Kreatech FISH Probe USA

Leica Biosystems

Chemwatch Hazard Alert Code: **3**

Version No: **2.8**
Safety Data Sheet according to OSHA HazCom Standard (2012) requirements

Issue Date: **10/15/2015**
Print Date: **10/15/2015**
Initial Date: **05/19/2015**
S.GHS.USA.EN

SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	Kreatech FISH Probe USA
Synonyms	pKI series, p01P-series to p24P-series, p01Q-series to p24Q-series, p01C-series to p24C-series, p40V-series
Other means of identification	Not Available

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

Relevant identified uses	Use in laboratories - Professional. Maximum volume 1 ml.

Details of the supplier of the safety data sheet

Registered company name	Leica Biosystems
Address	1700 Leider Lane, Buffalo Grove IL 60089 United States
Telephone	800-248-0123
Fax	Not Available
Website	www.LeicaBiosystems.com
Email	kreatech-support@leicabiosystems.com

Emergency telephone number

Association / Organisation	Leica Biosystems
Emergency telephone numbers	800-248-0123
Other emergency telephone numbers	Not Available

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

GHS Classification	Reproductive Toxicity Category 1B

Label elements

GHS label elements	
SIGNAL WORD	**DANGER**

Hazard statement(s)

H360	May damage fertility or the unborn child

Continued...

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P281	Use personal protective equipment as required.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/attention.

Precautionary statement(s) Storage

P405	Store locked up.

Precautionary statement(s) Disposal

P501	Dispose of contents/container in accordance with local regulations.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
7732-18-5	20-50	water
75-12-7	20-50	formamide
9063-02-9	5-20	dextran sulfate
7647-14-5	<1	sodium chloride
6132-04-3	<1	sodium citrate dihydrate

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

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	If this product comes in contact with eyes: • Wash out immediately with water. • If irritation continues, seek medical attention. • Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin or hair contact occurs: • Flush skin and hair with running water (and soap if available). • Seek medical attention in event of irritation.
Inhalation	• If fumes, aerosols or combustion products are inhaled remove from contaminated area.
Ingestion	• Immediately give a glass of water. • First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

Most important symptoms and effects, both acute and delayed

	See Section 11

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

	The product contains a substantial proportion of water, therefore there are no restrictions on the type of extinguishing media which may be used. Choice of extinguishing media should take into account surrounding areas. Though the material is non-combustible, evaporation of water from the mixture, caused by the heat of nearby fire, may produce floating layers of combustible substances. In such an event consider: • foam. • dry chemical powder. • carbon dioxide.

Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.

Advice for firefighters

Fire Fighting	Not Applicable

Continued...

Fire/Explosion Hazard	▸ The material is not readily combustible under normal conditions. ▸ However, it will break down under fire conditions and the organic component may burn. ▸ Not considered to be a significant fire risk. ▸ Heat may cause expansion or decomposition with violent rupture of containers. ▸ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO). ▸ May emit acrid smoke. , , ,

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Minor Spills	▸ Clean up all spills immediately. ▸ Avoid breathing vapours and contact with skin and eyes. ▸ Control personal contact with the substance, by using protective equipment. ▸ Contain and absorb spill with sand, earth, inert material or vermiculite. ▸ Wipe up. ▸ Place in a suitable, labelled container for waste disposal.
Major Spills	Not Applicable
	Personal Protective Equipment advice is contained in Section 8 of the SDS.

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	Not Applicable
Other information	Not Applicable

Conditions for safe storage, including any incompatibilities

Suitable container	▸ **DO NOT** use mild steel or galvanised containers ▸ Polyethylene or polypropylene container. ▸ Packing as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	Formamide: ▸ may be light- and impact-sensitive ▸ reacts slowly with water forming hydrocyanic acid and ammonium formate - this reaction will be sped up by elevated temperatures or increase or decrease in pH ▸ reacts violently, possibly explosively, when mixed with furfuryl alcohol, hydrogen peroxide, nitromethane, phosphorus pentoxide, titanium nitrate ▸ is incompatible with strong oxidisers, acids, bases, alkali metal acetates, ammonia, cellulose acetate, cresols, iodine, isocyanates, lignin, metal chlorides, nitrates, oleum, phenols, polyvinyl alcohol, pyridines, starch, inorganic sulfates, sulfur trioxide, tannins ▸ attacks metals, including brass and copper, and their alloys, aluminium, cobalt, iron, lead, ▸ nickel, tin, zinc attacks some plastics, coatings, rubbers and glues ▸ thermal decomposition may produce ammonia, oxides of carbon and nitrogen, and hydrogen cyanide None known

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US ACGIH Threshold Limit Values (TLV)	formamide	Formamide	10 ppm	Not Available	Not Available	TLV® Basis: Eye & skin irr; kidney & liver dam
US NIOSH Recommended Exposure Limits (RELs)	formamide	Carbamaldehyde, Methanamide	15 mg/m3 / 10 ppm	Not Available	Not Available	[skin]

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
formamide	Formamide	13 ppm	13 ppm	600 ppm
sodium chloride	Chloride; (Chloride(1-); Chloride ions)	1 ppm	2.52 ppm	30 ppm
sodium chloride	Sodium chloride	11 mg/m3	120 mg/m3	1100 mg/m3
sodium citrate dihydrate	Citric acid, trisodium salt, dihydrate	9.3 mg/m3	100 mg/m3	610 mg/m3
sodium citrate dihydrate	Trisodium citrate	9.3 mg/m3	100 mg/m3	610 mg/m3

Ingredient	Original IDLH	Revised IDLH
water	Not Available	Not Available
formamide	Not Available	Not Available
dextran sulfate	Not Available	Not Available
sodium chloride	Not Available	Not Available
sodium citrate dihydrate	Not Available	Not Available

Continued...

Exposure controls

Appropriate engineering controls	Not Applicable
Personal protection	
Eye and face protection	‣ Safety glasses with side ‣ shields Chemical goggles. ‣ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	Not Applicable
Body protection	See Other protection below
Other protection	Not Applicable
Thermal hazards	Not Available

Respiratory protection

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

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Not Available

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

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	‣ 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

Continued...

Inhaled	Not Available
Ingestion	Not Available
Skin Contact	Not Available
Eye	Not Available
Chronic	Not Available

	TOXICITY	IRRITATION
Kreatech FISH Probe USA	Not Available	Not Available
water	Oral (rat) LD50: >90000 mg/kg[2]	Not Available
formamide	dermal (rat) LD50: >3000 mg/kg[1] Inhalation (rat) LC50: >21 mg/l4 h[1] Inhalation (rat) LC50: >3900 ppm/6H[2] Oral (rat) LD50: ca.3200 mg/kg[1]	Eye (rabbit): 23 mg
dextran sulfate	Not Available	Not Available
sodium chloride	Dermal (rabbit) LD50: >10000 mg/kg[1] Oral (rat) LD50: 3000 mg/kgd[2]	Eye (rabbit): 10 mg - moderate Eye (rabbit):100 mg/24h - moderate Skin (rabbit): 500 mg/24h - mild
sodium citrate dihydrate	dermal (rat) LD50: >2000 mg/kg[1] Oral (mouse) LD50: 5400 mg/kg[1]	Not Available

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

Kreatech FISH Probe USA	No significant acute toxicological data identified in literature search. The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
FORMAMIDE	for formamide: **Acute toxicity**: Toxicokinetic studies with rats or mice following a single oral administration showed that formamide was rapidly and completely absorbed in rats and mice, with peak plasma levels occurring within 2 h. The elimination half-life was about 15 h in rats and 4-6 h in mice. The metabolism and distribution of formamide were studied in rats and mice treated with 14C-labelled formamide via intravenous injection or inhalation exposure. The results showed that about 30% of formamide was excreted unchanged in urine within 72 h; about 30% (for rats) or 50% (for mice) was excreted as carbon dioxide in breath, and only a minor quantity (1-3%) was excreted in the faeces. It was suggested that cytochrome P450 2E1 was the primary enzyme of formamide metabolism **Repeat dose toxicity**: In repeated-dose short-term and subchronic toxicity studies, the main effects found in rats or mice include changes in haematological parameters, irrespective of route of exposure. In a subchronic study, an oral LOAEL of 40 mg/kg-bw per day was determined based on significant increases in haematocrit values, haemoglobin concentrations and erythrocyte counts in both male and female F344/N rats administered 0, 10, 20, 40, 80 or 160 mg formamide/kg-bw per day by gavage, 5 days/week for 14 weeks. The incidences of degeneration of the germinal epithelium of the testes and epididymis were significantly increased in males at the highest dose. The same oral LOAEL of 40 mg/kg-bw per day was also obtained based on a significant decrease in body weight gains in male B6C3F1 mice administered 0, 10, 20, 40, 80 or 160 mg formamide/kg-bw per day by gavage, 5 days/week for 14 weeks. Increased incidences of non-neoplastic lesions (hyperplasia and inflammation) were seen in pancreatic ducts at the dose of 80 mg/kg-bw per day. In a short-term study, a higher oral LOAEL of 113 mg/kg-bw per day was identified based on changes in haematological parameters, body weight loss, failure of reflexes, organ atrophy and tissue disintegration (gastrointestinal tract, testes, adrenal gland and kidney) in rats administered formamide at 0, 34, 113, 340 or 1130 mg/kg-bw per day by gavage. For dermal exposure, a LOAEL of 300 mg/kg-bw per day was identified based on haematological changes (increases in erythrocyte counts and hemoglobin) in rats treated with dermal applications of formamide at 0, 300, 1000 or 3000 mg/kg-bw per day for 90 days. At the highest dose level, clinical signs (e.g., erythema), pathological effects and an increased incidence of bilateral testicular tubular atrophy were seen. In a 2-week inhalation study, Crl:CD BR male rats were exposed to formamide at concentrations of 0, 190, 930 or 2800 mg/3 (6 h/day, 5 days/week). At the highest concentration (2800 mg/m3), microscopic lesions in the kidney (necrosis and regeneration of renal tubular epithelial cells) and an increase in kidney weights were observed. A lowest-observed-effect concentration (LOEC) of 930 mg/m3 (500 ppm) was identified, based on a significant decrease in the platelet count (haematological effect) **Developmental toxicity:** Formamide was found to be embryotoxic and teratogenic in several oral gavage studies using rabbits, rats and mice. The maternal toxicity caused by formamide included reduced food consumption, reduced body weight gain and decreased gravid uterine weight; foetal toxicity included reduced foetal weight and increased incidences of foetal death; and teratogenicity included skeletal malformations, cleft palate anencephaly and fused ribs. In rabbit studies, the lowest lowest-observed-adverse-effect levels (LOAELs) for maternal toxicity, embryo/foetal toxicity and teratogenicity were 79, 79 and 79 mg/kg-bw per day, respectively; embryotoxicity and teratogenicity were observed at maternally toxic doses. In rats, embryotoxicity (LOAEL = 100 mg/kg-bw per day) was seen in the absence of maternal toxicity (LOAEL = 200 mg/kg-bw per day for maternal toxicity). In mice, the lowest LOAELs for maternal toxicity, embryotoxicity and teratogenicity were 396, 198 and 198 mg/kg-bw per day, respectively; embryotoxicity and teratogenicity were seen in the absence of maternal toxicity. Among the test animals, the rabbit was the species that was the most sensitive to formamide in terms of developmental toxicity. Thus, the lowest oral LOAEL for developmental toxicity (maternal toxicity, embryo/foetal toxicity and teratogenicity) is identified to be 79 mg/kg-bw per day in rabbits.

Continued...

	Embryotoxicity or teratogenicity was also observed in experimental rats or mice through dermal exposure. In rats, the lowest LOAELs for maternal toxicity, embryotoxicity (early foetal deaths) and teratogenicity (distorted face or subcutaneous hemorrhage) were 600, 600 and 600 mg/kg-bw per day, respectively . In mice, the lowest dermal LOAEL for embryotoxicity was determined to be 300 mg/kg-bw per day based on an increase in early foetal deaths The foetal abnormalities were observed only at the high-dose exposure (>2800 mg/kg-bw per day) in mice **Reproductive toxicity:** The reproductive toxicity of formamide was evaluated using the Reproductive Assessment by Continuous Breeding protocols in Swiss CD-1 mice treated at concentrations of 0, 100, 350 and 750 mg/L (equivalent to 0, 16-32, 48-110 and 144-226 mg/kg-bw per day, respectively) in drinking water. Reproductive toxicity was observed at 750 mg/L (144-226 mg/kg-bw per day) in parental F0 and offspring F1 generations, and the critical effects included decreases in fertility rate and reduction in live litter size. A crossover mating experiment suggested that the reduced fertility rate may be due to impairment of reproduction in females. In addition, after offspring F1 mating, reduced offspring F2 litter size, increased days to litter, reduced relative ovarian weight and lengthened oestrous cycles were observed at 750 mg/L. The no-observed-adverse-effect level (NOAEL) for the reproductive toxicity of formamide was 350 mg/L (48-110 mg/kg-bw per day), and the LOAEL for the reproductive toxicity of formamide was 750 mg/L (144-226 mg/kg-bw per day) for both generations **Carcinogenicity:** The results from NTP studies showed no evidence of carcinogenic activity of formamide in male or female rats. There was clear evidence of carcinogenic activity of formamide in male mice, based on increased incidences of haemangiosarcoma in the liver; there was equivocal evidence of carcinogenic activity in female mice, based on marginally increased incidences of hepatocellular adenoma or carcinoma (significant only when adenoma and carcinoma were combined. No neoplastic lesions were observed in either male or female F344/N rats exposed to formamide at doses up to 80 mg/kg-bw per day. However, an increased incidence of bone marrow hyperplasia occurred in male rats. **Genotoxicity:** Formamide showed no evidence for mutagenicity in a series of short-term bioassays. Formamide was not mutagenic in Ames tests with several strains of *Salmonella typhimurium* or in a mutagenicity assay with *Escherichia coli* strain WPuvrA pKM101, with or without liver S9 metabolic activation. Formamide gave negative results in the sex-linked recessive lethal mutation assay in germ cells of male *Drosophila melanogaster* treated with formamide by either feeding or injection. In *in vivo* micronucleus tests, formamide did not induce increases in micronucleated erythrocytes in male or female mice treated with formamide (0-160 mg/kg-bw per day) by gavage for 3 months, although in another study, a dose-dependent increase in the number of polychromatic erythrocytes containing micronuclei was seen in bone marrow of mice exposed to formamide via intraperitoneal injection at higher doses (225-1800 mg/kg-bw), with significance at doses of 900 mg/kg-bw or higher. However, at the dose of 160 mg/kg-bw, increased incidences of lesions of several tissues/organs and decreased body weights were seen in mice, suggesting that the observed induction of micronuclei may be attributed to the cell damage. In a review paper on the biological effects of formamide, it was suggested that formamide caused cancer by a non-genotoxic mode of action . Based on the evidence of carcinogenicity observed in only one organ (liver), one sex (male) and one species (mice) and the conclusion that formamide is not mutagenic, the tumours observed in the experimental animals are unlikely to have resulted from direct interaction with genetic material. The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).
SODIUM CHLORIDE	Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. 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. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production. The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.
SODIUM CITRATE DIHYDRATE	for citric acid (and its inorganic citrate salts) Based on many experimental data in animals and on human experience, citric acid is of low acute toxicity. The NOAEL for repeated dose toxicity for rats is 1200 mg/kg/d. The major, reversible (sub)chronic toxic effects seem to be limited to changes in blood chemistry and metal absorption/excretion kinetics. Citric acid is not suspected of being a carcinogen nor a reprotoxic or teratogenic agent. The NOAEL for reproductive toxicity for rats is 2500 mg/kg/d. Further, it is not mutagenic *in vitro* and *in vivo*. Also, the sensitising potential is seen as low. In contrast, irritation, in particular of the eyes but also of the respiratory pathways and the skin, is the major toxicological hazard presented by citric acid
WATER & DEXTRAN SULFATE	No significant acute toxicological data identified in literature search.

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 required to make classification available*
✘ *– Data available but does not fill the criteria for classification*
⊘ *– Data Not Available to make classification*

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Environmental fate: Formamide may be released into the environment as a result of its production and use as solvent in manufacturing and processing plastics, non-aqueous electrolysis, and crystallization of pharmaceuticals and separation of chlorosilanes. According to Level III fugacity modeling, formamide will partition primarily in water and soil, depending on the compartment of release. Study shows that formamide is readily biodegradable in water, soil and sediment. Therefore, the compound is not expected to persist in soil and sediment. If released to air, formamide is expected to exist solely as a vapor in the ambient atmosphere based on the model of gas/particle partitioning of semivolatile organic compounds in the atmosphere. Vapor-phase formamide will be degraded in the atmosphere by reaction with photochemically produced hydroxyl radicals.
Ecotoxicity: Experimental data shows that formamide does not cause significant effect to aquatic organisms at low concentration. Likewise, modelled toxicity data shows that formamide is not expected to cause ecological harm at environmental concentrations.
Measured data:
Fish LC50 (96h): golden orfe (Leuciscus idus) 6.57 mg/l; zebra fish (Danio rerio) 9.14 mg/l

Continued...

Amphipod EC50 (96h): Chaetogammarus marinus 19 mg/l
Daphnia magna EC50 (48h): >500 mg/l
Algae EC50 (72h): Scenedesmus subspicatus >500 mg/l
Lemna minor EC50 (24h): 81.2 mg/l
Modelled data:
Fish LC50 (96h): 82.6 mg/l (ECOSAR)
Daphnia LC50 (48h): 69 mg/l (ECOSAR)
Shrimp LC50 (96h): 313 mg/l (ECOSAR)
Alga EC50 (96h): 35 mg/l (ECOSAR)

For the Alkali Metal Cyanides:
Atmospheric Fate: It is unknown if atmospheric photolysis is an important fate process for alkali metal cyanides. Hydrogen cyanide is very resistant to photolysis in normal sunlight. The most important reaction of hydrogen cyanide in air is the reaction with photochemically-generated hydroxyl radicals and subsequent rapid oxidation to carbon monoxide (CO) and nitric oxide (NO); photolysis and reaction with ozone are not important transformation processes and reaction with singlet oxygen is not a significant transformation process except at stratospheric altitudes.
The residence time for the reaction of hydrogen cyanide vapor with hydroxyl radicals in the atmosphere is approximately 2 years.
Terrestrial Fate: Low concentrations of cyanide in soil biodegrade under aerobic conditions. Under anaerobic conditions, cyanides ions will denitrify to gaseous nitrogen. Complexation reactions with metal ions may occur in soil and cyanide ions are sorbed by various natural media, including clays, biological solids and sediments. Hydrogen cyanide and the alkali metal cyanides are not likely to be strongly sorbed onto sediments and suspended solids. Cyanides are fairly mobile in soil. Mobility is lowest in low pH soils with high concentrations of free iron oxides, positively charged particles and clays and highest in soils with high pH, high concentrations of free calcium carbonate ($CaCO_3$), negatively charged particles and low clay content. In soils where cyanide levels are high enough to be toxic to microorganisms, this compound may leach into groundwater. Volatilization of hydrogen cyanide would be a significant loss mechanism from soil surfaces at a pH <9.2. Aquatic Fate: The alkali metal cyanides are very soluble in water and readily dissociate. Depending on the pH of the water, the resulting cyanide ion may form hydrogen cyanide or react with various metals. The proportion of hydrogen cyanide formed from soluble cyanides increases as the water pH decreases. Volatilization is the dominant mechanism for the removal of free cyanide from water and is most effective under conditions of high temperatures, high dissolved oxygen levels, and at increased concentrations of atmospheric carbon dioxide. Insoluble metal cyanides are not expected to degrade to hydrogen cyanide. Oxidation, hydrolysis, and photolysis are the three predominant chemical processes that may cause loss of simple cyanides in aquatic media. Certain cyanides are oxidized to isocyanates by strong oxidizing agents which may be further hydrolyzed to ammonia and carbon dioxide; however, it has not yet been determined whether this is a significant fate process in waters containing peroxy radicals. Hydrogen cyanide can be hydrolyzed to formamide, which is subsequently hydrolyzed to ammonium and formate ions. Volatilization is a significant and probably dominant fate process for hydrogen cyanide and the most common alkali metal cyanides (e.g., sodium and potassium cyanide) in surface water. Copper (I) cyanide is removed from water predominantly by sedimentation and biodegradation. Volatilization is not an important fate process for cyanide in groundwater and is expected to persist for considerably longer periods of time in underground aquifers than in surface water. Photocatalytic oxidation may not be significant in natural waters. In clear water, or at water surfaces, some metallocyanides may decompose to the cyanide ion by photodissociation and subsequently form hydrogen cyanide. Biodegradation is an important transformation process for cyanide in natural surface waters, and is dependent on such factors as cyanide concentration, pH, temperature, nutrient availability and microbial acclimation.
Ecotoxicity: Cyanide is toxic to microorganisms in low concentration; however, acclimation increases tolerance. Actinomyces, Alcaligenes, Arthrobacter, Bacillus, Micrococcus, Neisseria, Paracoccus, Pseudomonas, and Thiobacillus bacteria are particularly effective at cyanide degradation. Certain metal cyanide complexes may bioaccumulate in aquatic organisms. It is difficult to evaluate the toxicologic significance of bioaccumulation of metal cyanide complexes because these compounds are much less toxic than soluble hydrogen cyanide, sodium cyanide, or potassium cyanide. There is no evidence of biomagnification of cyanides in the food chain and accumulation of cyanide in food webs is not expected.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
water	LOW	LOW
formamide	LOW	LOW
sodium chloride	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
water	LOW (LogKOW = -1.38)
formamide	LOW (LogKOW = -1.51)
sodium chloride	LOW (LogKOW = 0.5392)

Mobility in soil

Ingredient	Mobility
water	LOW (KOC = 14.3)
formamide	HIGH (KOC = 1.498)
sodium chloride	LOW (KOC = 14.3)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	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. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. 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.

SECTION 14 TRANSPORT INFORMATION

Labels Required

Marine Pollutant	NO

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

Continued...

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

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

Transport in bulk according to Annex II of MARPOL 73 / 78 and the IBC code

Source	Ingredient	Pollution Category
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	formamide	Y

SECTION 15 REGULATORY INFORMATION

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

WATER(7732-18-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

FORMAMIDE(75-12-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

US - Alaska Limits for Air Contaminants
US - California Permissible Exposure Limits for Chemical Contaminants
US - Hawaii Air Contaminant Limits
US - Michigan Exposure Limits for Air Contaminants
US - Minnesota Permissible Exposure Limits (PELs)
US - Tennessee Occupational Exposure Limits - Limits For Air Contaminants
US - Vermont Permissible Exposure Limits Table Z-1-A Final Rule Limits for Air Contaminants
US - Vermont Permissible Exposure Limits Table Z-1-A Transitional Limits for Air Contaminants
US - Washington Permissible exposure limits of air contaminants
US ACGIH Threshold Limit Values (TLV)
US NIOSH Recommended Exposure Limits (RELs)
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

DEXTRAN SULFATE(9063-02-9) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

SODIUM CHLORIDE(7647-14-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

SODIUM CITRATE DIHYDRATE(6132-04-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

Federal Regulations

Superfund Amendments and Reauthorization Act of 1986 (SARA)

SECTION 311/312 HAZARD CATEGORIES

Immediate (acute) health hazard	NO
Delayed (chronic) health hazard	YES
Fire hazard	NO
Pressure hazard	NO
Reactivity hazard	NO

US. EPA CERCLA HAZARDOUS SUBSTANCES AND REPORTABLE QUANTITIES (40 CFR 302.4)

None Reported

State Regulations

US. CALIFORNIA PROPOSITION 65

None Reported

National Inventory	Status
Australia - AICS	Y
Canada - DSL	N (dextran sulfate)
Canada - NDSL	N (water; sodium chloride; formamide; sodium citrate dihydrate)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	N (dextran sulfate)
Japan - ENCS	N (water; dextran sulfate)
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	N (dextran sulfate)
USA - TSCA	Y
Legend:	*Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 OTHER INFORMATION

Continued...

Other information

Ingredients with multiple cas numbers

Name	CAS No
sodium chloride	14762-51-7, 16887-00-6, 7647-14-5

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.
A list of reference resources used to assist the committee may be found at: www.chemwatch.net

The (M)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.