Ranvet	Chemwatch Hazard Alert Code: 1
Chemwatch: 4642-34	Issue Date: 04/15/2021
Version No: 8.1	Print Date: 07/18/2022
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements	L.GHS.AUS.EN.E

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

Product Identifier

Product name	Ranvet's L-Carnitine
Chemical Name	Not Applicable
Synonyms	Not Available
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Use according to manufacturer's directions.	

Details of the supplier of the safety data sheet

Registered company name	Ranvet
Address	10-12 Green Street Banksmeadow NSW 2019 Australia
Telephone	+61 2 9666 1744
Fax	+61 2 9666 1755
Website	http://www.ranvet.com.au/other_msds.htm
Email	info@ranvet.com.au

Emergency telephone number

Association / Organisation	Ranvet
Emergency telephonenet number	+61 425 061 584
Other emergency telephonenet	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

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

ChemWatch Hazard Ratings

	Min	Max
Flammability	0	
Toxicity	1	0 = Minimum
Body Contact	0	1 = Low
Reactivity	1	2 = Moderate
Chronic	0	3 = High 4 = Extreme

Poisons Schedule	Not Applicable
Classification [1]	Not Applicable

Label elements

Hazard pictogram(s)	Not Applicable
Signal word	Not Applicable

Hazard statement(s)

Not Applicable

Precautionary statement(s) Prevention Not Applicable

Precautionary statement(s) Response

Not Applicable

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

Not Applicable

Not Applicable

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
541-15-1	10-30	L-carnitine
Not Available		vitamin B5 as,
81-13-0	0-10	d-panthenol
Not Available	>60	performance additives nonhazardous
Legend:	egend: 1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L * EU IOELVs available	

SECTION 4 First aid measures

Description of first aid measures		
Eye Contact	 If in eyes, hold eyelids apart and flush the eye continuously with running water. Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. Seek medical attention without delay; if pain persists or recurs seek medical attention. Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. 	
Skin Contact	If skin contact occurs: Immediately remove all contaminated clothing, including footwear. Flush skin and hair with running water (and soap if available). Seek medical attention in event of irritation. 	
Inhalation	 If fumes, aerosols or combustion products are inhaled remove from contaminated area. Other measures are usually unnecessary. 	
Ingestion	 For advice, contact a Poisons Information Centre or a doctor at once. Urgent hospital treatment is likely to be needed. If swallowed do NOT induce vomiting. If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. Observe the patient carefully. Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. Transport to hospital or doctor without delay. 	

Indication of any immediate medical attention and special treatment needed

For exposures to quaternary ammonium compounds;

For ingestion of concentrated solutions (10% or higher): Swallow promptly a large quantity of milk, egg whites / gelatin solution. If not readily available, a slurry of activated

- charcoal may be useful. Avoid alcohol. Because of probable mucosal damage omit gastric lavage and emetic drugs.
- For dilute solutions (2% or less): If little or no emesis appears spontaneously, administer syrup of lpecac or perform gastric lavage.
- If hypotension becomes severe, institute measures against circulatory shock.
- If respiration laboured, administer oxygen and support breathing mechanically. Oropharyngeal airway may be inserted in absence of gag reflex. Epiglottic or laryngeal edema may necessitate a tracheotomy.

Persistent convulsions may be controlled by cautious intravenous injection of diazepam or short-acting barbiturate drugs. [Gosselin et al, Clinical Toxicology of Commercial Products]

SECTION 5 Firefighting measures

Extinguishing media

- There is no restriction on the type of extinguisher which may be used.
- Use extinguishing media suitable for surrounding area.

Special hazards arising from the substrate or mixture

Fire Incompatibility	ty Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result		
Advice for firefighters			
Fire Fighting	 Alert Fire Brigade and tell them location and nature of hazard. Wear breathing apparatus plus protective gloves 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	 Non combustible. Not considered a significant fire risk, however containers may burn. Decomposition may produce toxic fumes of: carbon dioxide (CO2) nitrogen oxides (NOx) other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	 Clean up all spills immediately. Avoid contact with skin and eyes. Wear impervious gloves and safety goggles. Trowel up/scrape up. Place spilled material in clean, dry, sealed container. Flush spill area with water.
Major Spills	 Clear area of personnel and move upwind. Alert Fire Brigade and tell them location and nature of hazard. Wear breathing apparatus plus protective gloves. Prevent, by any means available, spillage from entering drains or water course. Stop leak if safe to do so. Contain spill with sand, earth or vermiculite. Collect recoverable product into labelled containers for recycling. Neutralise/decontaminate residue (see Section 13 for specific agent). Collect solid residues and seal in labelled drums for disposal. Wash area and prevent runoff into drains. After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling	
Safe handling	 Avoid all personal contact, including inhalation. Wear protective clothing when risk of exposure occurs. Use in a well-ventilated area. Prevent concentration in hollows and sumps. DO NOT enter confined spaces until atmosphere has been checked. DO NOT allow material to contact humans, exposed food or food utensils. Avoid contact with incompatible materials. When handling, DO NOT eat, drink or smoke. Keep containers securely sealed when not in use. Avoid physical damage to containers. Always wash hands with soap and water after handling. Work clothes should be laundered separately. Launder contaminated clothing before re-use. Use good occupational work practice. Observe manufacturer's storage and handling recommendations contained within this SDS. Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	 Store in original containers. Keep containers securely sealed. Store in a cool, dry, well-ventilated area. Store away from incompatible materials and foodstuff containers. Protect containers against physical damage and check regularly for leaks. Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	Pack size: 100g, 500g. Polyethylene or polypropylene container. Packing as recommended by manufacturer. Check all containers are clearly labelled and free from leaks.
Storage incompatibility	Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

Emergency Limits					
Ingredient	TEEL-1	TEEL-2		TEEL-3	
Ranvet's L-Carnitine	Not Available	Not Available		Not Available	
to use the st					
Ingredient	Original IDLH		Revised IDLH		
L-carnitine	Not Available		Not Available		
d-panthenol	Not Available		Not Available		

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating Occupational Exposure Band Limit			
L-carnitine	E ≤ 0.01 mg/m ³			
Notes:	Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.			

MATERIAL DATA

Exposure 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 (i	n still air)	0.25-0.5 m/s (50-100 f/min)		
	aerosols, fumes from pouring operations, intermittent conta drift, plating acid fumes, pickling (released at low velocity in		0.5-1 m/s (100-200 f/min.)		
Appropriate engineering controls	direct spray, spray painting in shallow booths, drum filling, generation into zone of rapid air motion)	conveyer loading, crusher dusts, gas discharge (active	1-2.5 m/s (200-500 f/min)		
	grinding, abrasive blasting, tumbling, high speed wheel ge 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 decrease 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 point, for example, should be a minimu 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 b factors of 10 or more when extraction systems are installed or used.				
Personal protection					
Eye and face protection	 Safety glasses with side shields; or as required, 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	 Wear chemical protective gloves, e.g. PVC. Wear safety footwear or safety gumboots, e.g. Rubber NOTE: The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. 				

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Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. Body protection See Other protection below Overalls. P.V.C apron. Barrier cream. Skin cleansing cream. Eye wash unit.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

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

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Not normally a hazard due to non-volatile nature of product The material is not thought to produce either adverse health effects or irritation of the respiratory tract following inhalation (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. The very bitter taste is likely to give early warning of accidental ingestion. Concentrated solutions of many cationics may cause corrosive damage to mucous membranes and the oesophagus. Nausea and vomiting (sometimes bloody) may follow ingestion. Serious exposures may produce an immediate burning sensation of the mouth, throat and abdomen with profuse salivation, ulceration of mucous membranes, signs of circulatory shock (hypotension, laboured breathing, and cyanosis) and a feeling of apprehension, restlessness, confusion and weakness. Weak convulsive movements may precede central nervous system depression. Erosion, ulceration, and petechial haemorrhage may occur through the small intestine with glottic, brain and pulmonary oedema. Death may result from asphyxiation due to paralysis of the muscles of respiration or cardiovascular collapse. Fatal poisoning may arise even when the only pathological signs are visceral congestion, swallowing, mild pulmonary oedema or varying signs of gastrointestinal irritation. Individuals who survive a period of severe hypertension may develop kidney failure. Cloudy swelling, patchy necrosis and fatty infiltration in such visceral organs as the heart, liver and kidneys shows at death. Rats fed repeatedly on a similar material (a C12-C16 alkyl derivative), over several weeks, died of inanition associated with chronic diarrhoea; at autopsy the only lesion found was focal haemorrhagic necrosis of the gastric mucosa. Repeated administration of 0.5% in the diet was lethal to rats, while 25 mg/kg was lethal to dogs; toxic signs in dogs included conditioned salivation, vomiting, enteritis, pulmonary haemorrhage and inflammation and sloughing of the mucosa.
Skin Contact	The material is not thought to produce adverse health effects or skin irritation following contact (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting.

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 material 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). Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant number of individuals at a greater frequency than would be expected from the response of a normal population. Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking. There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals. Respiratory sensitisation may result in allergic/asthma like responses; from coughing and minor breathing difficulties to bronchitis with wheezing, gasping.

Ranvet's L-Carnitine	TOXICITY Not Available	IRRITATION Not Available	
L-carnitine	TOXICITY Not Available	IRRITATION Not Available	
d-panthenol	TOXICITY dermal (rat) LD50: >2000 mg/kg ^[1] Oral (Mouse) LD50; 15000 mg/kg ^[2]	IRRITATION Eye (rabbit): 0.5 mg - mild Eye: no adverse effect observed (not irritating) ^[1] Skin (rabbit): 500 mg/4h - mild Skin: no adverse effect observed (not irritating) ^[1]	
Legend:	1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances		

Long-chain acyl-CoAs are converted to acylcarnitines by carnitine palmitoyltransferase 1 (CPT1), which exchanges the CoA moiety for carnitine.CPT1 is an important regulator of FAO flux. Long-chain acyl-CoA dehydrogenase deficiencies in certain muscles may produce chronic myopathy in middle aged patients and celiac disease. Medium-chain acyl-CoA dehydrogenase deficiency, often known as MCAD deficiency or MCADD, is a disorder of fatty acid oxidation that impairs the body's ability to break down medium-chain fatty acids into acetyl-CoA. The disorder is characterized by hypoglycemia and sudden death without timely intervention, most often brought on by periods of fasting or vomiting. The enzyme MCAD is responsible for the dehydrogenation step of fatty acids with chain lengths between 6 and 12 carbons as they undergo beta-oxidation in the mitochondria. Fatty acid beta-oxidation provides energy after the body has used up its stores of glucose and glycogen. This	L-CARNITINE	Ashma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-altergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of previsios airways disease in a non-atopic individual, with sudden onset of previsios airways disease in a non-atopic individual, with sudden onset of previsios airways disease in a non-atopic individual, with sudden on set of previsios to be serve foronial hyperreactivity on methacholine challenge testing, and the lack of minimal hymphocytic inflammation of exposure to the initiating substance. On the other hand, industrial bronchis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance. On the other hand, industrial bronchis and a disorder that occurs as a result of exposure dues of energy production in the budy as carnitine acets y-L-carnitine, and propionyl-L-carnitine. It is most accumulated in cardace and skeletal muscles as it accounts for 0.1% of its dry matter. The body synthesizes enough carnitine from hysine side chains to keep up with the needs of energy production in the body as carnitine acid as at transporter of long-chain latty acids into the mitochondria to be oxidized and produce energy.
Long-chain acyl-CoAs are converted to acylcarnitines by carnitine palmitoyltransferase 1 (CPT1), which exchanges the CoA moiety for carnitine.CPT1 is an important regulator of FAO flux. Long-chain acyl-CoA dehydrogenase deficiencies in certain muscles may produce chronic myopathy in middle aged patients and celiac disease. Medium-chain acyl-CoA dehydrogenase deficiency, often known as MCAD deficiency or MCADD, is a disorder of fatty acid oxidation that impairs the body's ability to break down medium-chain fatty acids into acetyl-CoA. The disorder is characterized by hypoglycemia and sudden death without timely intervention, most often brought on by periods of fasting or vomiting. The enzyme MCAD is responsible for the dehydrogenation step of fatty acids with chain lengths between 6 and 12 carbons as they undergo		or the OCTN2 transporter aetiologically causes a carnitine deficiency that results in poor intestinal absorption of dietary L-carnitine, its impaired re-absorption by the kidney and, consequently, in increased urinary loss of L-carnitine. Determination of the qualitative pattern of acylcarnitines can be of diagnostic and therapeutic importance. The betaine structure of carnitine requires special analytical procedures for recording. The ionic nature of L-carnitine causes a high water solubility which decreases with increasing chain length of the ester group in the acylcarnitines. Therefore, the distribution of L-carnitine and acylcarnitines in various organs is defined by their function and their physico-chemical properties as well. High performance liquid chromatography (HPLC) permits screening for free and total carnitine, as well as complete quantitative acylcarnitine
		Long-chain acyl-CoAs are converted to acylcarnitines by carnitine palmitoyltransferase 1 (CPT1), which exchanges the CoA moiety for carnitine.CPT1 is an important regulator of FAO flux. Long-chain acyl-CoA dehydrogenase deficiencies in certain muscles may produce chronic myopathy in middle aged patients and celiac disease. Medium-chain acyl-CoA dehydrogenase deficiency, often known as MCAD deficiency or MCADD, is a disorder of fatty acid oxidation that impairs the body's ability to break down medium-chain fatty acids into acetyl-CoA. The disorder is characterized by hypoglycemia and sudden death without timely intervention, most often brought on by periods of fasting or vomiting. The enzyme MCAD is responsible for the dehydrogenation step of fatty acids with chain lengths between 6 and 12 carbons as they undergo

oxidation typically occurs during periods of extended fasting or illness when caloric intake is reduced, and energy needs are increased. Prior to expanded newborn screening, MCADD was an underdiagnosed cause of sudden death in infants. Individuals who have been identified prior to the onset of symptoms have an excellent prognosis. MCADD is most prevalent in individuals of Northern European Caucasian descent, with an incidence of 1:4000 to 1:17,000 depending on the population. Treatment of MCADD is mainly preventative, by avoiding fasting and other situations where the body relies on fatty acid oxidation to supply energy. Short-chain acyl-CoA dehydrogenase deficiency (SCADD) in human infants has been associated severe dysmorphic facial features, feeding difficulties / failure to thrive, metabolic acidosis, ketotic hypoglycemia, lethargy, developmental delay, seizures, hypotonia, dystonia, and myopathy. However, individuals with no symptoms were also reported.. SCADD is now viewed as a biochemical phenotype rather than a disease. As with other fatty acid oxidation deficiencies, characteristic biochemical findings of SCADD may be absent except during times of physiologic stress such as fasting and illness. Stearov/carnitine, also known as acylcarnitine C18:0, is a fatty ester lipid molecule. It is found in significantly greater amounts of patients with carnitine palmitoyltransferase (CPT) II deficiency when compared to controls Stearoylcarnitine is also found to be associated with celiac disease, another inborn error of metabolism. The carnitine palmitoyltransferase (CPT: EC 2.3.1.21) enzyme system, in conjunction with acyl-CoA synthetase and carnitine/acylcarnitine translocase, provides the mechanism whereby long-chain fatty acids are transferred from the cytosol to the mitochondrial matrix to undergo beta-oxidation

At first glance, correlations of acylcarnitines to surrogate markers of insulin resistance fit with mitochondrial overload and incomplete fatty cid oxidation (FAO). Acylcarnitines, however, also directly reflect the oxidation rate of fatty acid (FA) and amino acids, which is supported by human nutritional intervention studies. Long-chain FA such as palmitic acid were associated with insulin resistance, making a role for long-chain acylcarnitines such as C16 in insulin resistance conceivable.

Acylcarnitines with longer chain lengths are associated with insulin resistance, which seems logic in the light of known effects of long-chain FAs on insulin signaling. Indeed, acylcarnitines can reside in cell membranes because they are amphipathic molecules. Increasing chain length favours partitioning into the membrane phase (e.g., C16- and 18-carnitine)). It is interesting to speculate that long-chain acylcarnitines can interfere with insulin signaling directly within the cell membrane . In contrast, acylcarnitines seem to track with higher lipid flux and as such may only indicate higher FAO.

The concept of lipotoxicity is generally accepted in the field of obesity-induced impairment of insulin sensitivity, and more and more attention has attributed to intramitochondrial alterations and impairments in FAO, thereby focusing on acylcarnitines . Collected evidence shows that acylcarnitines have distinct functions in mitochondrial lipid metabolism. The transmembrane export of acylcarnitines suggests that they not only prevent the accumulation of noxious acyl-CoAs, but also reduce CoA trapping, which is crucial for many metabolic pathways

To guarantee continuous energy supply, the human body oxidizes considerable amounts of fat besides glucose. L-carnitine transports activated long-chain FAs from the cytosol into the mitochondrion and is therefore essential for FAO.

Once inside the cell, FAs are activated by esterification to coenzyme A (CoA). Then, the carnitine shuttle transports long-chain acyl-CoAs into mitochondria via their corresponding carnitine ester Acetyl-CoA (acetyl coenzyme A) is a molecule that participates in many biochemical reactions in protein, carbohydrate and lipid metabolism. Its main function is to deliver the acetyl group to the citric acid cycle (Krebs cycle) to be oxidized for energy production.

For quaternary ammonium compounds (QACs):

Quaternary ammonium compounds (QACs) are cationic surfactants. They are synthetic organically tetra-substituted ammonium compounds, where the R substituents are alkyl or heterocyclic radicals (where hydrogen atoms remain unsubstituted, the term "secondary- or "tertiaryammonium compounds" is preferred).

A common characteristic of these synthetic compounds is that one of the R's is a long-chain hydrophobic aliphatic residue The cationic surface active compounds are in general more toxic than the anionic and non-ionic surfactants. The positively-charged cationic portion is the functional part of the molecule and the local irritation effects of QACs appear to result from the quaternary ammonium cation. Due to their relative ability to solubilise phospholipids and cholesterol in lipid membranes, QACs affect cell permeability which may lead to cell death. Further QACs denature proteins as cationic materials precipitate protein and are accompanied by generalised tissue irritation. It has been suggested that the experimentally determined decrease in acute toxicity of QACs with chain lengths above C16 is due to decreased water solubility.

In general it appears that QACs with a single long-chain alkyl groups are more toxic and irritating than those with two such substitutions, The straight chain aliphatic QACs have been shown to release histamine from minced guinea pig lung tissue However, studies with benzalkonium chloride have shown that the effect on histamine release depends on the concentration of the solution. When cell suspensions (11% mast cells) from rats were exposed to low concentrations, a decrease in histamine release was seen. When exposed to high concentrations the opposite result was obtained.

In addition, QACs may show curare-like properties (specifically benzalkonium and cetylpyridinium derivatives, a muscular paralysis with no involvement of the central nervous system. This is most often associated with lethal doses Parenteral injections in rats, rabbits and dogs have resulted in prompt but transient limb paralysis and sometimes fatal paresis of the respiratory muscles. This effect seems to be transient. From human testing of different QACs the generalised conclusion is obtained that all the compounds investigated to date exhibit similar toxicological properties.

Acute toxicity: Studies in rats have indicated poor intestinal absorption of QACs. Acute toxicity of QACs varies with the compound and, especially, the route of administration. For some substances the LD50 value is several hundreds times lower by the i.p. or i.v. than the oral route, whereas toxicities between the congeners only differ in the range of two to five times.

At least some QACs are significantly more toxic in 50% dimethyl sulfoxide than in plain water when given orally

Probably all common QAC derivatives produce similar toxic reactions, but as tested in laboratory animals the oral mean lethal dose varies with the compound .

Oral toxicity: LD50 values for QACs have been reported within the range of 250-1000 mg/kg for rats, 150-1000 mg/kg for mice, 150-300 mg/kg for guinea pigs and about 500 mg/kg b.w. for rabbits and dogs. The ranges observed reflect differences in the study designs of these rather old experiments as well as differences between the various QACs.

The oral route of administration was characterised by delayed deaths, gastrointestinal lesions and respiratory and central nervous system depression. It was also found that given into a full stomach, the QACs lead to lower mortality and fewer gastrointestinal symptoms. This support the suggestion of an irritating effect

Dermal toxicity: It has been concluded that the maximum concentration that did not produce irritating effect on intact skin is 0.1%. Irritation became manifest in the 1-10% range. Concentrations below 0.1% have caused irritation in persons with contact dermatitis or broken skin. Although the absorption of QACs through normal skin probably is of less importance than by other routes , studies with excised guinea pig skin have shown that the permeability constants strongly depends on the exposure time and type of skin

Sensitisation: Topical mucosal application of QACs may produce sensitisation. Reports on case stories and patch test have shown that compounds such as benzalkonium chloride, cetalkonium chloride and cetrimide may possibly act as sensitisers. However, in general it is suggested that QACs have a low potential for sensitising man It is difficult to distinguish between an allergic and an irritative skin reaction due to the inherent skin irritating effect of QACs.

Long term/repeated exposure:

Inhalation: A group of 196 farmers (with or without respiratory symptoms) were evaluated for the relationship between exposure to QACs (unspecified, exposure levels not given) and respiratory disorders by testing for lung function and bronchial responsiveness to histamine. After histamine provocation statistically significant associations were found between the prevalence of mild bronchial responsiveness (including asthma-like symptoms) and the use of QACs as disinfectant. The association seems even stronger in people without respiratory symptoms. Genetic toxicity: QACs have been investigated for mutagenicity in microbial test systems. In Ames tests using Salmonella typhimurium with and without metabolic activation no signs of mutagenicity has been observed. Negative results were also obtained in E. coli reversion and B. subtilis rec assays. However, for benzalkonium chloride also positive and equivocal results were seen in the B. subtilis rec assays. Most undiluted cationic surfactants satisfy the criteria for classification as Harmful (Xn) with R22 and as Irritant (Xi) for skin and eyes with R38 and R41.

Lachrymation, convulsions recorded.

D-PANTHENOL	The material may be irritating to the eye, with prolonged contact causing 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.		
Acute Toxicity	×	Carcinogenicity	×
Skin Irritation/Corrosion	×	Reproductivity	×
Serious Eye Damage/Irritation	×	STOT - Single Exposure	×
Respiratory or Skin sensitisation	×	STOT - Repeated Exposure	×
Mutagenicity	×	Aspiration Hazard	×
			ot available or does not fill the criteria for classification le to make classification



Toxicity

Ranvet's L-Carnitine	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
L-carnitine	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
d-panthenol	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	72h	Algae or other aquatic plants	100mg/l	2
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
d-panthenol			Fish	>1000mg/l	2
d-panthenol	LC50	96h	Fish		

Ecotox database - Aquatic Toxicity Data 2. Ecotor Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data

Toxic to aquatic organisms.

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites. **DO NOT** discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
d-panthenol	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation	
d-panthenol	LOW (LogKOW = -1.9222)	

Mobility in soil

Ingredient	Mobility	
d-panthenol	LOW (KOC = 10)	

SECTION 13 Disposal considerations

Waste treatment methods	
Product / Packaging disposal	 Containers may still present a chemical hazard/ danger when empty. Return to supplier for reuse/ recycling if possible. Otherwise: If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. Where possible retain label warnings and SDS and observe all notices pertaining to the product.

SECTION 14 Transport information

 Labels Required

 Marine Pollutant
 NO

 HAZCHEM
 Not Applicable

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

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

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

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

Not Applicable

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

Product name	Group	
L-carnitine	Not Available	
d-panthenol	Not Available	

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
L-carnitine	Not Available
d-panthenol	Not Available

SECTION 15 Regulatory information

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

L-carnitine is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

d-panthenol is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

National Inventory Status

National Inventory	Status	
Australia - AIIC / Australia Non-Industrial Use	Yes	
Canada - DSL	No (L-carnitine)	
Canada - NDSL	No (L-carnitine; d-panthenol)	
China - IECSC	Yes	
Europe - EINEC / ELINCS / NLP	Yes	
Japan - ENCS	Yes	
Korea - KECI	No (L-carnitine)	
New Zealand - NZIoC	Yes	
Philippines - PICCS	Yes	
USA - TSCA	No (L-carnitine)	
Taiwan - TCSI	Yes	
Mexico - INSQ	Yes	
Vietnam - NCI	Yes	
Russia - FBEPH	Yes	
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.	

SECTION 16 Other information

Revision Date	04/15/2021
Initial Date	07/28/2005

SDS Version Summary

Version	Date of Update	Sections Updated
7.1	11/01/2019	One-off system update. NOTE: This may or may not change the GHS classification
8.1	04/15/2021	Classification change due to full database hazard calculation/update.

Other information

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

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

Definitions and abbreviations

PC-TWA: Permissible Concentration-Time Weighted Average

PC-STEL: Permissible Concentration-Short Term Exposure Limit

IARC: International Agency for Research on Cancer ACGIH: American Conference of Governmental Industrial Hygienists STEL: Short Term Exposure Limit TEEL: Temporary Emergency Exposure Limit_\circ IDLH: Immediately Dangerous to Life or Health Concentrations ES: Exposure Standard OSF: Odour Safety Factor NOAEL :No Observed Adverse Effect Level LOAEL: Lowest Observed Adverse Effect Level TLV: Threshold Limit Value LOD: Limit Of Detection OTV: Odour Threshold Value BCF: BioConcentration Factors BEI: Biological Exposure Index AIIC: Australian Inventory of Industrial Chemicals DSL: Domestic Substances List NDSL: Non-Domestic Substances List IECSC: Inventory of Existing Chemical Substance in China EINECS: European INventory of Existing Commercial chemical Substances ELINCS: European List of Notified Chemical Substances NLP: No-Longer Polymers ENCS: Existing and New Chemical Substances Inventory KECI: Korea Existing Chemicals Inventory NZIoC: New Zealand Inventory of Chemicals PICCS: Philippine Inventory of Chemicals and Chemical Substances TSCA: Toxic Substances Control Act TCSI: Taiwan Chemical Substance Inventory INSQ: Inventario Nacional de Sustancias Químicas NCI: National Chemical Inventory FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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