic lipase inhibitor), lomitapide (mitochondrial transfer protein inhibitor), and mipomersen (apolipoprotein B synthesis inhibitor), but poor gastrointestinal (GI) tolerability or safety considerations have limited their use and indications. In addition, several inhibitors of diacylglycerol transferase 1 (DGAT1) have advanced to the clinic but were withdrawn due to poor GI tolerability Acyltransferase (AT) catalyzes the transfer of an acyl moiety from acyl-coenzyme A (acyl-CoA) to an acceptor. ATs play important roles in the maintenance of homeostasis in the human body and have been linked to various diseases; therefore, several ATs have been proposed as potential targets for the treatment or prevention of such diseases. The AT family includes acyl-CoA:cholesterol AT (ACAT), diacylglycerol AT (DGAT), and monoacylglycerol AT (MGAT) for the metabolism of lipids. Molecular biological studies revealed the existence of their isozymes with distinct functions in the body. Isozymes of ACAT, DGAT and MGAT play distinct functions in neutral lipid metabolism in the human body and have been considered as potential therapeutic targets. Pyripyropene A derivatives, ACAT2-selective inhibitors, may be potential therapeutics for the treatment of atherosclerosis, homozygous familial hypercholesterolemia and nonalcoholic fatty liver disease. DGAT (acyl-CoA: diacylglycerol acyltransferase) is a transmembrane enzyme that acts in the final and committed step of triacylglycerides (TAGs) synthesis, and it has been proposed to be the rate-limiting enzyme in plant storage lipid accumulation. DGAT catalyzes the acylation of sn-1,2-diacylglycerol (DAG) at the sn-3 position using an acyl-CoA substrate. DGAT has been proposed to be the rate-limiting enzyme in plant storage lipid accumulation. DGAT is considered a key enzyme for biotechnological purposes; it might be utilized to increase oil content in oleaginous plant species. DGAT1 and DGAT2 are two of the enzymes that are responsible for the main part of TAG synthesis in most organisms, and they have been studied in many eukaryotic organisms. ACAT is an important enzyme which controls fatty acid metabolism. It regulates the absorption of dietary cholesterol which must be esterified before being transferred to the liver as chylomicrons. ACAT inhibitors can lower plasma cholesterol and triglyceride levels by inhibiting absorption and storage of metabolic fatty acid. The subsequent reduction in the hepatic production of very-low-density lipoprotein (VLDL) could directly reduce the formation of atherosclerotic plaques and the incidence of vascular disease. Most synthetic ACAT inhibitors have significant side effects, such as adrenal toxicity The pharmaceutical industry is therefore interested in the isolation of natural, non-toxic ACAT inhibitors. The discovery of pentacyclic triterpenoids (PTs) as a novel class of ACAT inhibitors suggests that plants such as olives which possess large amounts of natural PT could be used to develop agents to reduce plasma lipids and prevent atherosclerosis.Structurally similar PTs (i.e., oleanolic acid, ursolic acid and betulinic acid) , inhibited ACAT activities. Oleanolic acid and maslinic acid suppressed the gene expression of ACAT in rats with hyperlipidemia induced by ingestion of a high-cholesterol diet. ACAT catalyzes the formation of cholesteryl esters from cholesterol and long-chain fatty-acyl-coenzyme A. ACAT regulates intracellular cholesterol homeostasis and supplies cholesteryl esters for the production of lipoproteins in the liver and small intestine. Under pathological conditions, the accumulation of cholesteryl esters produced by ACAT in macrophages contributes to foam cell formation. Foam cells are a hallmark of the early stages in the development of atherosclerosis. ACAT is known to be involved in a variety of cholesterol trafficking events including cholesterol absorption in rodents. A high-cholesterol diet dramatically increases liver cholesteryl ester (CE) levels in animals, making them especially sensitive to ACAT inhibition. By contrast, serum cholesterol (SC) levels are not dramatically changed by cholesterol feeding in these animals, and most ACAT inhibitors have minimal effect on

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serum cholesterol levels.
There are two isoforms of ACAT; ACAT 1 and 2. These differ in function and distribution throughout the human body. Whilst ACAT1 is mainly responsible for the stabilization of cellular membrane function, ACAT2 is only expressed in hepatocytes and enterocytes. Inhibition of expression of the ACAT2 gene may become atheroprotective.
Monoacylglycerol acyltransferase 1 (MGAT) is a microsomal enzyme that catalyzes the synthesis of diacylglycerol (DAG) and triacylglycerol (TAG). Multiple compounds specifically inhibit intestinal MGAT activity. .MGAT1 is considered as an effective target for reducing hepatic steatosis and obesity. Inhibition of MGAT2 has been suggested as a therapeutic approach that may offer a distinct and superior efficacy versus GI tolerability profile. MGAT2 inhibitors have been proposed for the treatment of metabolic diseases and nonalcoholic steatohepatitis (NASH).
Mammalian cells synthesize triacylglycerol (TAG) via two convergent pathways. The classic pathway, called glycerol-3-phosphate pathway, starts first with glycerol-3-phosphate acyltransferase (GPAT) which catalyzes the acylation of glycerol-3-phosphate forming lysophosphatidic acid. Then 1-acylglycerol-3-phosphate acyltransferase (AGPAT) and lipin act to further acylate and dephosphorylate the lysophosphatidic acid, respectively, to produce diacylglycerol (DAG). On the other hand, the monoacylglycerol acyltransferase (MGAT) promotes alternative pathway to synthesize TAG. This enzyme directly catalyze the acylation of monoacylglycerol (MAG) to produce DAG. Finally, DAG is converted to TAG by DGAT1 or DGAT2, which is common in both pathways. MGAT1 was originally identified in mice as a microsomal enzyme that catalyzes the synthesis of DAG and TAG. MGAT2 is predominantly expressed in the small intestine, and plays a role in dietary fat absorption . MGAT3, which shares a higher sequence homology with DGAT2, is found only in higher mammals and humans, but not in rodents. All three MGAT family genes are localized in the endoplasmic reticulum (ER), but differ in tissue expression patterns and in catalytic properties
Murine MGAT1 localizes to the endoplasmic reticulum (ER) under normal conditions, whereas MGAT1 co-localize to the lipid droplets (LD) under conditions of enriching fatty acids, contributing to TAG synthesis and LD expansion.
A cytochrome P450 2E1 (abbreviated CYP2E1, EC 1.14.13.n7) inhibitor:
Besides the detoxifying role of CYP2E1 for compounds such as electrophilic agents, reactive oxygen species, free radical products, and the bioactivation of xenobiotics, CYP2E1 is also related in several diseases and pathophysiological conditions.
CYP2E1 inhibitors protect the cell from damage and the production of reactive oxygen species (ROS) induced by 1-methyl-4-phenylpyridinium ions (MPP+), LPS, and ethanol in wild-type primary astrocytes. CYP2E1 inhibitors diminish the accumulation of the ethanol derived acetaldehyde and acetate in brain homogenates
CYP2E1 is an enzyme that particularly participates in the metabolism of endogenous substrates, including acetone and fatty acids (abundant in the brain) and exogenous compounds such as anesthetics, ethanol, nicotine, acetaminophen, acetone, aspartame, chloroform, chlorzoxazone, tetrachloride, and some antiepileptic drugs like phenobarbital. CYP2E1 can also activate toxic compounds and procarcinogens found in tobacco smoke and nitrosamine compounds. CYP2E1 has an important player in the microsomal ethanol oxidizing system (MEOS).
A variety of heterocyclic compounds such as imidazole, pyrazole, 4-methylpyrazole, thiazole, isoniazid, solvents such as dimethyl sulfoxide, various alcohols, benzene, and acetone have been shown to elevate CYP2E1 levels.
Nonalcoholic steatohepatitis CYP2E1 expression is elevated in the same way as in many pathophysiological conditions, such as obesity, diabetes, fasting, and cancer,
CYP2E1 is expressed in high levels in the liver, where it works to clear toxins from the body. In doing so, CYP2E1 bioactivates a variety of common anesthetics, including acetaminophen, halothane, enflurane, and isoflurane The oxidation of these molecules by CYP2E1 can produce harmful substances such as trifluoroacetic acid chloride from halothane or NAPQI (N-acetyl-p--benzoquinone imine) from paracetamol (acetaminophen) and is a major reason for their observed hepatotoxicity in patients.
CYP2E1 has been implicated in different brain pathologies, possibly due to its role as a drug metabolism or activator enzyme.
CYP2E1 and other cytochrome P450 enzymes can inadvertently produce reactive oxygen species (ROS) in their active site when catalysis is not coordinated correctly, resulting in potential lipid peroxidation as well as protein and DNA oxidation. CYP2E1 is particularly susceptible to this phenomenon compared to other P450 enzymes, suggesting that its expression levels may be important for negative physiological effects observed in a number of disease states.
CYP2E1 expression levels have been correlated with a variety of dietary and physiological factors, such as ethanol consumption, diabetes, fasting, and obesity. It appears that cellular levels of the enzyme may be controlled by the molecular chaperone HSP90, which upon association with CYP2E1 allows for transport to the proteasome and subsequent degradation. Ethanol and other substrates may disrupt this association, leading to the higher expression levels observed in their presence. The increased expression of CYP2E1 accompanying these health conditions may therefore contribute to their pathogenesis by increasing the rate of production of ROS in the body
CYP2E1 is inhibited by a variety of small molecules, many of which act competitively. Two such inhibitors, indazole and 4-methylpyrazole, coordinate with the active site's iron atom. Other inhibitors include diethyldithiocarbamate (in cancer), and disulfiram (in alcoholism).
Cytochrome P450 2E1 (abbreviated CYP2E1, EC 1.14.13.n7) is a member of the cytochrome P450 mixed-function oxidase system, which is involved in the metabolism of xenobiotics in the body. While CYP2E1 itself carries out a relatively low number of these reactions (~4% of known P450-mediated drug oxidations), it and related enzymes CYP1A2 and CYP3A4 are responsible for the breakdown of many toxic environmental chemicals and carcinogens that enter the body, in addition to basic metabolic reactions such as fatty acid oxidations.
CYP2E1 is a membrane protein expressed in high levels in the liver, where it composes nearly 50% of the total hepatic cytochrome P450 mRNA and 7% of the hepatic cytochrome P450 protein The liver is therefore where most drugs undergo deactivation by CYP2E1, either directly or by facilitated excretion from the body.
CYP2E1 metabolizes mostly small, polar molecules, including toxic laboratory chemicals such as dimethylformamide, aniline, and halogenated hydrocarbons. While these oxidations are often of benefit to the body, certain carcinogens and toxins are bioactivated by CYP2E1, implicating the enzyme in the onset of hepatotoxicity caused by certain classes of drugs
CYP2E1 also plays a role in several important metabolic reactions, including the conversion of ethanol to acetaldehyde and to acetate in humans, where it works alongside alcohol dehydrogenase and aldehyde dehydrogenase.
CYP2E1 also carries out the metabolism of endogenous fatty acids such as the omega-1 hydroxylation of fatty acids such as arachidonic acid, involving it in important signaling pathways that may link it to diabetes and obesity. Thus, it acts as a monooxygenase to metabolize arachidonic acid to 19-hydroxyeicosatetraenoic acid (19-HETE) . However, it also acts as an epoxygenase activity to metabolize docosahexaenoic acid to epoxides, primarily 19R,20S-epoxyeicosapentaenoic acid and 19S,20R-epoxyeicosapentaenoic acid isomers (termed 19,20-EDP) and eicosapentaenoic acid to epoxides, primarily 17R,18S-eicosatetraenic acid and 17S,18R-eicosatetraenic acid isomers (termed 17,18-EEQ).
19-HETE is an inhibitor of 20-HETE, a broadly active signaling molecule, e.g. it constricts arterioles, elevates blood pressure, promotes inflammation responses, and stimulates the growth of various types of tumor cells; however the in vivo ability and significance of 19-HETE in inhibiting 20-HETE has not been demonstrated. EDP (epoxydocosapentaenoic acid) and EEQ (epoxyeicosatetraenoic acid) metabolites have a broad range of activities. In various animal models and in vitro studies on animal and human tissues, they decrease hypertension and pain perception; suppress inflammation; inhibit angiogenesis, endothelial cell migration and endothelial cell proliferation; and inhibit the growth and metastasis of human breast and prostate cancer cell lines. It is suggested that the EDP and EEQ metabolites function in humans as they do in animal models and that, as products of the omega-3 fatty acids, docosahexaenoic acid and eicosapentaenoic acid, the EDP and EEQ metabolites contribute to many of the beneficial effects attributed to dietary omega-3 fatty acids. EDP and EEQ metabolites are short-lived, being inactivated within seconds or minutes of formation by epoxide hydrolases, particularly soluble epoxide hydrolase, and therefore act locally.
CYP2E1 is not regarded as being a major contributor to forming the cited epoxides but could act locally in certain tissues to do so.
A cytochrome P450 3A4 (CYP3A4) inhibitor:
Cytochrome P450 3A4 (abbreviated CYP3A4) (EC 1.14.13.97) is an important enzyme in the body, mainly found in the liver and in the intestine. It oxidizes small foreign organic molecules (xenobiotics), such as toxins or drugs, so that they can be removed from the body.
Cytochrome P450 enzymes are essential for the metabolism of many medicines and endogenous compounds. The CYP3A family is the most abundant subfamily of the CYP isoforms in the liver. There are at least four isoforms: 3A4, 3A5, 3A7 and 3A43 of which 3A4 is the most important. Like all members of this family, it is a hemoprotein, i.e. a protein containing a heme group with an iron atom. The cytochrome P450 proteins are monooxygenases that catalyze many reactions involved in drug metabolism and synthesis of cholesterol, steroids, and other lipids components.
The CYP3A4 protein localizes to the endoplasmic reticulum, and its expression is induced by glucocorticoids and some pharmacological agents. Cytochrome P450 enzymes metabolize approximately 60% of prescribed drugs, with CYP3A4 responsible for about half of this metabolism. The enzyme also metabolizes some steroids and carcinogens. Most drugs undergo deactivation by CYP3A4, either directly or by facilitated excretion from the body. Also, many substances are bioactivated by CYP3A4 to form their active compounds, and many protoxins being toxicated into their

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toxic forms
CYP3A4 also possesses epoxygenase activity in that it metabolizes arachidonic acid to epoxyeicosatrienoic acids (EETs), i.e. (±)-8,9-, (±)-11,12-, and (±)-14,15-epoxyeicosatrienoic acids. The EETs have a wide range of activities including the promotion of certain types of cancers (notably epoxyeicosatetraenoic acid). CYP3A4 promotes the growth of various types of human cancer cell lines in culture by producing (±)-14,15-epoxyeicosatrienoic acids which stimulate these cells to grow. The cytochrome P450 is also reported to have fatty acid monooxgenase activity for metabolizing arachidonic acid to 20-Hydroxyeicosatetraenoic acid (20-HETE). 20-HETE has a wide range of activities that also include growth stimulation in breast and other types of cancers
CYP3A4 is absent in fetal liver but increases to approximately 40% of adult levels in the fourth month of life and 72% at 12 months. There is considerable variability in CYP3A4 activity in the population. Women have higher CYP3A4 activity than men.
Although CYP3A4 is predominantly found in the liver, it is also present in other organs and tissues of the body, where it may play an important role in metabolism. CYP3A4 in the intestine plays an important role in the metabolism of certain drugs. Often this allows prodrugs to be activated and absorbed
While many drugs are deactivated by CYP3A4, there are also some drugs which are activated by the enzyme. Some substances, such as grapefruit juice and some drugs, interfere with the action of CYP3A4. These substances will therefore either amplify or weaken the action of those drugs that are modified by CYP3A4.
CYP3A4 is subject to reversible and mechanism-based (irreversible) inhibition. The latter involves the inactivation of the enzyme via the formation of metabolic intermediates that bind irreversibly to the enzyme and then inactivate it.. The clinical effects of a mechanistic inactivator are more prominent after multiple dosing and last longer than those of a reversible inhibitor.
Inhibitors of CYP3A4 can be classified by their potency, such as:
· Strong inhibitor being one that causes at least a 5-fold increase in the plasma AUC values, or more than 80% decrease in clearance.
· Moderate inhibitor being one that causes at least a 2-fold increase in the plasma AUC values, or 50-80% decrease in clearance.
· Weak inhibitor being one that causes at least a 1.25-fold but less than 2-fold increase in the plasma AUC values, or 20-50% decrease in clearance.
Inflammatory mediators associated with a range of disease states are capable of having profound effects on CYP3A4 gene expression. Patients with inflammation, particularly elevated acute phase proteins such as C-reactive protein (CRP) have been noted to have reduced CYP3A4 function. This is clinically relevant in cancer patients because tumours can be a source of systemically circulating cytokines.
Acute systemic hypoxia (eg, in chronic respiratory or cardiac insufficiency) appears to up-regulate CYP3A4 activity.
CYP3A4 contributes to bile acid detoxification, the termination of action of steroid hormones, and elimination of phytochemicals in food and the majority of medicines.
CYP3A4 activity is induced via the pregnane X receptor (PXR), the constitutive androstane receptor (CAR), peroxisome proliferator-activated receptor (PPARa) and probably the glucocorticoid receptor (GR).
The magnitude of CYP3A4 induction can be substantial. Induction becomes apparent more slowly than inhibition and it takes more time for the induction to stop affecting medicine metabolism. For example, the induction of CYP3A4 by rifampicin takes around six days to develop and 11 days to disappear
Induction normally results in a decrease in the effect of the medicine. However, it can lead to increased toxicity if the increased metabolism of the parent compound is accompanied by an increase in exposure to a toxic metabolite
A cytochrome P450 2D6 (CYP2D6) inhibitor
CYP2D6, a member of the cytochrome P450 mixed-function oxidase system, is one of the most important enzymes involved in the metabolism of xenobiotics in the body. In particular, CYP2D6 is responsible for the metabolism and elimination of approximately 25% of clinically used drugs, via the addition or removal of certain functional groups – specifically, hydroxylation, demethylation, and dealkylation. CYP2D6 also activates some prodrugs. This enzyme also metabolizes several endogenous substances, such as hydroxytryptamines, neurosteroids, and both m-tyramine and p-tyramine which CYP2D6 metabolizes into dopamine in the brain and liver
Inhibitors of CYP2D6 can be classified by their potency, such as:
· Strong inhibitor being one that causes at least a 5-fold increase in the plasma AUC values of sensitive substrates metabolized through CYP2D6, or more than 80% decrease in clearance thereof.
· Moderate inhibitor being one that causes at least a 2-fold increase in the plasma AUC values of sensitive substrates metabolized through CYP2D6, or 50-80% decrease in clearance thereof
· Weak inhibitor being one that causes at least a 1.25-fold but less than 2-fold increase in the plasma AUC values of sensitive substrates metabolized through CYP2D6, or 20-50% decrease in clearance thereof.
CYP2D6 is primarily expressed in the liver. It is also highly expressed in areas of the central nervous system, including the substantia nigra.
Considerable variation exists in the efficiency and amount of CYP2D6 enzyme produced between individuals. Hence, for drugs that are metabolized by CYP2D6 (that is, are CYP2D6 substrates), certain individuals will eliminate these drugs quickly (ultrarapid metabolizers) while others slowly (poor metabolizers). If a drug is metabolized too quickly, it may decrease the drug's efficacy while if the drug is metabolized too slowly, toxicity may result. So, the dose of the drug may have to be adjusted to take into account of the speed at which it is metabolized by CYP2D6.
Other drugs may function as inhibitors of CYP2D6 activity or inducers of CYP2D6 enzyme expression that will lead to decreased or increased CYP2D6 activity respectively. If such a drug is taken at the same time as a second drug that is a CYP2D6 substrate, the first drug may affect the elimination rate of the second through what is known as a drug-drug interaction.
CYP2D6 shows the largest phenotypical variability among the CYPs, largely due to genetic polymorphism. The genotype accounts for normal, reduced, and non-existent CYP2D6 function in subjects. Pharmacogenomic tests are available to identify patients with variations in the CYP2D6 allele and have been shown to have widespread use in clinical practice. The CYP2D6 function in any particular subject may be described as one of the following:
· poor metabolizer – little or no CYP2D6 function
· intermediate metabolizers – metabolize drugs at a rate somewhere between the poor and extensive metabolizers
· extensive metabolizer – normal CYP2D6 function
· ultrarapid metabolizer – multiple copies of the CYP2D6 gene are expressed, so greater-than-normal CYP2D6 function occurs
A patient's CYP2D6 phenotype is often clinically determined via the administration of debrisoquine (a selective CYP2D6 substrate) and subsequent plasma concentration assay of the debrisoquine metabolite (4-hydroxydebrisoquine)
Race is a factor in the occurrence of CYP2D6 variability. The lack of the liver cytochrome CYP2D6 enzyme occurs approximately in 7-10% in white populations, and is lower in most other ethnic groups such as Asians and African-Americans at 2% each. The occurrence of CYP2D6 ultrarapid metabolizers appears to be greater among Middle Eastern and North African populations
Caucasians with European descent predominantly (around 71%) have the functional group of CYP2D6 alleles, while functional alleles represent only around 50% of the allele frequency in populations of Asian descent
The type of CYP2D6 function of an individual may influence the person's response to different doses of drugs that CYP2D6 metabolizes. The nature of the effect on the drug response depends not only on the type of CYP2D6 function, but also on the extent to which processing of the drug by CYP2D6 results in a chemical that has an effect that is similar, stronger, or weaker than the original drug, or no effect at all. For example, if CYP2D6 converts a drug that has a strong effect into a substance that has a weaker effect, then poor metabolizers (weak CYP2D6 function) will have an exaggerated response to the drug and stronger side-effects; conversely, if CYP2D6 converts a different drug into a substance that has a greater effect than its parent chemical, then ultrarapid metabolizers (strong CYP2D6 function) will have an exaggerated response to the drug and stronger side-effects.

Details of the supplier of the safety data sheet

Registered company name	Ingredients Plus Pty Limited	Ingredients Plus Pty Limited	Ingredients Plus NZ Ltd.
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Emergency telephone number

Association / Organisation	**CHEMWATCH EMERGENCY RESPONSE**	**CHEMWATCH EMERGENCY RESPONSE**	**CHEMWATCH EMERGENCY RESPONSE**
Emergency telephone numbers	+61 1800 951 288	+64 800 700 112	+61 1800 951 288
Other emergency telephone numbers	+61 2 9186 1132	Not Available	+61 3 9573 3188

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SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2A, Sensitisation (Skin) Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

Label elements

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

Hazard statement(s)

H315	Causes skin irritation.
H319	Causes serious eye irritation.
H317	May cause an allergic skin reaction.
H335	May cause respiratory irritation.

Precautionary statement(s) Prevention

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.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P302+P352	IF ON SKIN: Wash with plenty of water.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER/doctor/physician/first aider/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.
P362+P364	Take off contaminated clothing and wash it before reuse.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

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.

Not Applicable

SECTION 3 Composition / information on ingredients

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Substances

CAS No	%[weight]	Name
98-92-0	>99	Niacinamide

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*

Mixtures

See section above for composition of Substances

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.
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.

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 substrate or mixture

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

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	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. May emit poisonous fumes. May emit corrosive fumes.
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	▸ Clean up waste regularly and abnormal 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. ▸ Vacuum up or sweep up. **NOTE:** Vacuum cleaner must be fitted with an exhaust micro filter (HEPA type) (consider explosion-proof machines designed to be grounded during storage and use). ▸ Dampen with water to prevent dusting before sweeping. ▸ Place in suitable containers for disposal.
Major Spills	Moderate hazard. ▸ **CAUTION**: Advise personnel in area. ▸ Alert Emergency Services and tell them location and nature of hazard. ▸ Control personal contact by wearing protective clothing. ▸ Prevent, by any means available, spillage from entering drains or water courses. ▸ Recover product wherever possible. ▸ **IF DRY:** Use dry clean up procedures and avoid generating dust. Collect residues and place in sealed plastic bags or other containers for disposal. **IF WET:** Vacuum/shovel up and place in labelled containers for disposal. ▸ **ALWAYS:** Wash area down with large amounts of water and prevent runoff into drains. ▸ If contamination of drains or waterways occurs, advise Emergency Services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Prevent concentration in hollows and sumps. ▸ **DO NOT** enter confined spaces until atmosphere has been checked. ▸ **DO NOT** allow material to contact humans, exposed food or food utensils. ▸ Avoid contact with incompatible materials. ▸ When 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

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	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. ▸ 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	▸ Glass container is suitable for laboratory quantities ▸ Polyethylene or polypropylene container. ▸ 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

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

Emergency Limits

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

Ingredient	Original IDLH	Revised IDLH
Niacinamide	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
Niacinamide	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.*	

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.

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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.

Personal protection

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.
- 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], [AS/NZS 1336 or national equivalent]

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.

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	As defined in ASTM F-739-96 in any application, gloves are rated as: · 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. ▸ 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. 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	▸ 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

Particulate. (AS/NZS 1716 & 1715, EN 143:2000 & 149:001, 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.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Colourless crystals or white powder, soluble in water, ethanol and glycerol. Nearly odourless. Bitter and salty taste.

Physical state	Divided Solid	**Relative density (Water = 1)**	1.36
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available

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pH (as supplied)	Not Applicable	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	129	**Viscosity (cSt)**	Not Applicable
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	122.14
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Applicable	**Explosive properties**	Not Available
Flammability	Not Available	**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 (Not Available%)**	6.0-7.0 (10%).
Vapour density (Air = 1)	Not Applicable	**VOC g/L**	Not Applicable

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	The material can cause respiratory irritation in some persons. The body's response to such irritation can cause further lung damage. 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	Accidental ingestion of the material may be damaging to the health of the individual. Nicotinic acid (vitamin B3) and some of its derivatives may dilate the blood vessels. When given by mouth or by injection during treatment, it may produce a temporary flushing of the face, a sensation of heat and a pounding in the head.
Skin Contact	This material can cause inflammation of the skin on contact in some persons. 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. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream, through, for example, cuts, abrasions or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals. Prolonged eye contact may cause inflammation characterised by a temporary redness of the conjunctiva (similar to windburn).
Chronic	Long exposure or overingestion may cause vasodilation, skin dryness, allergic skin rashes, abdominal cramps, diarrhoea, nausea, vomiting, liver problems and excessive pigmentation. [Roche] Long-term exposure to respiratory irritants may result in airways disease, involving difficulty breathing and related whole-body problems. Skin contact with the material is more likely to cause a sensitisation reaction in some persons compared to the general population. There has been some concern that this material can cause cancer or mutations but there is not enough data to make an assessment. Substance accumulation, in the human body, may occur and may cause some concern following repeated or long-term occupational exposure. 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

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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.
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.
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.

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]	

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

NIACINAMIDE

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 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.
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.
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.
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

Continued...

	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. 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

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

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 Pyridine and its Derivatives:
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

Continued...

Ingredient	Persistence: Water/Soil	Persistence: Air
Niacinamide	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
Niacinamide	LOW (LogKOW = -0.37)

Mobility in soil

Ingredient	Mobility
Niacinamide	LOW (KOC = 51.56)

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

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

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

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
Niacinamide	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

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
Australian Inventory of Industrial Chemicals (AIIC)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes

Continued...

National Inventory	Status
Canada - NDSL	No (Niacinamide)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	Yes
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	22/03/2021
Initial Date	01/07/2001

SDS Version Summary

Version	Date of Update	Sections Updated
5.1	22/03/2021	Chronic Health, Classification, Disposal, Engineering Control, Exposure Standard, Personal Protection (other), Personal Protection (eye), Personal Protection (hands/feet), Physical Properties, Spills (minor), Storage (storage incompatibility), Storage (storage requirement), Synonyms, Toxicity and Irritation (Other), Use

Other information

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

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

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
PC—STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists
STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit。
IDLH: Immediately Dangerous to Life or Health Concentrations
ES: Exposure 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

end of SDS