

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

Catalogue Number	CS-EB-01471
Product Name	Niacinamide (OOL486)
CAS No.	98-92-0
Category	USP Standards
Synonyms	Nicotinamide
Brand	Clearsynth Labs Ltd.
Identified uses	Laboratory Chemicals
Uses advised against	Not available
Company	Clearsynth Labs Ltd. Mumbai, India
Emergency Phone #	+91-22-245045900
REACH No.	Not available

SECTION 2: Hazards identification

Disclaimer: This is sample MSDS. Please email sales@clearsynth.com for more details.

2.1 Classification of the substance or mixture-Regulation (EC) No 1272/2008:

Skin irritation (Category 2)

Serious eye damage/eye irritation (Category 2)

2.2 Label Elements

Signal Word: Warning



Hazard Statement(s)

Code	Statement
H315	Causes skin irritation.
H319	Causes serious eye irritation.
H335	Not available

Precautionary Statement(s)

Code	Statement
P261	Avoid breathing dust/fume/gas/mist/vapours/spray.
P264	Wash hands thoroughly after handling.
P264+P265	Not available
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present.
P319	Get medical help if you feel unwell.
P321	Specific treatment (see ... on this label).
P332+P317	If skin irritation occurs: Get medical help.
P337+P317	If eye irritation persists: Get medical help.
P362+P364	Take off contaminated clothing and wash it before reuse.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.
P405	Store locked up.
P501	Dispose of contents/container in accordance with local/regional/national/international regulations.

SECTION 3: Composition / information on ingredients

3.1 Substance

Component : Niacinamide (OOL486)

CAS Number : 98-92-0

Molecular Formula : C₆H₆N₂O

Molecular Weight : 122.12 g/mol

Parent Chemical : Niacin

Synonyms : Nicotinamide

Concentration : Not available

SECTION 4: First aid measures

Not available

SECTION 5: Firefighting measures

Not available

SECTION 6: Accidental release measures

Not available

SECTION-7: Handling and storage

Not available

SECTION 8: Exposure controls / personal protection

Not available

SECTION 9: Physical and chemical properties

9.1 Information on basic physical and chemical properties

Test	Result
Appearance	No data available
IR spectrum	No data available
pH	No data available
Solubility	No data available

Property	Value
a) Physical State	No data available
b) Color	No data available
c) Odor	No data available
d) pH	2.7
e) Vapour Pressure	No data available
f) Viscosity	No data available
g) Initial Boiling Point and boiling range	No data available
h) Melting Point / Freezing Point	No data available
i) Auto Ignition Temperature	No data available
j) Flash Point	No data available

Property	Value
k) Explosion Limit, Lower	No data available
l) Explosion Limit, Upper	No data available
m) Decomposition Temperature	No data available
n) Loss on Drying	No data available
o) Relative Density	No data available
p) Solubility (in DMSO)	No data available
q) Oxidizing Properties	No data available

SECTION 10: Stability and reactivity

Not available

SECTION 11: Toxicological information

11.1 Information on toxicological effects

- Acute toxicity: Uremic toxins such as nicotinamide are actively transported into the kidneys via organic ion transporters (especially OAT3). Increased levels of uremic toxins can stimulate the production of reactive oxygen species. This seems to be mediated by the direct binding or inhibition by uremic toxins of the enzyme NADPH oxidase (especially NOX4 which is abundant in the kidneys and heart) (A7868). Reactive oxygen species can induce several different DNA methyltransferases (DNMTs) which are involved in the silencing of a protein known as KLOTHO. KLOTHO has been identified as having important roles in anti-aging, mineral metabolism, and vitamin D metabolism. A number of studies have indicated that KLOTHO mRNA and protein levels are reduced during acute or chronic kidney diseases in response to high local levels of reactive oxygen species (A7869). /GENOTOXICITY/ The effect of 2 mM Niacinamide on unscheduled DNA synthesis on resting human lymphocytes /was studied/. In cells treated with UV irradiation or with N-methyl-N-nitro- N-nitrosoguanidine, niacinamide caused a two-fold stimulation of unscheduled DNA synthesis and retarded the rate of NAD⁺ lowering caused by these treatments. Niacinamide also reduced the burst of poly(ADP-ribose) synthesis caused by N-methyl-N-nitro- N-nitrosoguanidine treatment. The effect of niacinamide on unscheduled DNA synthesis was shown to be independent of protein or polyamine synthesis.

- Skin corrosion/irritation: No data available.

- Serious eye damage/eye irritation: No data available.

- Respiratory or skin sensitization: IDENTIFICATION AND USE: Nicotinamide is a white, crystalline powder.

Nicotinamide is used to prevent niacin deficiency and to treat pellagra. Nicotinamide is also used in cosmetics as a hair and skin conditioning agent. It has been used in the enrichment of bread, flour, and other grain-derived products. Animal feed is routinely supplemented with nicotinamide. It is also used in multi-vitamin preparations. Nicotinamide and niacin are similarly effective as a vitamin because they can be converted into each other within the organism. The blanket term vitamin B(3) is used for both. Niacinamide is a component of important coenzymes involved in hydrogen transfer. HUMAN STUDIES: In humans, nicotinamide is required for lipid metabolism, tissue respiration, and glycogenolysis. In vivo, nicotinamide is formed from conversion of niacin. In addition, some dietary tryptophan is oxidized to niacin and then to nicotinamide in vivo. Nicotinamide is incorporated into 2 coenzymes:

nicotinamide adenine dinucleotide (NAD) and nicotinamide adenine dinucleotide phosphate (NADP). NAD and NADP act as hydrogen-carrier molecules in glycogenolysis, tissue respiration, and lipid metabolism. In a study with 6 volunteers (single dose between 3 and 9 g/day) toxic symptoms associated with nicotinamide were mild and consisted mainly of nausea. The effect of 2 mM nicotinamide on unscheduled DNA synthesis on resting human lymphocytes was studied. In cells treated with UV irradiation or with N-methyl-N-nitro- N-nitrosoguanidine, nicotinamide caused a two-fold stimulation of unscheduled DNA synthesis. ANIMAL STUDIES: Application of 0.1 g nicotinamide to the eyes of rabbits induced reversible irritation. It did not produced sensitization in guinea pig test. Single ip injection of nicotinamide (100 mg/kg) to male rats was shown to significantly induce all components of the hepatic microsomal mixed function oxidase system as well as activities of drug-metabolizing enzymes. Groups of 12 male rats were fed nicotinamide in their diet for a period of 8 to 12 weeks. At 0.1% of the diet (100 mg/kg bw per day), nicotinamide caused no significant change in the growth rate, at 0.2%, growth rate was enhanced, but at 0.4%, a marked inhibition of growth rate resulted. Almost complete growth inhibition occurred in rats fed 1% nicotinamide. Lifelong treatment with 1% nicotinamide had no carcinogenic effect in mice. However, nicotinamide promoted diethylnitrosamine-induced renal tubular cell tumorigenesis in rats. In mice nicotinamide in doses of 500-2000 mg/kg depresses orientation reflexes and exploring behavior, and has antiaggressive and anticonvulsant properties. Nicotinamide supplementation in pregnant rats led to a decrease in placental and fetal hepatic genomic DNA methylation and genomic uracil contents (a factor modifying DNA for diversity) in the placenta and fetal liver and brain, which could be completely or partially prevented by betaine. Moreover, nicotinamide supplementation induced tissue-specific alterations in the mRNA expression of the genes encoding nicotinamide N-methyltransferase, DNA methyltransferase 1, catalase and tumor protein p53 in the placenta and fetal liver. High-dose nicotinamide supplementation increased fetal hepatic a-fetoprotein mRNA level, which was prevented by betaine supplementation. It is concluded that maternal nicotinamide supplementation can induce changes in fetal epigenetic modification and DNA base composition. Nicotinamide was negative in an Ames test performed with Salmonella strains TA 98, TA 100, TA 1535, TA 1537 and TA 1538 both with and without metabolic activation. Nicotinamide was not mutagenic in Saccharomyces stain D4. It was reported that nicotinamide at concentrations of 3 mg/mL (25 mM) induced large structural chromosome aberrations in vitro in Chinese hamster ovary cells.

- Germ cell mutagenicity: IDENTIFICATION AND USE: Nicotinamide is a white, crystalline powder. Nicotinamide is used to prevent niacin deficiency and to treat pellagra. Nicotinamide is also used in cosmetics as a hair and skin conditioning agent. It has been used in the enrichment of bread, flour, and other grain-derived products. Animal feed is routinely supplemented with nicotinamide. It is also used in multi-vitamin preparations. Nicotinamide and niacin are similarly effective as a vitamin because they can be converted into each other within the organism. The blanket term vitamin B(3) is used for both. Niacinamide is a component of important coenzymes involved in hydrogen transfer. HUMAN STUDIES: In humans, nicotinamide is required for lipid metabolism, tissue respiration, and glycogenolysis. In vivo, nicotinamide is formed from conversion of niacin. In addition, some dietary tryptophan is oxidized to niacin and then to nicotinamide in vivo. Nicotinamide is incorporated into 2 coenzymes: nicotinamide adenine dinucleotide (NAD) and nicotinamide adenine dinucleotide phosphate (NADP). NAD and NADP act as hydrogen-carrier molecules in glycogenolysis, tissue respiration, and lipid metabolism. In a study with 6 volunteers (single dose between 3 and 9 g/day) toxic symptoms associated with nicotinamide were mild and consisted mainly of nausea. The effect of 2 mM nicotinamide on unscheduled DNA synthesis on resting human lymphocytes was studied. In cells treated with UV irradiation or with N-methyl-N-nitro- N-nitrosoguanidine, nicotinamide caused a two-fold stimulation of unscheduled DNA synthesis. ANIMAL STUDIES: Application of 0.1 g nicotinamide to the eyes of rabbits induced reversible irritation. It did not produced sensitization in guinea pig test. Single ip injection of nicotinamide (100 mg/kg) to male rats was shown to significantly induce all components of the hepatic microsomal mixed function oxidase system as well as activities of drug-metabolizing enzymes. Groups of 12 male rats were fed nicotinamide in their diet for a period of 8 to 12 weeks. At 0.1% of the diet (100 mg/kg bw per day), nicotinamide caused no significant change in the growth rate, at 0.2%, growth rate was enhanced, but at 0.4%, a marked

inhibition of growth rate resulted. Almost complete growth inhibition occurred in rats fed 1% nicotinamide. Lifelong treatment with 1% nicotinamide had no carcinogenic effect in mice. However, nicotinamide promoted diethylnitrosamine-induced renal tubular cell tumorigenesis in rats. In mice nicotinamide in doses of 500-2000 mg/kg depresses orientation reflexes and exploring behavior, and has antiaggressive and anticonvulsant properties. Nicotinamide supplementation in pregnant rats led to a decrease in placental and fetal hepatic genomic DNA methylation and genomic uracil contents (a factor modifying DNA for diversity) in the placenta and fetal liver and brain, which could be completely or partially prevented by betaine. Moreover, nicotinamide supplementation induced tissue-specific alterations in the mRNA expression of the genes encoding nicotinamide N-methyltransferase, DNA methyltransferase 1, catalase and tumor protein p53 in the placenta and fetal liver. High-dose nicotinamide supplementation increased fetal hepatic α -fetoprotein mRNA level, which was prevented by betaine supplementation. It is concluded that maternal nicotinamide supplementation can induce changes in fetal epigenetic modification and DNA base composition. Nicotinamide was negative in an Ames test performed with Salmonella strains TA 98, TA 100, TA 1535, TA 1537 and TA 1538 both with and without metabolic activation. Nicotinamide was not mutagenic in Saccharomyces stain D4. It was reported that nicotinamide at concentrations of 3 mg/mL (25 mM) induced large structural chromosome aberrations in vitro in Chinese hamster ovary cells. /GENOTOXICITY/ The effect of 2 mM Niacinamide on unscheduled DNA synthesis on resting human lymphocytes /was studied/. In cells treated with UV irradiation or with N-methyl-N-nitro- N-nitrosoguanidine, niacinamide caused a two-fold stimulation of unscheduled DNA synthesis and retarded the rate of NAD⁺ lowering caused by these treatments. Niacinamide also reduced the burst of poly(ADP-ribose) synthesis caused by N-methyl-N-nitro- N-nitrosoguanidine treatment. The effect of niacinamide on unscheduled DNA synthesis was shown to be independent of protein or polyamine synthesis.

- Carcinogenicity: IDENTIFICATION AND USE: Nicotinamide is a white, crystalline powder. Nicotinamide is used to prevent niacin deficiency and to treat pellagra. Nicotinamide is also used in cosmetics as a hair and skin conditioning agent. It has been used in the enrichment of bread, flour, and other grain-derived products. Animal feed is routinely supplemented with nicotinamide. It is also used in multi-vitamin preparations. Nicotinamide and niacin are similarly effective as a vitamin because they can be converted into each other within the organism. The blanket term vitamin B(3) is used for both. Niacinamide is a component of important coenzymes involved in hydrogen transfer. HUMAN STUDIES: In humans, nicotinamide is required for lipid metabolism, tissue respiration, and glycogenolysis. In vivo, nicotinamide is formed from conversion of niacin. In addition, some dietary tryptophan is oxidized to niacin and then to nicotinamide in vivo. Nicotinamide is incorporated into 2 coenzymes: nicotinamide adenine dinucleotide (NAD) and nicotinamide adenine dinucleotide phosphate (NADP). NAD and NADP act as hydrogen-carrier molecules in glycogenolysis, tissue respiration, and lipid metabolism. In a study with 6 volunteers (single dose between 3 and 9 g/day) toxic symptoms associated with nicotinamide were mild and consisted mainly of nausea. The effect of 2 mM nicotinamide on unscheduled DNA synthesis on resting human lymphocytes was studied. In cells treated with UV irradiation or with N-methyl-N-nitro- N-nitrosoguanidine, nicotinamide caused a two-fold stimulation of unscheduled DNA synthesis. ANIMAL STUDIES: Application of 0.1 g nicotinamide to the eyes of rabbits induced reversible irritation. It did not produced sensitization in guinea pig test. Single ip injection of nicotinamide (100 mg/kg) to male rats was shown to significantly induce all components of the hepatic microsomal mixed function oxidase system as well as activities of drug-metabolizing enzymes. Groups of 12 male rats were fed nicotinamide in their diet for a period of 8 to 12 weeks. At 0.1% of the diet (100 mg/kg bw per day), nicotinamide caused no significant change in the growth rate, at 0.2%, growth rate was enhanced, but at 0.4%, a marked inhibition of growth rate resulted. Almost complete growth inhibition occurred in rats fed 1% nicotinamide. Lifelong treatment with 1% nicotinamide had no carcinogenic effect in mice. However, nicotinamide promoted diethylnitrosamine-induced renal tubular cell tumorigenesis in rats. In mice nicotinamide in doses of 500-2000 mg/kg depresses orientation reflexes and exploring behavior, and has antiaggressive and anticonvulsant properties. Nicotinamide supplementation in pregnant rats led to a decrease in placental and fetal hepatic genomic DNA methylation and genomic uracil contents (a factor

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- Reproductive toxicity: No data available.
- STOT-single exposure: No data available.
- STOT-repeated exposure: Uremic toxins such as nicotinamide are actively transported into the kidneys via organic ion transporters (especially OAT3). Increased levels of uremic toxins can stimulate the production of reactive oxygen species. This seems to be mediated by the direct binding or inhibition by uremic toxins of the enzyme NADPH oxidase (especially NOX4 which is abundant in the kidneys and heart) (A7868). Reactive oxygen species can induce several different DNA methyltransferases (DNMTs) which are involved in the silencing of a protein known as KLOTHO. KLOTHO has been identified as having important roles in anti-aging, mineral metabolism, and vitamin D metabolism. A number of studies have indicated that KLOTHO mRNA and protein levels are reduced during acute or chronic kidney diseases in response to high local levels of reactive oxygen species (A7869). Chronic exposure to uremic toxins can lead to a number of conditions including renal damage, chronic kidney disease and cardiovascular disease.
- Aspiration hazard: No data available.

Likely routes of exposure

- No data available.

Symptoms related to the physical, chemical and toxicological characteristics

- IDENTIFICATION AND USE: Nicotinamide is a white, crystalline powder. Nicotinamide is used to prevent niacin deficiency and to treat pellagra. Nicotinamide is also used in cosmetics as a hair and skin conditioning agent. It has been used in the enrichment of bread, flour, and other grain-derived products. Animal feed is routinely supplemented with nicotinamide. It is also used in multi-vitamin preparations. Nicotinamide and niacin are similarly effective as a vitamin because they can be converted into each other within the organism. The blanket term vitamin B(3) is used for both. Niacinamide is a component of important coenzymes involved in hydrogen transfer. HUMAN STUDIES: In humans, nicotinamide is required for lipid metabolism, tissue respiration, and glycogenolysis. In vivo, nicotinamide is formed from conversion of niacin. In addition, some dietary tryptophan is oxidized to niacin and then to nicotinamide in vivo. Nicotinamide is incorporated into 2 coenzymes: nicotinamide adenine dinucleotide (NAD) and nicotinamide adenine dinucleotide phosphate (NADP). NAD and NADP act as hydrogen-carrier molecules in glycogenolysis, tissue respiration, and lipid metabolism. In a study with 6 volunteers (single dose between 3 and 9 g/day) toxic symptoms associated with nicotinamide were mild and consisted mainly of nausea. The effect of 2 mM nicotinamide on unscheduled DNA synthesis on resting human lymphocytes was studied. In cells treated with UV irradiation or with N-methyl-N-nitro- N-nitrosoguanidine, nicotinamide caused a two-fold stimulation of unscheduled DNA synthesis. ANIMAL STUDIES: Application of 0.1 g nicotinamide to the eyes of rabbits induced reversible irritation. It did not produced sensitization in guinea pig test. Single ip injection of nicotinamide (100 mg/kg) to male rats was shown to significantly induce all components of the hepatic microsomal mixed function oxidase system as well as activities of drug-metabolizing enzymes. Groups of 12 male rats were fed nicotinamide in their diet for a period of 8 to 12 weeks. At 0.1% of the diet (100 mg/kg bw per day), nicotinamide caused no significant change in the growth

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SECTION 12: Ecological information

Not available

SECTION 13: Disposal considerations

Not available

SECTION 14: Transport information

Not available

SECTION 15: Regulatory information

Not available

SECTION 16: Other information

Not available

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