Kre-HYB-40

Leica Biosystems Amsterdam

Chemwatch Hazard Alert Code: **3**

Version No: **1.5**
Safety Data Sheet (Conforms to Regulations (EC) No 453/2010)

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

1.1.Product Identifier

Product name	Kre-HYB-40
Synonyms	KBI-FHB, LK-111x, LK-084x
Other means of identification	Not Available

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

Relevant identified uses	Use according to manufacturer's directions.
Uses advised against	Not Applicable

1.3.Details of the manufacturer/importer

Registered company name	Leica Biosystems Amsterdam
Address	Vlierweg 20 Amsterdam Netherlands
Telephone	0031-20 6919181
Fax	0031-20 6963531
Website	www.leicabiosystems.com
Email	info.amsterdam@leicabiosystems.com

1.4.Emergency telephone number

Association / Organisation	Leica Biosystems Amsterdam
Emergency telephone numbers	0031-20 6919181
Other emergency telephone numbers	Not Available

SECTION 2 HAZARDS IDENTIFICATION

2.1.Classification of the substance or mixture

DSD classification	In case of mixtures, classification has been prepared by following DPD (Directive 1999/45/EC) and CLP Regulation (EC) No 1272/2008 regulations
DPD classification [1]	R61(2) May cause harm to the unborn child. *LIMITED EVIDENCE*
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI*
Classification according to regulation (EC) No 1272/2008 [CLP] [1]	Reproductive Toxicity Category 1B *LIMITED EVIDENCE*
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI*

2.2. Label elements

Continued...

CLP label elements	
SIGNAL WORD	**DANGER**

Hazard statement(s)

H360D	May damage the unborn child.

**LIMITED EVIDENCE*

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P280	Wear protective gloves/protective clothing/eye protection/face protection.

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 to authorised chemical landfill or if organic to high temperature incineration

2.3. Other hazards

formamide	Listed in the European Chemicals Agency (ECHA) Candidate List of Substances of Very High Concern for Authorisation

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

3.1.Substances

See 'Composition on ingredients' in Section 3.2

3.2.Mixtures

1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classification according to directive 67/548/EEC [DSD]	Classification according to regulation (EC) No 1272/2008 [CLP]
1.7732-18-5 2.231-791-2 3.Not Available 4.Not Available	20-50	water	Not Applicable	Not Applicable
1.75-12-7 2.200-842-0 3.616-052-00-8 4.01-2119496064-35-XXXX, 01-2119841903-36-XXXX, 01-2119856136-36-XXXX	20-50	formamide	R61 [2]	Repr. 1B; H360D *** [3]
1.9063-02-9 2.Not Available 3.Not Available 4.Not Available	5-20	dextran sulfate	Not Applicable	Not Applicable
1.7647-14-5 2.231-598-3 3.Not Available 4.01-2119485491-33-XXXX	<1	sodium chloride	R36/37/38R33? [1]	Skin Corrosion/Irritation Category 2, Eye Irritation Category 2, STOT - SE (Resp. Irr.) Category 3; H315, H319, H335 [1]
1.6132-04-3 2.200-675-3 3.Not Available 4.01-2119457027-40-XXXX	<1	sodium citrate	Not Applicable	Not Applicable
1.26628-22-8 2.247-852-1 3.011-004-00-7 4.01-2119457019-37-XXXX	<1	sodium azide	R28R32R50/53 [2]	Acute Tox. 2 *, Aquatic Acute 1, Aquatic Chronic 1; H300, H400, H410, EUH032 [3]

Legend:	*1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I; 3. Classification drawn from EC Directive 1272/2008 - Annex VI 4. Classification drawn from C&L*

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

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SECTION 4 FIRST AID MEASURES

4.1. Description of first aid measures

General	▸ Immediately give a glass of water. ▸ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor. ▸ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▸ Other measures are usually unnecessary. 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. If skin or hair contact occurs: ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
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. ▸ Other measures are usually unnecessary.
Ingestion	▸ Immediately give a glass of water. ▸ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

4.2 Most important symptoms and effects, both acute and delayed

	See Section 11

4.3. Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 FIREFIGHTING MEASURES

5.1. 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.

5.2. Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.

5.3. Advice for firefighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear breathing apparatus plus protective gloves in the event of a fire. ▸ Prevent, by any means available, spillage from entering drains or water courses. ▸ Use fire fighting procedures suitable for surrounding 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	▸ 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. Decomposes on heating and produces toxic fumes of; carbon dioxide (CO_2) nitrogen oxides (NOx) other pyrolysis products typical of burning organic material May emit poisonous fumes.

SECTION 6 ACCIDENTAL RELEASE MEASURES

6.1. Personal precautions, protective equipment and emergency procedures

	See section 8

Continued...

6.2. Environmental precautions

	See section 12

6.3. Methods and material for containment and cleaning up

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

6.4. Reference to other sections

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

SECTION 7 HANDLING AND STORAGE

7.1. 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 MSDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Fire and explosion protection	See section 5
Other information	Not Applicable

7.2. 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

PACKAGE MATERIAL INCOMPATIBILITIES

Not Available

7.3. Specific end use(s)

See section 1.2

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

8.1. Control parameters

DERIVED NO EFFECT LEVEL (DNEL)

Not Available

PREDICTED NO EFFECT LEVEL (PNEC)

Not Available

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes

Continued...

European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (English)	sodium azide	Sodium azide	0,1 mg/m3	0,3 mg/m3	Not Available	Skin
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	sodium azide	Sodium azide	0.1 mg/m3	0.3 mg/m3	Not Available	Skin
Netherlands Occupational Exposure Limits (Dutch)	sodium azide	Natriumazide	0,1 mg/m3	0,3 mg/m3	Not Available	Table A: Lijst met wettelijke grenswaarden

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	Citric acid, trisodium salt, dihydrate	9.3 mg/m3	100 mg/m3	610 mg/m3
sodium citrate	Trisodium citrate	9.3 mg/m3	100 mg/m3	610 mg/m3
sodium azide	Sodium azide	1.8 mg/m3	20 mg/m3	32 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	Not Available	Not Available
sodium azide	Not Available	Not Available

MATERIAL DATA

for formamide:
Formamide is somewhat less toxic than its congener, dimethylformamide (DMF) which has an identical TLV TWA. However, evidence of its cumulative effects suggests caution in departing too far from the DMF value. A skin notation reflects systemic effects arising from skin application. The TLV TWA is thought to be protective against the significant risk of eye and skin irritation and also to ensure a reduced risk of other health effects, such as testicular toxicity and teratogenicity, arising from exposure

8.2. Exposure controls

8.2.1. Appropriate engineering controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
Employers may need to use multiple types of controls to prevent employee overexposure.

General exhaust is adequate under normal operating conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. 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, degreasing 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, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 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 m/s (200-400 f/min.) for extraction of solvents generated in a tank 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.

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8.2.2. 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	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. 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. 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. ▸ Neoprene rubber gloves ▸ Wear chemical protective gloves, e.g. PVC. ▸ Wear safety footwear or safety gumboots, e.g. Rubber
Body protection	See Other protection below
Other protection	No special equipment needed when handling small quantities.
Thermal hazards	Not Available

Respiratory protection

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

8.2.3. Environmental exposure controls

See section 12

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

9.1. 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

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9.2. Other information

	Not Available

SECTION 10 STABILITY AND REACTIVITY

10.1.Reactivity	See section 7.2
10.2.Chemical stability	▸ Unstable in the presence of incompatible materials. ▸ Product is considered stable. ▸ Hazardous polymerisation will not occur.
10.3. Possibility of hazardous reactions	See section 7.2
10.4. Conditions to avoid	See section 7.2
10.5. Incompatible materials	See section 7.2
10.6. Hazardous decomposition products	See section 5.3

SECTION 11 TOXICOLOGICAL INFORMATION

11.1. Information on toxicological effects

Inhaled	The material is not thought to produce adverse health effects or irritation of the respiratory tract (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting. The intensity and time of exposure to hydrogen cyanide determines effects, symptoms. Short term inhalation of 20 to 40 ppm hydrogen cyanide may result in slight symptoms. Higher concentrations can cause death within minutes or hours; a concentration of 270 ppm can be fatal in one minute. Acute exposure to cyanides can cause death by cyanosis, asphyxia. At very low doses, symptoms of hydrogen cyanide exposure may be weakness, headaches, confusion, giddiness, dizziness, confusion, anxiety, nausea and vomiting. Normal blood pressure with rapid pulse is usual in mild cases. The respiratory rate varies with the intensity of exposure: rapid with mild exposure, or slow and gasping with severe exposure. Symptoms of mild exposure to hydrogen cyanide are completely reversed when exposure ceases. In severe cases, breathing is rapid and deep, then becomes slow and gasping. The victim may feel an irregular heartbeat and tightness in the chest. The skin appears bright pink or red. Fluid may fill the lungs and interfere with breathing. Unconsciousness, convulsions and death can quickly follow depending on the degree of exposure. Massive exposures may produce sudden collapse and death. concentration of 270 If death does not result, recovery is usually complete. There have however been a few reports of after-effects. These are similar to those seen in people deprived of oxygen, e.g. near-drowning victims
Ingestion	Rats fed formamide for up to ten days, at 1.5 g/kg, all died. Autopsy indicated a cumulative effect with changes characteristic of gastritis and malnutrition The material has **NOT** been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.
Skin Contact	The liquid may be miscible with fats or oils and may degrease the skin, producing a skin reaction described as non-allergic contact dermatitis. The material is unlikely to produce an irritant dermatitis as described in EC Directives . Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	Although the liquid is not thought to be an irritant (as classified by EC Directives), direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn).
Chronic	There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of: - clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects. Repeated exposure to formamide may affect the central nervous system and may cause liver and kidney damage. Rats treated with 3000 mg/kg formamide by semiocclusive patches to the skin, 6 hours/day, 5 days/week for 3 months showed general poor health including a number of organ weight changes. Rats receiving 300 mg/kg developed polycythaemia. Formamide is listed as a 'suggested' teratogen. This possible hazard should be discussed with all employees who may become pregnant. Although it has been reported that application of formamide to the skin of pregnant mice resulted in the inhibition of foetal growth and foetal malformations, gross foetal abnormalities were not observed following dermal application to rats. In mice such effects were weak and could only be produced by application of large volumes.

Kre-HYB-40	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
water	**TOXICITY**	**IRRITATION**
	Oral (rat) LD50: >90000 mg/kg[2]	Not Available
formamide	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >3000 mg/kg[1]	/kg
	Inhalation (rat) LC50: >21 mg/l4 h[1]	Eye (rabbit): 23 mg
	Inhalation (rat) LC50: >3900 ppm/6H[2]	kg
	Oral (rat) LD50: ca.3200 mg/kg[1]	

Continued...

dextran sulfate	

TOXICITY	IRRITATION
Not Available	Not Available

sodium chloride	

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >10000 mg/kg[1]	Eye (rabbit): 10 mg - moderate
Oral (rat) LD50: 3000 mg/kgd[2]	Eye (rabbit):100 mg/24h - moderate
	Skin (rabbit): 500 mg/24h - mild

sodium citrate	

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	Not Available
Oral (mouse) LD50: 5400 mg/kg[1]	

sodium azide	

TOXICITY	IRRITATION
dermal (rat) LD50: 50 mg/kg[2]	Not Available
Oral (rat) LD50: 27 mg/kg[2]	

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

Kre-HYB-40

No significant acute toxicological data identified in literature search.
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.
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

Continued...

	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.
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. 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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).

Continued...

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 a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
SODIUM CITRATE	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
SODIUM AZIDE	General anaesthesia, somnolence, convulsions, headache, irritability, arrhythmias, dyspnae, respiratory stimulation, diarrhoea recorded.
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*

CMR STATUS

SKIN	sodium azide	European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) - Skin	Skin

SECTION 12 ECOLOGICAL INFORMATION

12.1. Toxicity

NOT AVAILABLE

Ingredient	Endpoint	Test Duration	Effect	Value	Species	BCF
water	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
formamide	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
dextran sulfate	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
sodium chloride	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
sodium citrate	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
sodium azide	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available

for formamide:
log Kow : -1.51
Half-life (hr) air : 2.1
Henry's atm m3 /mol: 1.53E-08
BOD 5: nil
BCF : 0.042
Anaerobic effects : significant degradation
Environmental fate:
Based on its physical and chemical properties, the results of Level III fugacity modelling suggest that formamide will reside predominantly in water and soil, depending on the compartment of release.
Based on the available information, formamide is not persistent in the principal environmental media into which it tends to partition (water and soil) and is not bioaccumulative based on criteria defined in the *Persistence and Bioaccumulation Regulations* (Canada 2000). While formamide is persistent (i.e., is not rapidly transformed) in air, it will not remain in that medium but will partition into soil and water, where it is not persistent
Results of biodegradability tests show degradation ranging from 22.6% to 100% after 14 days in a Japanese Ministry of International Trade & Industry (MITI) test and 28 days in a dissolved organic carbon (DOC) die-away test, respectively. According to MITI criteria, formamide is classified as “well biodegradable”. These test data indicate that the ultimate degradation half-life in water is shorter than 182 days (6 months) and that the substance is considered to not persist in that environmental compartment. All of the aerobic probability models (BIOWIN submodels 1, 2, 5 and 6) suggest that this substance biodegrades quickly. The ultimate degradation model CATABOL predicts that formamide will undergo complete mineralization in a 28-day timeframe. That model predicted a 100% rate of biodegradation based on the Organisation for Economic Co-operation and Development (OECD) Test Guideline 301 ready biodegradation test (% BOD), which has been suggested to mean “not persistent” and having a half-life in water of <182 days, assuming first-order rate kinetics. Overall, modelled and empirical data indicate fast biodegradation of formamide in water.
Based on the empirical and modelled data and using an extrapolation ratio of 1:1:4 for a water:soil:sediment biodegradation half-life, the ultimate degradation half-life in soil and sediments is <60 days. This indicates that formamide is expected to not be persistent in soil and sediment.

Continued...

According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere, formamide is expected to exist solely as a vapour in the ambient atmosphere (SRC 1988). Vapour-phase formamide is degraded in the atmosphere by reaction with photochemically produced hydroxyl radicals. According to AOPWIN models, formamide has a calculated half-life in air of 5.348 days, assuming a hydroxyl radical concentration of 1.5 × 10+6 molecules/cm3. That concentration assumes an estimated global daylight average of 12 h, which is currently recommended by the US Environmental Protection Agency (EPA).
Formamide is predicted to hydrolyse very slowly at room temperature, although the rate of hydrolysis increases rapidly in the presence of acids or bases and is further accelerated at elevated temperatures.
Bioaccumulation: The modelled log Kow value of -1.15 for formamide suggests that this chemical has low potential to bioaccumulate in the environment
Ecotoxicity:
Fish LC50 (96 h): Leuciscus idus 6.5 mg/l; Danio rerio 9.135 mg/l (harmful to fish)
Daphnia magna EC50 (48 h): >500 mg/l (not harmful to aquatic invertebrates)
Algae EC50 (72 h): Scenedesmus subspicatus >500
Experimental ecotoxicological data indicate that formamide does not cause significant harm to aquatic organisms at low concentrations. The lowest modelled acute ecotoxicity value for fish was 1.3 mg/L, and measured acute toxicity values ranged from 81.2 mg/L for the duckweed *Lemna minor* to 19,031 mg/L for the amphipod *Chaetogammarus marinus* . Taking the modelled 1.3 mg/L result as the critical toxicity value (CTV) and applying an assessment factor of 100 to account for inter- and intraspecies variability and extrapolation from an estimate of acute effects to a no-effect concentration in the field gives a PNEC of 0.013 mg/L.
Dividing the PEC of 0.0056 mg/L, obtained using the conservative Industrial Generic Exposure Tool – Aquatic (Environment Canada 2008 a, b), by the PNEC of 0.013 mg/L yields a conservative risk quotient of 0.43, which indicates that formamide is not anticipated to cause ecological harm at environmental concentrations predicted from existing use pattern information.
The alkali metal cyanides (and other metal cyanides) are very soluble in water. As a result, they readily dissociate into their respective anions and cations when released into water. Depending on the pH of the water, the resulting cyanide ion may then form hydrogen cyanide or react with various metals in natural water. The proportion of hydrogen cyanide formed from soluble cyanides increases as the water pH decreases. At pH <7, >99% of the cyanide ions in water are converted to hydrogen cyanide. As the pH increases, cyanide ions in the water may form complex metallocyanides in the presence of excess cyanides; however, if metals are prevalent, simple metal cyanides are formed. Volatilization is the dominant mechanism for the removal of free cyanide. At pH >9.2, most of the free cyanide should exist as HCN, a volatile form of cyanide. Wide variations in the rate of volatilization are expected since this process is affected by a number of parameters such as temperature, pH, wind speed, and cyanide concentration. Volatilization of free cyanide from concentrated solutions is most effective under conditions of high temperatures, high dissolved oxygen levels, and at increased concentrations of atmospheric carbon dioxide
Unlike water-soluble alkali metal cyanides, insoluble metal cyanides are not expected to degrade to hydrogen cyanide. Cyanide occurs most commonly as hydrogen cyanide in water, although it can also occur as the cyanide ion, alkali and alkaline earth metal cyanides (potassium cyanide, sodium cyanide, calcium cyanide), relatively stable metallocyanide complexes (ferricyanide complex [Fe(CN)6]-3), moderately stable metallocyanide complexes (complex nickel and copper cyanide), or easily decomposable metallocyanide complexes (zinc cyanide [Zan(CN)2], cadmium cyanide [Cd(CN)2]). Oxidation, hydrolysis, and photolysis (photodegradation) are the three predominant chemical processes that may cause loss of simple cyanides in aquatic media.
Certain cyanides are oxidised to isocyanates by strong oxidising agents; the isocyanates may be further hydrolysed to ammonia and carbon dioxide. However, it has not yet been determined whether such oxidation and subsequent hydrolysis of isocyanate is a significant fate process in natural waters known to contain peroxy radicals. In water, hydrogen cyanide and cyanide ion exist in equilibrium with their relative concentrations primarily dependent on pH and temperature. At pH <8, >93% of the free cyanide in water will exist as undissociated hydrogen cyanide. Hydrogen cyanide can be hydrolysed to formamide, which is subsequently hydrolysed to ammonium and formate ions. However, the relatively slow rates of hydrolysis reported for hydrogen cyanide in acidic solution and of cyanides under alkaline conditions indicate that hydrolysis is not competitive with volatilisation and biodegradation for removal of free cyanide from ambient waters At pH <9.2, most of the free cyanide in solution should exist as hydrogen cyanide, a volatile cyanide form. On the basis of Henry's law constant and the volatility characteristics associated with various ranges of Henry's law constant, volatilization is a significant and probably dominant fate process for hydrogen cyanide in surface water. The most common alkali metal cyanides (e.g., sodium and potassium cyanide) may also be lost from surface water primarily through volatilization; whereas, the sparingly soluble metal cyanides such as copper (I) cyanide are removed from water predominantly by sedimentation and biodegradation. Because volatilisation is not an important fate process for cyanide in groundwater, cyanide would be expected to persist for considerably longer periods of time in underground aquifers than in surface water.
The significance of photolysis in the fate of cyanides in water has not been fully investigated. Hydrogen cyanide and cyanide ions in aqueous solution have been found to be very resistant to photolysis by natural sunlight, except under heterogeneous photocatalytic conditions. Photocatalytic oxidation may not be significant in natural waters, however, because of significant light reduction at increasingly greater depths. In clear water or at water surfaces, some metallocyanides, such as ferrocyanides and ferricyanides, 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 concentrations, pH, temperature, availability of nutrients, and acclimation of microbes. Although the cyanide ion is toxic to microorganisms at concentrations as low as 5-10 mg/L, acclimation increases tolerance to this compound. Mixed microorganisms in sewage sludge or activated sludge acclimated to cyanide also significantly biodegrade concentrations <=100 mg/L of most simple and complex cyanides. It is known that there is a natural attentuation of the cyanide ion and thiocyanide concentrations in waste waters, for example those obtained gold mill tails, that is due the acclimation of indigenous microflora in the tailings. A number of microorganisms have been identified that are capable of uptake, conversion, sorption, and/or precipitation of the cyanide ion, cyanate, and thiocyanate, including species of the genera, *Actinomyces, Alcaligenes, Arthrobacter, Bacillus, Micrococcus, Neisseria, Paracoccus, Pseudomonas*, and *Thiobacillus*. Some of these species, for example *Pseudomonas*, are capable of using the cyanide ion and thiocyanate as the sole source of carbon and nitrogen and therefore, are particularly effective at cyanide degradation. In fact, *Pseudomonas* is the basis of commercial applications for degrading the cyanide ion to ammonia and carbonate in waste waters generated in mining operations that use the cyanide ion to leach gold and other precious metals for low-grade ores. Sulfur transferases such as rhodanese are involved in substitution reactions that result in the conversion of the cyanide ion to the less toxic thiocyanate, whereas pyridoxal phosphate enzymes are involved in substitution/addition reactions that result in production of nitrile derivatives of a-amino acids. These organic nitriles may then be ultimately degraded via enzyme catalysed hydrolysis to either the corresponding amino acid and ammonia or the carboxylic acid and ammonia. The cyanide hydratase and cyanidase enzymes catalyse the hydrolysis of the cyanide ion to formamide or formic acid and ammonia, respectively In soil, cyanide present at low concentrations would biodegrade under aerobic conditions with the initial formation of ammonia, which would be converted to nitrite and nitrate in the presence of nitrifying bacteria. Under anaerobic conditions, the cyanides ion will denitrify to gaseous nitrogen. Upper limits of 200 and 2 ppm (mg/kg CN–), respectively, have been reported for uninhibited aerobic and anaerobic biodegradation of cyanide in soil; however, these limits have not been confirmed in other studies. Cyanide ions in soil are not involved in oxidation-reduction reactions but may undergo complexation reactions with metal ions in soil
Cyanides 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 because of their high water solubilities. Soluble metal cyanides may show somewhat stronger sorption than hydrogen cyanide, with the extent of sorption increasing with decreasing pH and increasing iron oxide, clay, and organic material contents of sediment and suspended solids. However, sorption is probably insignificant even for metal cyanides when compared to volatilisation and biodegradation. Cyanides are fairly mobile in soil. Mobility is lowest in soils with low pH and high concentrations of free iron oxides, positively charged particles, and clays (e.g., chlorite, kaolin, gibbsite), and highest in soils with high pH, high concentrations of free CaCO3 and negatively charged particles, and low clay content. Although cyanide has a low soil sorption capability, it is usually not detected in groundwater, probably because of fixation by trace metals through complexation or transformation by soil microorganisms. In soils where cyanide levels are high enough to be toxic to microorganisms (i.e., landfills, spills), this compound may leach into groundwater. Leaching of cyanide into a shallow aquifer has been demonstrated.
Volatilisation of hydrogen cyanide would be a significant loss mechanism for cyanides from soil surfaces at a pH <9.2.
Most cyanide in the atmosphere exists almost entirely as hydrogen cyanide gas, although small amounts of metal cyanides may be present as particulate matter in the air. Hydrogen cyanide is very resistant to photolysis at wavelengths of 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 where singlet oxygen is present in significant concentrations. The rate of hydroxyl radical reaction with hydrogen cyanide in the atmosphere depends on the altitude, and the rate of the reaction is at least an order of magnitude faster at lower tropospheric altitudes (0–8 km) than at upper tropospheric altitudes (10–12 km). Based on a reaction rate constant of 3x10-14 cm3/(molecule-sec) at 25 °C and assuming an average hydroxyl radical concentration of 5x105 molecules/cm3, the residence time for the reaction of hydrogen cyanide vapor with hydroxyl radicals in the atmosphere is approximately 2 years
There is some evidence that certain metal cyanide complexes bioaccumulate in aquatic organisms. Fish from water with soluble silver and copper cyanide complexes were found to have metal cyanides in their tissues at concentrations ranging up to 168 and 304 µg/g, respectively (wet or dry weight not specified). 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. Accumulation of cyanide in food webs is not expected, considering the rapid detoxification of cyanide by most species and the lethal effects of large doses of cyanide
DO NOT discharge into sewer or waterways.

12.2. Persistence and degradability

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

Continued...

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
water	LOW (LogKOW = -1.38)
formamide	LOW (LogKOW = -1.51)
sodium chloride	LOW (LogKOW = 0.5392)
sodium azide	LOW (LogKOW = 0.1631)

12.4. Mobility in soil

Ingredient	Mobility
water	LOW (KOC = 14.3)
formamide	HIGH (KOC = 1.498)
sodium chloride	LOW (KOC = 14.3)
sodium azide	HIGH (KOC = 1.342)

12.5.Results of PBT and vPvB assessment

	P	B	T
Relevant available data	Not Available	Not Available	Not Available
PBT and vPvB Criteria fulfilled?	Not Available	Not Available	Not Available

12.6. Other adverse effects

No data available

SECTION 13 DISPOSAL CONSIDERATIONS

13.1. 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 MSDS 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. 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. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible. ▸ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▸ Dispose of by: burial in a land-fill specifically licenced to accept chemical and / or pharmaceutical wastes or incineration in a licenced apparatus (after admixture with suitable combustible material). ▸ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.
Waste treatment options	Not Available
Sewage disposal options	Not Available

SECTION 14 TRANSPORT INFORMATION

Labels Required

Marine Pollutant	NO

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

14.1. UN number	Not Applicable
14.2. Packing group	Not Applicable
14.3. UN proper shipping name	Not Applicable
14.4. Environmental hazard	No relevant data

Continued...

14.5. Transport hazard class(es)	Class: Not Applicable Subrisk: Not Applicable
14.6. Special precautions for user	Hazard identification (Kemler): Not Applicable Classification code: Not Applicable Hazard Label: Not Applicable Special provisions: Not Applicable Explosive Limit and Limited Quantity Index: Not Applicable ERAP Index: Not Applicable Limited quantity: Not Applicable

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

14.1. UN number	Not Applicable
14.2. Packing group	Not Applicable
14.3. UN proper shipping name	Not Applicable
14.4. Environmental hazard	No relevant data
14.5. Transport hazard class(es)	ICAO/IATA Class: Not Applicable ICAO / IATA Subrisk: Not Applicable ERG Code: Not Applicable
14.6. Special precautions for user	Special provisions: Not Applicable Cargo Only Packing Instructions: Not Applicable Cargo Only Maximum Qty / Pack: Not Applicable Passenger and Cargo Packing Instructions: Not Applicable Passenger and Cargo Maximum Qty / Pack: Not Applicable Passenger and Cargo Limited Quantity Packing Instructions: Not Applicable Passenger and Cargo Limited Maximum Qty / Pack: Not Applicable

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

14.1. UN number	Not Applicable
14.2. Packing group	Not Applicable
14.3. UN proper shipping name	Not Applicable
14.4. Environmental hazard	Not Applicable
14.5. Transport hazard class(es)	IMDG Class: Not Applicable IMDG Subrisk: Not Applicable
14.6. Special precautions for user	EMS Number: Not Applicable Special provisions: Not Applicable Limited Quantities: Not Applicable

Inland waterways transport (ADN): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

14.1. UN number	Not Applicable
14.2. Packing group	Not Applicable
14.3. UN proper shipping name	Not Applicable
14.4. Environmental hazard	No relevant data
14.5. Transport hazard class(es)	Not Applicable: Not Applicable
14.6. Special precautions for user	Classification code: Not Applicable Limited quantity: Not Applicable Equipment required: Not Applicable

Continued...

Fire cones number	Not Applicable

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

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

water(7732-18-5) is found on the following regulatory lists	"European Customs Inventory of Chemical Substances ECICS (English)","European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)","EU REACH Regulation (EC) No 1907/2006 - Annex IV - Exemptions from the Obligation to Register in Accordance with Article 2(7)(a) (English)"
formamide(75-12-7) is found on the following regulatory lists	"EU REACH Regulation (EC) No 1907/2006 - Proposals to identify Substances of Very High Concern: Annex XV reports for commenting by Interested Parties","European Customs Inventory of Chemical Substances ECICS (English)","EU REACH Regulation (EC) No 1907/2006 - Annex XVII (Appendix 6) Toxic to reproduction: category 1B (Table 3.1)/category 2 (Table 3.2)","European Trade Union Confederation (ETUC) Priority List for REACH Authorisation","European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)","Netherlands Non-exhaustive list of reproductive toxins which additional registration requirement applicable under Article 4.2a, second paragraph of the Working Conditions Decree (Dutch)","European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances (updated by ATP: 31) - Reprotoxic Substances","Europe European Chemicals Agency (ECHA) Candidate List of Substances of Very High Concern for Authorisation","EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles","European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI","European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31","Europe AeroSpace and Defence Industries Association of Europe (ASD) REACH Implementation Working Group Priority Declarable Substances List (PDSL)"
dextran sulfate(9063-02-9) is found on the following regulatory lists	"Not Applicable"
sodium chloride(7647-14-5) is found on the following regulatory lists	"European Customs Inventory of Chemical Substances ECICS (English)","European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)"
sodium citrate(6132-04-3) is found on the following regulatory lists	"European Customs Inventory of Chemical Substances ECICS (English)","European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)"
sodium azide(26628-22-8) is found on the following regulatory lists	"EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)","European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Polish)","European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Slovenian)","European Customs Inventory of Chemical Substances ECICS (English)","European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Slovak)","European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (French)","European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Lithuanian)","European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Swedish)","European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Danish)","European Trade Union Confederation (ETUC) Priority List for REACH Authorisation","European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Maltese)","European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Italian)","European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)","European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Latvian)","European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (English)","European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Spanish)","European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (German)","European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI","European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Finnish)","European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31","European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Czech)","European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Hungarian)","European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Portuguese)","European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Romanian)","European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Greek)","Netherlands Occupational Exposure Limits (Dutch)","European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Dutch)","European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Estonian)","European Union (EU) First List of Indicative Occupational Exposure Limit Values (IOELVs) (Bulgarian)"

This safety data sheet is in compliance with the following EU legislation and its adaptations - as far as applicable - : 67/548/EEC, 1999/45/EC, 98/24/EC, 92/85/EC, 94/33/EC, 91/689/EEC, 1999/13/EC, Regulation (EU) No 453/2010, Regulation (EC) No 1907/2006, Regulation (EC) No 1272/2008 and their amendments as well as the following British legislation: - The Control of Substances Hazardous to Health Regulations (COSHH) 2002 - COSHH Essentials - The Management of Health and Safety at Work Regulations 1999

15.2. Chemical safety assessment

For further information please look at the Chemical Safety Assessment and Exposure Scenarios prepared by your Supply Chain if available.

National Inventory	Status
Australia - AICS	Y
Canada - DSL	N (dextran sulfate)
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

Continued...

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

Full text Risk and Hazard codes

H300	Fatal if swallowed
H315	Causes skin irritation
H319	Causes serious eye irritation
H335	May cause respiratory irritation
H360D ***	H360D ***
H400	Very toxic to aquatic life
H410	Very toxic to aquatic life with long lasting effects

R28	Very toxic if swallowed.
R32	Contact with acids liberates very toxic gas.
R33?	Cumulative effects may result following exposure*.
R36/37/38	Irritating to eyes, respiratory system and skin.
R50/53	Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
R61	May cause harm to the unborn child.

Other information

DSD / DPD label elements

Relevant risk statements are found in section 2.1

Indication(s) of danger	T

SAFETY ADVICE

S02	Keep out of reach of children.
S21	When using do not smoke.
S23	Do not breathe gas/fumes/vapour/spray.
S35	This material and its container must be disposed of in a safe way.
S36	Wear suitable protective clothing.
S37	Wear suitable gloves.
S40	To clean the floor and all objects contaminated by this material, use water and detergent.
S46	If swallowed, seek medical advice immediately and show this container or label.
S53	Avoid exposure - obtain special instructions before use.
S56	Dispose of this material and its container at hazardous or special waste collection point.

Ingredients with multiple cas numbers

Name	CAS No
sodium chloride	14762-51-7, 16887-00-6, 7647-14-5
sodium citrate	114456-61-0, 6132-04-3, 68-04-2, 6858-44-2

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.

For detailed advice on Personal Protective Equipment, refer to the following EU CEN Standards:
EN 166 Personal eye-protection
EN 340 Protective clothing
EN 374 Protective gloves against chemicals and micro-organisms
EN 13832 Footwear protecting against chemicals
EN 133 Respiratory protective devices

Continued...

end of SDS