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

Product identifier

Product name	24·7 Smartrace Adult Sheep Oral Boluses
Chemical name	Not Applicable
Synonyms	Not Applicable
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains zinc)
Chemical formula	Not Applicable
Other means of identification	Not Applicable

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

Relevant identified uses	Intraruminal boluses to supplement the trace elements iodine, cobalt and selenium for production and/or breeding in adult sheep over 40 kg liveweight.

Details of the supplier of the safety data sheet

	Manufacturer	**Distributor in Australia**
Registered company name	Agrimin Ltd.	Pacific Biologics Pty. Ltd
Address	Humberside Airport, Kirmington, DN39 6YH, UK	35 Beach Street, Kippa-Ring, QLD 4021, Australia
Telephone	+44 1652 688046	+61 7 3283 5077
Fax	+44 1652 688049	+61 7 3283 5088
Website	www.agrimin.co.uk	www.pacificbiologics.com.au
Email	info@agrimin.co.uk	factory@pacificbiologics.com.au

Emergency telephone number

	Manufacturer	**Distributor in Australia**
Association / Organisation	Agrimin Ltd.	Pacific Biologics Pty. Ltd.
Emergency telephone numbers	+44 1652 688046	+61 7 3283 5077
Other emergency telephone numbers	Not Available	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

HAZARDOUS CHEMICAL. DANGEROUS GOODS. According to the WHS regulations and the ADG Code.

Poisons Schedule	Not Applicable
Classification[1]	Serious Eye Damage/Eye Irritation Category 2A, Carcinogenicity Category 1B, Reproductive Toxicity Category 1B, Hazardous to the Aquatic Environment Long-Term Hazard Category 1
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 – Annex VI*

Label elements

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

Hazard statement(s)

H319	Causes serious eye irritation.
H350	May cause cancer.
H360F	May damage fertility.
H410	Very toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P337+P313	If eye irritation persists: Get medical advice/attention.
P391	Collect spillage.

Precautionary statement(s) Storage

P405	Store locked up.

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waster collection point in accordance with any local regulation.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No.	%[weight]	Name
7440-66-6	>60	Zinc
8002-74-2	>30	Paraffin wax
7789-80-2	1-10	Calcium iodate
51839-24-8	<1	Cobalt (II) carbonate basic
13410-001-0	<1	Sodium selenate, anhydrous
Not Available	balance	Ingredients determined not to be hazardous
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 – Annex VI*	

SECTION 4 First aid measures

Description of first aid measures

Eye contact	If this product comes into contact with the eyes: – Wash out immediately with fresh running water. – Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. – Seek medical attention without delay; if pain persists or recurs seek medical attention. – Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin contact	If skin contact occurs: – Immediately remove all contaminated clothing, including footwear. – Flush skin and hair with running water (and soap if available). – Seek medical attention in event of irritation.
Inhalation	– If fumes or combustion products are inhaled remove from contaminated area. – Lay patient down. Keep warm and rested. – Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. – Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. – Transport to hospital, or doctor, without delay.
Ingestion	– **IF SWALLOWED, REFER FOR MEDICAL ATTENTION, WHERE POSSIBLE, WITHOUT DELAY.** – For advice, contact a Poisons Information Centre or a doctor. – Urgent hospital treatment is likely to be needed. – In the meantime, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition. – If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS should be provided. Further action will be the responsibility of the medical specialist. – If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS. **Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:**

	– **INDUCE** vomiting with fingers down the back of the throat, **ONLY IF CONSCIOUS**. Lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. **NOTE:** Wear a protective glove when inducing vomiting by mechanical means.

Indication of any immediate medical attention and special treatment needed

- Absorption of zinc compounds occurs in the small intestine.
- The metal is heavily protein bound.
- Elimination results primarily from faecal excretion.
- The usual measure for decontamination (Ipecac Syrup, lavage, charcoal or cathartics) may be administered, although patients usually have sufficient vomiting not to require them
- $CaNa_2EDTA$ has been used successfully to normalise zinc levels and is the agent of choice

[Ellenhorn and Barceloux: Medical Technology]

Copper, magnesium, aluminium, antimony, iron, manganese, nickel, zinc (and their compounds) in welding, brazing, galvanising or smelting operations all give rise to thermally produced particulates of smaller dimension than may be produced if the metals are divided mechanically. Where insufficient ventilation or respiratory protection is available these particulates may produce "metal fume fever" in workers from an acute or long-term exposure.

- Onset occurs in 4-6 hours generally on the evening following exposure. Tolerance develops in workers but may be lost over the weekend. (Monday Morning Fever)
- Pulmonary function tests may indicate reduced lung volumes, small airway obstruction and decreased carbon monoxide diffusing capacity but these abnormalities resolve after several months.
- Although mildly elevated urinary levels of heavy metal may occur they do not correlate with clinical effects.
- The general approach to treatment is recognition of the disease, supportive care and prevention of exposure.
- Seriously symptomatic patients should receive chest x-rays, have arterial blood gases determined and be observed for the development of tracheobronchitis and pulmonary oedema.

[Ellenhorn and Barceloux: Medical Toxicology]

SECTION 5 Firefighting measures

Extinguishing media

- **DO NOT** use halogenated fire extinguishing agents.

Metal dust fires need to be smothered with sand, inert dry powders.
DO NOT USE WATER, CO_2 or FOAM.

- Use DRY sand, graphite powder, dry sodium chloride based extinguishers, G-1 or Met L-X to smother fire.
- Confining or smothering material is preferable to applying water as chemical reaction may produce flammable and explosive hydrogen gas.
- Chemical reaction with CO2 may produce flammable and explosive methane.
- If impossible to extinguish, withdraw, protect surroundings and allow fire to burn itself out.

Special hazards arising from the substrate or mixture

Fire incompatibility	– Reacts with acids producing flammable / explosive hydrogen (H_2) gas. – 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 water delivered as a fine spray to control fire and cool adjacent area. – **DO NOT** approach containers suspected to be hot. – Cool fire exposed containers with water spray from a protected location. – If safe to do so, remove containers from path of fire. – Equipment should be thoroughly decontaminated after use.
Fire/Explosion hazard	Combustion products include: – carbon monoxide (CO) – carbon dioxide (CO_2) – hydrogen iodide – silicon dioxide (SiO_2) – metal oxides – other pyrolysis products typical of burning organic material. Combustible. Will burn if ignited. – Solid which exhibits difficult combustion or is difficult to ignite. – Avoid generating dust, particularly clouds of dust in a confined or unventilated space as dusts may form an explosive mixture with air, and any source of ignition, i.e. flame or spark, will cause fire or explosion. – Dust clouds generated by the fine grinding of the solid are a particular hazard; accumulations of fine dust (420 micron or less) may burn rapidly and fiercely if ignited; once initiated larger particles up to 1400 microns diameter will contribute to the propagation of an explosion. – A dust explosion may release large quantities of gaseous products; this in turn creates a subsequent pressure rise of explosive force capable of damaging plant and buildings and injuring people.

	– Usually, the initial or primary explosion takes place in a confined space such as plant or machinery, and can be of sufficient force to damage or rupture the plant. If the shock wave from the primary explosion enters the surrounding area, it will disturb any settled dust layers, forming a second dust cloud, and often initiate a much larger secondary explosion. All large-scale explosions have resulted from chain reactions of this type. – Dry dust can also be charged electrostatically by turbulence, pneumatic transport, pouring, in exhaust ducts and during transport. – Build-up of electrostatic charge may be prevented by bonding and grounding. – Powder handling equipment such as dust collectors, dryers and mills may require additional protection measures such as explosion venting. – All movable parts coming in contact with this material should have a speed of less than 1-metre/sec.
HAZCHEM	2Z

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	Environmental hazard – contain spillage. – 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	Environmental hazard – contain spillage. – 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 12 for specific agent). – Collect solid residues and seal in labelled drums for disposal. – Wash area and prevent runoff into drains or waterways. – After clean up operations, decontaminate and lauder 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 incompatibilities

Suitable container	– **CARE:** Packing of high-density product in light weight metal or plastic packages may result in container collapse with product release. – Heavy gauge metal packages / Heavy gauge metal drums. – Metal can or drum. – Packaging as recommended by manufacturer. – Check all containers are clearly labelled and free from leaks.
Storage incompatibility	– Avoid strong acids, bases. – Avoid reaction with oxidising agents.

SECTION 8 Exposure controls

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	Paraffin wax	Paraffin wax (fume)	2 mg/m^3	Not Available	Not Available	Not Available
Australia Exposure Standards	Sodium selenate, anhydrous	Selenium compounds (as Se) excluding hydrogen selenide	0.1 mg/m^3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
Zinc	6 mg/m^3	21 mg/m^3	120 mg/m^3
Sodium selenate, anhydrous	1.4 mg/m^3	1.6 mg/m^3	2 mg/m^3

Ingredient	Original IDLH	Revised IDLH
Zinc	Not Available	Not Available
Paraffin wax	Not Available	Not Available
Calcium iodate	Not Available	Not Available
Cobalt (II) carbonate basic	Not Available	Not Available
Sodium selenate, anhydrous	1 mg/m^3	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
Calcium iodate	E	≤ 0.01 mg/m^3
Cobalt (II) carbonate basic	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

Appropriate engineering controls	Metal dusts must be collected at the source of generation as they are potentially explosive. – Avoid ignition sources. – Good housekeeping practices must be maintained. – Dust accumulation on the floor, ledges and beams can present a risk of ignition, flame propagation and secondary explosions. – Do not use compressed air to remove settled materials from floors, beams or equipment – Vacuum cleaners, of flame-proof design, should be used to minimise dust accumulation. – Use non-sparking handling equipment, tools and natural bristle brushes. Cover and reseal partially empty containers. Provide grounding and bonding where necessary to prevent accumulation of static charges during metal dust handling and transfer operations. – Do not allow chips, fines or dusts to contact water, particularly in enclosed areas. – Metal spraying and blasting should, where possible, be conducted in separate rooms. This minimises the risk of supplying oxygen, in the form of metal oxides, to potentially reactive finely divided metals such as aluminium, zinc, magnesium or titanium. – Workshops designed for metal spraying should possess smooth walls and a minimum of obstructions, such as ledges, on which dust accumulation is possible. – Wet scrubbers are preferable to dry dust collectors. – Bag or filter-type collectors should be sited outside the workrooms and be fitted with explosion relief doors.

	– Cyclones should be protected against entry of moisture as reactive metal dusts are capable of spontaneous combustion in humid or partially wetted states. – Local exhaust systems must be designed to provide a minimum capture velocity at the fume source, away from the worker, of 0.5 metre/sec. – Local ventilation and vacuum systems must be designed to handle explosive dusts. Dry vacuum and electrostatic precipitators must not be used, unless specifically approved for use with flammable/ explosive dusts. 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** Welding, brazing fumes (released at relatively low velocity into moderately still air) | 0.5-1.0 m/s (100-200 f/min) Within each range the appropriate value depends on: **Lower end of the range** | **Upper end of the range** 1: Room air currents minimal or favourable to capture | 1: Disturbing room air currents 2: Contaminants of low toxicity or of nuisance value only | 2: Contaminants of high toxicity 3: Intermittent, low production | 3: High production, heavy use 4: Large hood or large air mass in motion | 4: Small hood-local control only Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore, the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2.5 m/s (200-500 f/min.) for extraction of gases discharged 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.
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	– 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. – Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. – Protective gloves e.g. Leather gloves or gloves with Leather facing
Body protection	See Other protection below
Other protection	No special equipment needed when handling small quantities. **OTHERWISE**: – Overalls. – Barrier cream. – Eyewash unit.

Respiratory protection

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
Up to 10 x ES	A P1 Air-line*	- -	A PAPR-P1 -
Up to 50 x ES	Air-line**	A P2	A PAPR-P2
Up to 100 x ES	-	A P3	-
		Air-line*	-
100+ x ES	-	Air-line**	A PAPR-P2

*** -** Negative pressure demand ** - Continuous flow

A(All classes) = Organic vapours, B AUS or B1 = Acid gases, B2 = Acid gas or hydrogen cyanide (HCN), B3 = Acid gas or hydrogen cyanide (HCN), E = sulfur dioxide (SO_2), G = Agricultural chemicals, K = Ammonia (NH_3), Hg = Mercury, NO = Oxides of nitrogen, MB – Methyl bromide, AX = Low boiling point organic compounds (below 65 °C)

- Respirators may be necessary when engineering and administrative controls do not adequately prevent exposures.
- The decision to use respiratory protection should be based on professional judgment that takes into account toxicity information, exposure measurement data, and frequency and likelihood of the worker's exposure - ensure users are not subject to high thermal loads which may result in heat stress or distress due to personal protective equipment (powered, positive flow, full face apparatus may be an option).
- Published occupational exposure limits, where they exist, will assist in determining the adequacy of the selected respiratory protection. These may be government mandated or vendor recommended.
- Certified respirators will be useful for protecting workers from inhalation of particulates when properly selected and fit tested as part of a complete respiratory protection program.
- Where protection from nuisance levels of dusts are desired, use type N95 (US) or type P1 (EN143) dust masks. Use respirators and components tested and approved under appropriate government standards such as NIOSH (US) or CEN (EU)
- Use approved positive flow mask if significant quantities of dust becomes airborne.
- Try to avoid creating dust conditions.

Where significant concentrations of the material are likely to enter the breathing zone, a Class P3 respirator may be required.
Class P3 particulate filters are used for protection against highly toxic or highly irritant particulates.
Filtration rate: Filters at least 99.95% of airborne particles
Suitable for:

- Relatively small particles generated by mechanical processes e.g. grinding, cutting, sanding, drilling, sawing.
- Sub-micron thermally generated particles e.g. welding fumes, fertilizer and bushfire smoke.
- Biologically active airborne particles under specified infection control applications e.g. viruses, bacteria, COVID-19, SARS
- Highly toxic particles e.g. Organophosphate Insecticides, Radionuclides, Asbestos

Note: P3 Rating can only be achieved when used with a Full-Face Respirator or Powered Air-Purifying Respirator (PAPR). If used with any other respirator, it will only provide filtration protection up to a P2 rating.

- Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Grey cylindrical bolus with no odour; insoluble in water.

Physical state	Solid	Relative density (Water = 1)	2.6-3.3
Odour	No odour	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	>200
pH (as supplied)	Not Applicable	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Applicable
Initial boiling point and boiling range (°C)	Not Applicable	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	>100	**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 Applicable
Lower Explosive Limit (%)	Not Applicable	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Applicable	**Gas group**	Not Available
Solubility in water	Immiscible	**pH as a solution (pH)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See Section 7
Chemical stability	– Unstable in the presence of incompatible materials. – Product is considered stable. – Hazardous polymerisation will not occur.

Possibility of hazardous reactions	See Section 7
Conditions to avoid	See Section 7
Incompatible materials	See Section 7
Hazardous decomposition products	See Section 5

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual. Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in "metal fume fever". Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure. Inhalation of freshly formed zinc oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in "metal fume fever", with symptoms resembling influenza. Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure. Leucocytosis, a transient increase in white blood cell counts, is reported as a common finding in metal fume fever but is not known to be common amongst welders. Severe over-exposure to zinc oxide, following inhalation of fumes or finely divided dusts may result in bronchitis or pneumonia; a bluish skin tint may be present. Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.
Ingestion	Not normally a hazard due to physical form of product. Considered an unlikely route of entry in commercial/industrial environments. Soluble zinc salts produces irritation and corrosion of the alimentary tract (in a manner similar to copper salts) with pain, vomiting, etc. Delayed deaths have been ascribed to inanition (weakness and extreme weight loss resulting from prolonged and severe food insufficiency) following severe strictures of the oesophagus, and pylorus. Vomiting, abdominal cramps, and diarrhoea, in several cases with blood, have been observed after ingestion of zinc sulfate. Several cases of gastrointestinal disturbances have been reported after ingestion of zinc sulfate. A significant reduction in erythrocyte superoxide dismutase activity (47% decrease), haematocrit, and serum ferritin, compared to pre-treatment levels, occurred in female subjects who received supplements (as capsules) of 50 mg zinc/day as zinc gluconate for 10 weeks. A 15% decrease in erythrocyte superoxide dismutase activity was reported in male volunteers receiving 50 mg zinc/day as zinc gluconate for 6 weeks. Another study reported increases in bone specific alkaline phosphatase levels (~25%) and extracellular superoxide dismutase (~15%), while significant decreases were seen in mononuclear white cell 5'-nucleotidase (~30%) and plasma 5'-nucleotidase activity (~36%) following exposure of postmenopausal women to a combined (dietary+supplemental) 53 mg zinc/day as zinc glycine chelate. Healthy men given 200 mg zinc/day as elemental zinc for 6 weeks showed a reduction in lymphocyte stimulation response to phytohemagglutinin as well as chemotaxis and phagocytosis of bacteria by polymorphonuclear leukocytes.; however, no changes in lymphocyte cell number or in the proportion of lymphocyte populations were noted. Exposure of male volunteers to 0.48 mg zinc/kg/day, as zinc glycine chelate, had no effect on markers of coagulation relative to unexposed subjects. While the changes in haematological end points following long-term zinc exposure in humans are noteworthy, they were subclinical in nature, and therefore, are generally considered to be non-adverse. In animals, following oral administration of zinc compounds, decreased haemoglobin, haematocrit, erythrocyte, and/or leukocyte levels were observed in rats, mice, rabbits , dogs, ferrets, and pre-ruminant calves A number of intermediate duration studies have demonstrated renal effects in animals exposed to zinc oxide, zinc sulfate, and zinc acetate. Zinc sulfate caused an increase in the absolute and relative kidney weights and regressive kidney lesions (not specified) in female mice that consumed 1,110 mg zinc/kg/day in the diet for 13 weeks, but no effects occurred in rats that consumed 565 mg zinc/kg/day or in mice that consumed 104 mg zinc/kg/day under similar conditions. Severe diffuse nephrosis was observed in ferrets exposed to 195 mg zinc/kg/day as zinc oxide in the diet . In rats exposed to 191 mg zinc/kg/day as zinc acetate for 3 months, epithelial cell damage in the glomerulus and proximal convoluted tubules and increased plasma creatinine and urea levels were observed. Zinc plays a role in the normal development and maintenance of the immune system, such as in the lymphocyte response to mitogens

	and as a cofactor for the thymic hormone thymulin. Oral exposure to zinc at levels much higher than the recommended daily dose has impaired immune and inflammatory responses. This was observed in in vivo investigations of the immune competence of blood components taken from 11 healthy adult men after ingestion of 4.3 mg zinc/kg/day as zinc sulfate for 6 weeks. The mitogenic response elicited from peripheral blood lymphocytes and the chemotactic and phagocytic responses of polymorphonuclear leukocytes were impaired after zinc ingestion. No effects were seen on total numbers of lymphocytes or relative numbers of T cells, T cell subsets, or B cells. The relationship between these observations and decreased levels of immune competence that might lead to increased susceptibility to disease is unknown. A later study reported no effects of supplementation of male volunteers with 30 mg zinc/day (0.43 mg zinc/kg/day assuming a reference male body weight of 70 kg) as zinc glycine chelate for 14 weeks on levels of peripheral blood leucocytes or on the frequency of lymphocyte subsets. Zinc appears to be necessary for normal brain function, but excess zinc is toxic. A 16-year-old boy who ingested .86 mg zinc/kg/day of metallic zinc over a 2-day period in an attempt to promote wound healing, developed signs and symptoms of lethargy, light-headedness, staggering, and difficulty in writing clearly . Lethargy was also observed in a 2-year-old child who ingested a zinc chloride solution (1,000 mg zinc/kg). It is not known whether these observations represent direct effects on the nervous system. Very limited data were located regarding neurological effects in animals. Minor neuron degeneration and proliferation of oligodendroglia occurred in rats dosed with 487 mg zinc/kg/day as zinc oxide for 10 days. Rats receiving 472 mg zinc/kg/day for 10 days had increased levels of secretory material in the neurosecretory nuclei of the hypothalamus. Mice exposed postnatally to 0.5 mg zinc/kg/day as zinc acetate for 28 days showed no changes in memory formation, but showed a gradual decrease in learning extinction throughout the study.
Skin contact	The material may produce mild skin irritation; limited evidence or practical experience suggests, that the material either: – produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or – produces significant, but mild, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (non-allergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. Open cuts, abraded or irritated skin should not be exposed to this material. Entry into the bloodstream 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. Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.
Eye	Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	On the basis of epidemiological data, it has been concluded that prolonged inhalation of the material, in an occupational setting, may produce cancer in humans. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in impaired fertility on the basis of: clear evidence in animal studies of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects but which is not a secondary non-specific consequence of other toxic effects. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Implantation studies in rats show that paraffin oils may be tumourigen. As a general rule the highly refined paraffins contain a lower level of suspect polyaromatic hydrocarbons than less refined grades and also less than waxes derived from naphthenic base-stocks. Following an oral intake of extremely high doses of zinc (where 300 mg Zn/d – 20 times the US Recommended Dietary Allowance (RDA) – is a "low intake" overdose), nausea, vomiting, pain, cramps and diarrhoea may occur. There is evidence of induced copper deficiency, alterations of blood lipoprotein levels, increased levels of LDL, and decreased levels of HDL at long-term intakes of 100 mg Zn/d. The USDA RDA is 15 mg Zn/d. There is also a condition called the "zinc shakes" or "zinc chills" or metal fume fever that can be induced by the inhalation of freshly formed zinc oxide formed during the welding of galvanized materials. Supplemental zinc can prevent iron absorption, leading to iron deficiency and possible peripheral neuropathy, with loss of sensation in extremities. Zinc is necessary for normal foetal growth and development. Foetal damage may result from zinc deficiency. Only one report in the literature suggested adverse developmental effects in humans due to exposure to excessive levels of zinc. Four women were given zinc supplements of 0.6 mg zinc/kg/day as zinc sulfate during the third trimester of pregnancy. Three of the women had premature deliveries, and one delivered a stillborn infant. However, the significance of these results cannot be determined because very few details were given regarding the study protocol, reproductive histories, and the nutritional status of the women. Other human studies have found no developmental effects in the newborns of mothers consuming 0.3 mg zinc/kg/day as zinc sulfate or zinc citrate or 0.06 mg zinc/kg/day as zinc aspartate during the last two trimesters. There has been a suggestion that increased serum zinc

levels in pregnant women may be associated with an increase in neural tube defects, but others have failed to confirm this association. The developmental toxicity of zinc in experimental animals has been evaluated in a number of investigations. Exposure to high levels of zinc in the diet prior to and/or during gestation has been associated with increased foetal resorptions, reduced foetal weights, altered tissue concentrations of foetal iron and copper, and reduced growth in the offspring.
Animal studies suggest that exposure to very high levels of dietary zinc is associated with reduced foetal weight, alopecia, decreased haematocrit, and copper deficiency in offspring. For example, second generation mice exposed to zinc carbonate during gestation and lactation (260 mg/kg/day in the maternal diet), and then continued on that diet for 8 weeks, had reduced body weight, alopecia, and signs of copper deficiency (e.g., lowered haematocrit and occasional achromotrichia [loss of hair colour]. Similarly, mink kits from dams that ingested a time-weighted average dose of 20.8 mg zinc/kg/day as zinc sulfate also had alopecia and achromotrichia. It is likely that the alopecia resulted from zinc-induced copper deficiency, which is known to cause alopecia in monkeys. However, no adverse effects were observed in parental mice or mink. No effects on reproduction were reported in rats exposed to 50 mg zinc/kg/day as zinc carbonate; however, increased stillbirths were observed in rats exposed to 250 mg zinc/kg/day.
Welding or flame cutting of metals with zinc or zinc dust coatings may result in inhalation of zinc oxide fume; high concentrations of zinc oxide fume may result in "metal fume fever"; also known as "brass chills", an industrial disease of short duration. [I.L.O] Symptoms include malaise, fever, weakness, nausea and may appear quickly if operations occur in enclosed or poorly ventilated areas.
Genotoxicity studies conducted in a variety of test systems have failed to provide evidence for mutagenicity of zinc. However, there are indications of weak clastogenic effects following zinc exposure.
Metallic dusts generated by the industrial process give rise to a number of potential health problems. The larger particles, above 5 micron, are nose and throat irritants. Smaller particles however, may cause lung deterioration. Particles of less than 1.5 micron can be trapped in the lungs and, dependent on the nature of the particle, may give rise to further serious health consequences.
Metals are widely distributed in the environment and are not biodegradable. Biologically, many metals are essential to living systems and are involved in a variety of cellular, physiological, and structural functions. They often are cofactors of enzymes, and play a role in transcriptional control, muscle contraction, nerve transmission, blood clotting, and oxygen transport and delivery. Although all metals are potentially toxic at some level, some are highly toxic at relatively low levels. Moreover, in some cases the same metal can be essential at low levels and toxic at higher levels, or it may be toxic via one route of entry but not another. Toxic effects of some metals are associated with disruption of functions of essential metals. Metals may have a range of effects, including cancer, neurotoxicity, immunotoxicity, cardiotoxicity, reproductive toxicity, teratogenicity, and genotoxicity. Biological half-lives of metals vary greatly, from hours to years. Furthermore, the half-life of a given metal varies in different tissues. Lead has a half-life of 14 days in soft tissues and 20 years in bone.
In considering how to evaluate the toxicity of metals of potential concern, a number of aspects of metal toxicity should be kept in mind:
Different species vary in their responses to different metals; in some cases, humans are more sensitive than rodents. Thus, there is a need for broad-based testing of metals;

- The route of exposure may affect the dose and site where the metal concentrates, and thus the observed toxic effects;
- Metal-metal interactions can reduce or enhance toxicity; biotransformation can reduce or enhance toxicity;
- It is difficult to predict the toxicity of one metal based on the adverse effects of another; in trying to evaluate the toxicity of one particular metal compound, predictions based on similar compounds of the same metal may be valid.

Principal route of exposure is by skin contact; lesser exposures include inhalation of fumes from hot oils, oil mists or droplets. Prolonged contact with mineral oils carries with it the risk of skin conditions such as oil folliculitis, eczematous dermatitis, pigmentation of the face (melanosis) and warts on the sole of the foot (plantar warts). With highly refined mineral oils no appreciable systemic effects appear to result through skin absorption.
Exposure to oil mists frequently elicits respiratory conditions, such as asthma; the provoking agent is probably an additive. High oil mist concentrations may produce lipoid pneumonia although clinical evidence is equivocal. In animals exposed to concentrations of 100 mg/m^3 oil mist, for periods of 12 to 26 months, the activity of lung and serum alkaline phosphatase enzyme was raised; 5 mg/m^3 oil mist did not produce this response. These enzyme changes are sensitive early indicators of lung damage. Workers exposed to vapours of mineral oil and kerosene for 5 to 35 years showed an increased prevalence of slight basal lung fibrosis.
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.

24·7 Smartrace Adult Sheep Oral Boluses

TOXICITY	IRRITATION
Not Available	Not Available

Zinc

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 1130 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
Oral (Rat) LD50: >2,000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]

Paraffin wax	**TOXICITY** / **IRRITATION** Dermal (Rat) LD50: >2,000 mg/kg[1] / Eye (Rabbit): 100 mg/24 hr-mild Oral (Rat) LD50: >5,000 mg/kg[1] / Eye: no adverse effect observed (not irritating)[1] Skin (Rabbit): 500 mg/24 hr-mild Skin: no adverse effect observed (not irritating)[1]
Calcium iodate	**TOXICITY** / **IRRITATION** Oral (Dog) LD50: 200-250 mg/kg[1] / Not available
Cobalt (II) carbonate basic	**TOXICITY** / **IRRITATION** Not available / Not available
Sodium selenate, anhydrous	**TOXICITY** / **IRRITATION** Inhalation (Rat) LC50: >0.052≤0.51 mg/l4h[1] / Eye: no adverse effect observed (not irritating)[1] Oral (Rat) LD50: 1.6 mg/kg[2] / Skin: adverse effect observed (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*

Zinc	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.
Paraffin wax	"Hydrocarbon wax" describes a group of solid C20 to C36 paraffinic hydrocarbons which are not absorbed in the gastro-intestinal tract and in small quantity will pass through undigested. The widespread use in cosmetic and in cosmetic surgery over many years demonstrates the low toxicity of refined waxes and many guidelines exist for their safe use Notwithstanding this, there are occasional reports of adverse effects with these products. Subcutaneous deposits often referred to as paraffinoma, have been described frequently following injection of these materials under the skin but these are not normally associated with other progressive changes. Paraffin wax and microcrystalline were each administered orally as a solution in arachis oil to groups of 5 male and 5 female rats at dose levels of 1000 and 5000 g/kg BW. produced no clinical signs of toxicity during the seven day observation period and growth rates were normal. There were no mortalities and no macroscopic changes were observed at autopsy. Three samples of 50% paraffin in petrolatum were tested in repeated, open patch applications to 6 rabbits. Two samples produced erythema in four animals that lasted three days, and one produced erythema in one rabbit that lasted two days. A microcrystalline wax was slightly irritating, to rabbit skin, in a 24 hour occluded patch test. Four 50% solutions of paraffin in petrolatum were each instilled into the eyes of six albino rabbits with no rinse. Eyes were observed for irritation for three days. Two of the samples caused mild irritation in one rabbit on day 1; the other samples were not irritating. In a long-term feeding study with Sprague-Dawley rats, no wax-related effects were observed. In a series of 180-day feeding studies in rats that were performed over a period of approximately 15 years (beginning in 1955) on chewing-gum bases containing hydrocarbon wax in proportions varying from 2% to 57% of the gum base, no compound-related effects were observed. Long-term toxicity studies indicated that petroleum-derived paraffin and microcrystalline waxes are non-toxic and non-carcinogenic. Eight slack waxes and eight aromatic hydrocarbon extracts derived from the slack waxes were tested for carcinogenicity after applying these to the skin of mice. The slack waxes showed only a low order of carcinogenicity at 250 days. However by 450 days every sample of slack wax had elicited papillomas and for 5 of them cancers as well. The aromatic extracts on the other hand exhibited a greater potency. At 250 days all but one sample had produced papillomas and 5 samples had produced cancers. At 450 days all but one sample had elicited cancers and all had elicited papillomas. The authors concluded that the carcinogenicity of slack wax can be attributed to the aromatic compounds found in the oils from which the waxes were pressed and which are retained on the waxes as impurities, and.is not due to paraffins. Five petrolatum waxes were negative for local and systemic carcinogenicity or toxicity in skin-painting studies in mice and rabbits. However, wax disk implants, but not ground wax implants, were associated with the development of fibrosarcomas at the implantation site in rats. A description of the accumulation of long-chain alkanes (C29, C31, and C33) in a patient who had died of heart disease led the author to conclude that these hydrocarbons were of dietary (plant) origin as judged by the tissue distribution of the alkanes. The EU Scientific Committee for Food (SCF) reviewed the available information on mineral hydrocarbons, which included the petroleum waxes. Their opinion was published in 1995. The SCF reached the following conclusion: There are sufficient data to allow a full Group ADI (Average daily Intake)of 0-20 mg/kg bw for waxes conforming to the following specification: – Highly refined waxes derived from petroleum based or synthetic hydrocarbon feedstocks, with viscosity not less than 11 m^3/s (cSt) at 100 °C – Carbon number not less than 25 at the 5% boiling point – Average molecular weight not less than 500 Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length, with little absorption

above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent that iso- or cyclo-paraffins.
The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption, is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.
The materials included in the Lubricating Base Oils category are related from both process and physical-chemical perspectives;
The potential toxicity of a specific distillate base oil is inversely related to the severity or extent of processing the oil has undergone, since:

- The adverse effects of these materials are associated with undesirable components, and
- The levels of the undesirable components are inversely related to the degree of processing;
- Distillate base oils receiving the same degree or extent of processing will have similar toxicities;
- The potential toxicity of residual base oils is independent of the degree of processing the oil receives.
- The reproductive and developmental toxicity of the distillate base oils is inversely related to the degree of processing.

The degree of refining influences the carcinogenic potential of the oils. Whereas mild acid / earth refining processes are inadequate to substantially reduce the carcinogenic potential of lubricant base oils, hydrotreatment and / or solvent extraction methods can yield oils with no carcinogenic potential.
Unrefined and mildly refined distillate base oils contain the highest levels of undesirable components, have the largest variation of hydrocarbon molecules and have shown the highest potential carcinogenic and mutagenic activities. Highly and severely refined distillate base oils are produced from unrefined and mildly refined oils by removing or transforming undesirable components. In comparison to unrefined and mildly refined base oils, the highly and severely refined distillate base oils have a smaller range of hydrocarbon molecules and have demonstrated very low mammalian toxicity. Mutagenicity and carcinogenicity testing of residual oils has been negative, supporting the belief that these materials lack biologically active components or the components are largely non-bioavailable due to their molecular size.
Toxicity testing has consistently shown that lubricating base oils have low acute toxicities. Numerous tests have shown that a lubricating base oil's mutagenic and carcinogenic potential correlates with its 3-7 ring polycyclic aromatic compound (PAC) content, and the level of DMSO extractables (e.g. IP346 assay), both characteristics that are directly related to the degree/conditions of processing Skin irritating is not significant (CONCAWE) based on 14 tests on 10 CASs from the OLBO class (Other Lubricant Base Oils). Each study lasted for 24 hours, a period of time 6 times longer than the duration recommended by the OECD method).
Eye irritation is not significant according to experimental data (CONCAWE studies) based on 9 "in vivo" tests on 7 CASs from the OLBO class.

Sensitisation:

The substance does not cause the sensitization of the respiratory tract or of the skin. (CONCAWE studies based on 14 tests on 11 CASs from the OLBO class)
Germ cell mutagenicity: The tests performed within the 'in vivo" studies regarding gene mutation at mice micronuclei indicated negative results (CONCAWE studies. AMES tests had negative results in 7 studies performed on 4 CASs from the OLBO class).

Reproduction toxicity:

Reproduction / development toxicity monitoring according to OECD 421 or 422 methods. CONCAWE tests gave negative results in oral gavage studies. Pre-birth studies regarding toxicity in the unborn foetus development process showed a maternal LOAEL (Lowest Observed Adverse Effect Level) of 125 mg/kg body/day, based on dermal irritation and a NOAEL (No Observable Adverse Effect Level) of 2000 mg/kg body/day, which shows that the substance is not toxic for reproduction.
STOT (toxicity on specific target organs) – repeated exposure: Studies with short term repeated doses (28-day test) on rabbit skin indicated the NOAEL value of 1000 mg/kg. NOAEL for inhalation, local effects → 280 mg/m^3 and for systemic effects NOAEL > 980 mg/m^3.

Sub-chronic toxicity:

90-day study Dermal: NOAEL → 2000 mg/kg (CONCAWE studies).

Repeat dose toxicity:

Oral

NOAEL for heavy paraffinic distillate aromatic extract could not be identified and is less than 125 mg/kg/day when administered orally.

Inhalation

The NOAEL for lung changes associated with oil deposition in the lungs was 220 mg/m^3. As no systemic toxicity was observed, the overall NOAEL for systemic effects was > 980 mg/m^3.

Dermal

In a 90-day subchronic dermal study, the administration of Light paraffinic distillate solvent extract had an adverse effect on survivability, body weights, organ weights (particularly the liver and thymus), and variety of haematology and serum chemistry parameters in exposed animals.

Histopathological changes which were treatment-related were most prominent in the adrenals, bone marrow, kidneys, liver, lymph nodes, skin, stomach, and thymus. Based on the results of this study, the NOAEL for the test material is less than 30 mg/kg/day.

Toxicity to reproduction:
Mineral oil (a white mineral oil) caused no reproductive or developmental toxicity with 1 mL/kg/day (i.e., 1000 mg/kg/day) in an OECD 421 guideline study, but did cause mild to moderate skin irritation. Therefore, the reproductive/developmental NOAEL for this study is =1000 mg/kg/day and no LOAEL was determined.

Developmental toxicity, teratogenicity:
Heavy paraffinic distillate furfural extract produced maternal, reproductive and foetal toxicity. Maternal toxicity was exhibited as vaginal discharge (dose-related), body weight decrease, reduction in thymus weight and increase in liver weight (125 mg/kg/day and higher) and aberrant haematology and serum chemistry (125 and/or 500 mg/kg/day). Evidence of potential reproductive effects was shown by an increased number of dams with resorptions and intrauterine death. Distillate aromatic extract (DAE) was developmentally toxic regardless of exposure duration as indicated by increased resorptions and decreased foetal body weights. Furthermore, when exposures were increased to 1000 mg/kg/day and given only during gestation days 10 through 12, cleft palate and ossification delays were observed. Cleft palate was considered to indicate a potential teratogenic effect of DAE.
The following Oil Industry Note (OIN) has been applied: OIN 8 - The classifications as a reproductive toxicant category 2; H361d (Suspected of damaging the unborn child) and specific target organ toxicant category 1; H372 (Causes damage to organs through prolonged or repeated exposure) need not apply if the substance is not classified as carcinogenic.
Toxicokinetics of lubricant base oils has been examined in rodents. Absorption of other lubricant base oils across the small intestine is related to carbon chain length; hydrocarbons with smaller chain length are more readily absorbed than hydrocarbons with a longer chain length. The majority of an oral dose of mineral hydrocarbon is not absorbed and is excreted unchanged in the faeces. Distribution of mineral hydrocarbons following absorption has been observed in liver, fat, kidney, brain and spleen. Excretion of absorbed mineral hydrocarbons occurs via the faeces and urine. Based on the pharmacokinetic parameters and disposition profiles, the data indicate inherent strain differences in the total systemic exposure (~4 fold greater systemic dose in F344 vs SD rats), rate of metabolism, and hepatic and lymph node retention of C26H52, which may be associated with the different strain sensitivities to the formation of liver granulomas and MLN histiocytosis.

Highly and Severely Refined Distillate Base Oils

Acute toxicity:
Multiple studies of the acute toxicity of highly & severely refined base oils have been reported. Irrespective of the crude source or the method or extent of processing, the oral LD50s have been observed to be >5 g/kg (bw) and the dermal LD50s have ranged from >2 to >5g/kg (bw). The LC50 for inhalation toxicity ranged from 2.18 mg/l to > 4 mg/l. When tested for skin and eye irritation, the materials have been reported as "non-irritating" to "moderately irritating"
Testing in guinea pigs for sensitization has been negative

Repeat dose toxicity:
Several studies have been conducted with these oils. The weight of evidence from all available data on highly & severely refined base oils support the presumption that a distillate base oil's toxicity is inversely related to the degree of processing it receives. Adverse effects have been reported with even the most severely refined white oils - these appear to depend on animal species and/ or the peculiarities of the study.

- The granulomatous lesions induced by the oral administration of white oils are essentially foreign body responses. The lesions occur only in rats, of which the Fischer 344 strain is particularly sensitive,
- The testicular effects seen in rabbits after dermal administration of a highly to severely refined base oil were unique to a single study and may have been related to stress induced by skin irritation, and
- The accumulation of foamy macrophages in the alveolar spaces of rats exposed repeatedly via inhalation to high levels of highly to severely refined base oils is not unique to these oils, but would be seen after exposure to many water insoluble materials.

Reproductive and developmental toxicity:
A highly refined base oil was used as the vehicle control in a one-generation reproduction study.
The study was conducted according to the OECD Test Guideline 421. There was no effect on fertility and mating indices in either males or females. At necropsy, there were no consistent findings and organ weights and histopathology were considered normal by the study's authors.
A single generation study in which a white mineral oil (a food/ drug grade severely refined base oil) was used as a vehicle control is reported.
Two separate groups of pregnant rats were administered 5 ml/kg (BW)/day of the base oil via gavage, on days 6 through 19 of gestation. In one of the two base oil dose groups, three malformed foetuses were found among three litters. The study authors considered these malformations to be minor and within the normal ranges for the strain of rat.

Genotoxicity:
In vitro (mutagenicity): Several studies have reported the results of testing different base oils for mutagenicity using a modified Ames assay Base oils with no or low concentrations of 3-7 ring PACs had low mutagenicity indices.
In vivo (chromosomal aberrations): A total of seven base stocks were tested in male and female Sprague-Dawley rats using a bone marrow cytogenetics assay. The test materials were administered via gavage at dose levels ranging from 500 to 5000 mg/kg (BW). Dosing occurred for either a single day or for five consecutive days. None of the base oils produced a significant increase in aberrant cells.

Carcinogenicity: Highly & severely refined base oils are not carcinogens, when given either orally or dermally. Tumorigenic in rats.

Calcium iodate	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 dyspnoea, cough and mucus production.
Cobalt (II) carbonate basic	Bacterial mutagen. *CTM Potters Supply SDS Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies. The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested. Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens). Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis. Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure.
Sodium selenate, anhydrous	Eye effects, general anaesthesia, convulsions, muscle weakness, spasticity, cardiac EKG changes, cyanosis, lung tumours, diarrhoea, impaired liver function tests, leukaemia, specific developmental changes, effects on newborn recorded. The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.
Zinc & Calcium iodate	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 available to make classification* ✗ *- Data either not available or does not fill the criteria for classification*		

SECTION 12 Ecological information

Toxicity

24·7 Smartrace Adult Sheep Oral Boluses

Endpoint	Test Duration	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

Zinc

Endpoint	Test Duration	Species	Value	Source
EC50 (ECx)	72 hr	Algae or other aquatic plants	0.005 mg/l	4
EC50	72 hr	Algae or other aquatic plants	0.005 mg/l	4

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	LC50	96 hr	Fish	0.16 mg/ml	4
	EC50	48 hr	Crustacea	1.4 mg/ml	2
	EC50	96 hr	Algae or other aquatic plants	0.264-0.881 mg/ml	4

Paraffin wax

Endpoint	Test Duration	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

Calcium iodate

Endpoint	Test Duration	Species	Value	Source
NOEC (ECx)	168 hr	Fish	100 mg/l	2
LC50	96 hr	Fish	350 mg/l	2

Cobalt (II) carbonate basic

Endpoint	Test Duration	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

Sodium selenate, anhydrous

Endpoint	Test Duration	Species	Value	Source
EC50	48 h	Crustacea	0.52-0.63 mg/l	4
LC50	96 h	Fish	2.1-2.7 mg/l	4
NOEC (ECx)	4320 h	Fish	<0.005 mg/l	2
EC50	96 h	Algae or other aquatic plants	12 mg/l	4

Legend: *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered substances – Ecotoxicological Information – Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) – Aquatic Toxicity Data (Estimated) 4. US EPA Ecotox database – Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) Bioconcentration Data 7. METI (Japan) – Bioconcentration Data 8. Vendor Data.*

Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
Sodium selenate, anhydrous	HIGH	HIGH

Bioaccumulation potential

Ingredient	Bioaccumulation
Sodium selenate, anhydrous	LOW (LogKOW = -3.1818)

Mobility in soil

Ingredient	Mobility
Sodium selenate, anhydrous	LOW (KOC = 48.64)

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 cannot 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.
- **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 or consult manufacturer for recycling options.
- Consult State Land Waste Authority for disposal.
- Bury or incinerate residue at an approved site.
- Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information

Labels required

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Marine pollutant	
HAZCHEM	2Z

Land transport (ADG)

Each Inner is < 5 kg and therefore is not subject to the provisions of ADG, as per UN Special Provision 375.

UN number	3077
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains zinc)
Transport hazard class(es)	Class: 9 Subrisk: Not Applicable
Packing group	III
Environmental hazard	Environmentally hazardous
Special precautions for user	Special provisions: 274 331 335 375 AU01 Limited quantity: 5 kg

Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082 are not subject to this Code when transported by road or rail in;
(a) packagings;
(b) IBCs; or
(c) any other receptacle not exceeding 500 kg(L).
- Australian Special Provisions (SP AU01) - ADG Code 7th Ed.

Air transport (ICAO-IATA / DGR)

Each Inner is < 5 kg and therefore is not subject to the provisions of IMDG, as per Special Provision A197.

UN number	3077
UN proper shipping name	Environmentally hazardous substance, solid, n.o.s. * (contains zinc)
Transport hazard class(es)	ICAO/IATA Class: 9 ICAO/IATA Subrisk: Not Applicable ERG Code: 9L
Packing group	III
Environmental hazard	Environmentally hazardous
Special precautions for user	Special provisions: A97 A158 A179 A197 A215 Cargo Only Packing Instructions: 956 Cargo Only Maximum Qty / Pack: 400 kg Passenger and Cargo Packing Instructions: 956 Passenger and Cargo Maximum Qty / Pack: 400 kg Passenger and Cargo Limited Quantity Packing Instructions: Y956 Passenger and Cargo Limited Maximum Qty / Pack: 30 kg G

Sea transport (IMDG-Code / GGVSee)

Each Inner is < 5 kg and therefore is not subject to the provisions of IMDG, as per UN Special Provision 375.

UN number	3077
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains zinc)
Transport hazard class(es)	IMDG Class: 9 IMDG Subrisk: Not Applicable
Packing group	III
Environmental hazard	Marine pollutant
Special precautions for user	EMS Number: F-A , S-F Special provisions: 274 335 966 967 969 Limited quantities: 5 kg

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

Not applicable

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

Product name	Group

Zinc	Not Available
Paraffin wax	Not Available
Calcium iodate	Not Available
Cobalt (II) carbonate basic	Not Available
Sodium selenate, anhydrous	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
Zinc	Not Available
Paraffin wax	Not Available
Calcium iodate	Not Available
Cobalt (II) carbonate basic	Not Available
Sodium selenate, anhydrous	Not Available

SECTION 15 Regulatory information

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

Zinc is found on the following regulatory lists

Australian Hazardous Chemical Information System (HCIS) – Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)

Paraffin wax is found in the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

Calcium iodate is found in the following regulatory lists

Australian Hazardous Chemical Information System (HCIS) – Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)

Cobalt (II) carbonate is found in the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

Sodium selenate, anhydrous is found in the following regulatory lists

Australian Hazardous Chemical Information System (HCIS) – Hazardous Chemicals
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
Australian Inventory of Industrial Chemicals (AIIC)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (Cobalt (II) carbonate basic)
Canada - DSL	Yes
Canada - NDSL	No (Zinc; Paraffin wax; Calcium iodate; Cobalt (II) carbonate basic; Sodium selenate, hydrated)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (Zinc; Cobalt (II) carbonate basic)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (Calcium iodate)
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (Calcium iodate; Cobalt (II) carbonate basic)
Vietnam - NCI	Yes
Russia - FBEPH	No (Calcium iodate; Cobalt (II) carbonate basic; Sodium selenate, anhydrous)
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	29 Nov 2021
Initial date	29 Nov 2021

SDS Version Summary

Version	Date of Update	Sections Updated
1.0	29 Nov 2021	New SDS

Other information

Ingredients with multiple CAS numbers

Name	CAS No.
Paraffin wax	8002-74-2, 12704-91-5, 105054-93-1, 105845-08-7, 115251-23-5, 115251-24-6, 12704-92-6, 12795-75-4, 160936-34-5, 37220-23-8, 37339-80-3, 39355-22-1, 39373-78-9, 51331-35-2, 54692-42-1, 57572-43-7, 57608-84-1, 58057-11-7, 64742-43-4, 64742-51-4, 68607-08-9, 68649-50-3, 70431-26-4, 72993-88-5, 72993-89-6, 72993-90-9, 8035-62-9, 8044-02-8, 8044-79-9, 9083-41-4, 92045-74-4
Cobalt (II) carbonate basic	12602-23-2, 62647-83-0, 51839-24-8, 186361-92-2

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

Definitions and abbreviations

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

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