Apparent Dingo 500 Insecticide

AIRR Apparent Pty Ltd.

Chemwatch Hazard Alert Code: **4**

Chemwatch: **9656-86**
Version No: **2.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Initial Date: **16/11/2026**
Revision Date: **16/11/2026**
Print Date: **23/11/2026**
L.GHS.AUS.EN.E

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

Product Identifier

Product name	Apparent Dingo 500 Insecticide
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	ORGANOPHOSPHORUS PESTICIDE, LIQUID, TOXIC (contains chlorpyrifos)
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	A liquid broad spectrum insecticide. Use according to manufacturer's directions.

Details of the manufacturer or importer of the safety data sheet

Registered company name	**AIRR Apparent Pty Ltd.**
Address	15/16 Princes Street, Newport NSW 2106 Australia
Telephone	+61 3 5820 8400
Fax	Not Available
Website	www.apparent.com.au
Email	enquiries@apparentag.com.au

Emergency telephone number

Association / Organisation	**AIRR Apparent Pty Ltd.**
Emergency telephone number(s)	1800 033 111 (24 Hours)
Other emergency telephone number(s)	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

COMBUSTIBLE LIQUID, regulated for storage purposes only

Poisons Schedule	S6
Classification [1]	Flammable Liquids Category 4, Acute Toxicity (Oral) Category 3, Aspiration Hazard Category 1, Acute Toxicity (Dermal) Category 3, Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 1, Acute Toxicity (Inhalation) Category 1, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Specific Target Organ Toxicity - Repeated Exposure Category 2, 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)

Page 1 continued...

H227	Combustible liquid.
H301	Toxic if swallowed.
H304	May be fatal if swallowed and enters airways.
H311	Toxic in contact with skin.
H315	Causes skin irritation.
H318	Causes serious eye damage.
H330	Fatal if inhaled.
H336	May cause drowsiness or dizziness.
H373	May cause damage to organs through prolonged or repeated exposure.
H410	Very toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P260	Do not breathe mist/vapours/spray.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P271	Use only a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P273	Avoid release to the environment.
P284	[In case of inadequate ventilation] wear respiratory protection.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.
P331	Do NOT induce vomiting.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P330	Rinse mouth.
P370+P378	In case of fire: Use alcohol resistant foam or fine spray/water fog to extinguish.
P302+P352	IF ON SKIN: Wash with plenty of water.
P361+P364	Take off immediately all contaminated clothing and wash it before reuse.
P391	Collect spillage.
P332+P313	If skin irritation occurs: Get medical advice/attention.

Precautionary statement(s) Storage

P403+P233	Store in a well-ventilated place. Keep container tightly closed.
P405	Store locked up.

Precautionary statement(s) Disposal

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

No further product hazard information.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
2921-88-2	50	chlorpyrifos
64742-94-5	49.5	solvent naphtha petroleum, heavy aromatic
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; 4. Classification drawn from C&L; * EU IOELVs available***	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Immediately hold eyelids apart and flush the eye continuously with 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. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.

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Skin Contact	If product comes in contact with skin: ▸ Contact a Poisons Information Centre or a doctor. ▸ **DO NOT** allow clothing wet with product to remain in contact with skin, strip all contaminated clothing including boots. ▸ Quickly wash affected areas vigorously with soap and water. ▸ **DO NOT** give anything by mouth to a patient showing signs of narcosis, i.e. losing consciousness. ▸ Give atropine if instructed. ▸ **DO NOT** delay, get to a doctor or hospital quickly.
Inhalation	▸ If spray mist, vapour are inhaled, remove from contaminated area. ▸ Contact a Poisons Information Centre or a doctor at once. ▸ Lay patient down in a clean area and strip any clothing wet with spray. ▸ 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. ▸ **DO NOT** give anything by mouth to a patient showing signs of narcosis, i.e. losing consciousness. ▸ Give atropine if instructed. ▸ Get to doctor or hospital quickly.
Ingestion	▸ Avoid giving milk or oils. ▸ Avoid giving alcohol. If swallowed: ▸ Contact a Poisons Information Centre or a doctor at once. ▸ If swallowed, activated charcoal may be advised. ▸ Give atropine if instructed. ▸ **REFER FOR MEDICAL ATTENTION WITHOUT DELAY.** ▸ In the mean time, 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. ▸ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

Indication of any immediate medical attention and special treatment needed

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.
For petroleum distillates
· In case of ingestion, gastric lavage with activated charcoal can be used promptly to prevent absorption - decontamination (induced emesis or lavage) is controversial and should be considered on the merits of each individual case; of course the usual precautions of an endotracheal tube should be considered prior to lavage, to prevent aspiration.
· Individuals intoxicated by petroleum distillates should be hospitalized immediately, with acute and continuing attention to neurologic and cardiopulmonary function.
· Positive pressure ventilation may be necessary.
· Acute central nervous system signs and symptoms may result from large ingestions of aspiration-induced hypoxia.
· After the initial episode,individuals should be followed for changes in blood variables and the delayed appearance of pulmonary oedema and chemical pneumonitis. Such patients should be followed for several days or weeks for delayed effects, including bone marrow toxicity, hepatic and renal impairment Individuals with chronic pulmonary disease will be more seriously impaired, and recovery from inhalation exposure may be complicated.
· Gastrointestinal symptoms are usually minor and pathological changes of the liver and kidneys are reported to be uncommon in acute intoxications.
· Chlorinated and non-chlorinated hydrocarbons may sensitize the heart to epinephrine and other circulating catecholamines so that arrhythmias may occur.Careful consideration of this potential adverse effect should precede administration of epinephrine or other cardiac stimulants and the selection of bronchodilators.

- Most organophosphate compounds are rapidly well absorbed from the skin, conjunctiva, gastro-intestinal tract and lungs.
- They are detoxified by Cytochrome P450-mediated monoxygenases in the liver but some metabolites are more toxic than parent compounds.
- Metabolites are usually detected 12-48 hours postexposure.
- Organophosphates phosphorylate acetylcholinesterase with resultant accumulation of large amounts of acetylcholine causing initial stimulation, then exhaustion of cholinergic synapse.
- gamma-aminobutyric acid (GABA)-ergic and dopaminergic pathways provide compensatory inhibition.
- The clinical manifestation of organophosphate toxicity results from muscarinic, nicotinic and CNS symptoms.
- A garlic-like odour emanating from the patient or involved container may aid the diagnosis.
- Immediate life-threatening symptoms usually are respiratory problems.
- Frequent suction and, if necessary, endotracheal intubation and assisted ventilation may be necessary to maintain adequate oxygenation.
- Theophylline compounds, to treat bronchospasm, should be used cautiously as they may lower the seizure threshold.
- Excessive secretions and bronchospasm should respond to adequate doses of atropine.
- Diazepam is the drug of choice for convulsions.
- Usual methods of decontamination, (activated charcoal and cathartics) should be used when patients present within 4-6 hours postexposure.
- The administration of atropine, as an antidote, does not require confirmation by acetylcholinesterase levels. Severely poisoned patients develop marked resistance to the usual doses of atropine. [Atropine should not be given to a cyanosed patient - ICI] **NOTE:** Hypoxia must be corrected before atropine is given. For adult: 2 mg repeatedly SC or IV until atropinization is achieved and maintained (atropinization is characterised by decreased bronchial secretions, heart rate >100 bpm, dry mouth, diluted pupils).
- Pralidoxime (2-PAM, Protopam) is a specific antidote when given within 24 hours and perhaps up to 36-48 hours postexposure. Although it ameliorates muscle weakness, fasciculations and alterations of consciousness, it does not relieve bronchospasm or bronchorrhea and must be given concurrently with atropine. **NOTE:** Pralidoxime should be given as an adjunct to, **NOT** a replacement for atropine and should be given in every case where atropine therapy is deemed necessary. Traditional dose: 1 g (or 2 g in severe cases) by slow IV injection over 5-10 minutes. 1-2 g, 4 hourly (maximum dose 12 g in 24 hours) until clinical and analytical recovery is achieved and maintained.
- Avoid parasympathomimetic agents. Phenothiazines and antihistamines may potentiate organophosphate activity. [Ellenhorn and Barceloux: Medical Toxicology]

NOTE: Acute pancreatitis in organophosphate intoxication may be more common than reported. The possible pathogenesis of pancreatic insult are excessive cholinergic stimulation of the pancreas and ductular hypertension. Early recognition and appropriate therapy for acute pancreatitis may lead to an improved prognosis.

Cheng-Tin Hsiao, et al; Clinical Toxicology 34(3), 343-347 (1996)

BIOLOGICAL EXPOSURE INDEX - BEI

These represent the determinants observed in specimens collected from a healthy worker exposed at the Exposure Standard (ES or TLV):

Determinant	Index	Sampling Time	Comments
1. Cholinesterase activity in red cells	70% of individual's baseline	Discretionary	NS

B: Background levels occur in specimens collected from subjects **NOT** exposed

NS:Non-specific determinant; Also observed after exposure to other materials

SQ:Semi-quantitative determinant; Interpretation may be ambiguous. Should be used as a screening test or confirmatory test.

Some jurisdictions require that health surveillance be conducted on occupationally exposed workers. Such surveillance should emphasise

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- demography, occupational and medical history and health advice
- physical examination
- baseline estimation of red cell and plasma cholinesterase activity levels by the Ellman method. Estimation of red cell and plasma cholinesterase activity towards the end of the working day

SECTION 5 Firefighting measures

Extinguishing media

- Foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.
- Water spray or fog - Large fires only.

Special hazards arising from the substance or mixture

Fire Incompatibility	‣ 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 full body protective clothing with breathing apparatus. ‣ Prevent, by any means available, spillage from entering drains or water course. ‣ 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	‣ Combustible. ‣ Slight fire hazard when exposed to heat or flame. ‣ Heating may cause expansion or decomposition leading to violent rupture of containers. ‣ On combustion, may emit toxic fumes of carbon monoxide (CO). ‣ May emit acrid smoke. ‣ Mists containing combustible materials may be explosive. Combustion products include: ‣ carbon dioxide (CO_2) hydrogen chloride phosgene nitrogen oxides (NOx) phosphorus oxides (POx) sulfur oxides (SOx) ‣ other pyrolysis products typical of burning organic material. May emit poisonous fumes.
HAZCHEM	2X

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. ‣ Remove all ignition sources. ‣ Clean up all spills immediately. ‣ Avoid breathing vapours and contact with skin and eyes. ‣ Control personal contact with the substance, by using protective equipment. ‣ Contain and absorb spill with sand, earth, inert material or vermiculite. ‣ Wipe up. ‣ Place in a suitable, labelled container for waste disposal.
Major Spills	Environmental hazard - contain spillage. Chemical Class: organophosphates For release onto land: recommended sorbents listed in order of priority.

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS
LAND SPILL - SMALL				
cross-linked polymer - particulate	1	shovel	shovel	R, W, SS
cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT
wood fiber - pillow	1	throw	pitchfork	R,P, DGC, RT
foamed glass - pillow	2	shovel	shovel	R, W, P, DGC
sorbent clay - particulate	2	shovel	shovel	R, I, P
wood fibre - particulate	3	shovel	shovel	R,W, P, DGC
LAND SPILL - MEDIUM				
cross-linked polymer -particulate	1	blower	skiploader	R, W, SS
sorbent clay - particulate	2	blower	skiploader	R, I, P
polypropylene - particulate	2	blower	skiploader	R, SS, DGC

Continued...

expanded mineral - particulate	3	blower	skiploader	R,I, W, P, DGC
wood fiber- particulate	3	blower	skiploader	R, W, P, DGC
polypropylene - mat	3	throw	skiploader	DGC, RT

Legend
DGC: Not effective where ground cover is dense
R; Not reusable
I: Not incinerable
P: Effectiveness reduced when rainy
RT:Not effective where terrain is rugged
SS: Not for use within environmentally sensitive sites
W: Effectiveness reduced when windy
Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;
R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988

- **DO NOT** touch the spill material
- Clear area of personnel and move upwind.
- Alert Fire Brigade and tell them location and nature of hazard.
- Wear full body protective clothing with breathing apparatus.
- 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

- Containers, even those that have been emptied, may contain explosive vapours.
- Do NOT cut, drill, grind, weld or perform similar operations on or near containers.
- **DO NOT** allow clothing wet with material to stay in contact with skin
- Avoid skin 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 come in direct contact with human skin or eyes.
- **DO NOT** allow material to come in contact with exposed food or food contact surfaces.
- Suitable PPE must be worn at all times.
- 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

- Lined metal can, lined metal pail/ can.
- Plastic pail.
- Polyliner drum.
- Packing as recommended by manufacturer.
- Check all containers are clearly labelled and free from leaks.

For low viscosity materials

- Drums and jerricans must be of the non-removable head type.
- Where a can is to be used as an inner package, the can must have a screwed enclosure.

For materials with a viscosity of at least 2680 cSt. (23 deg. C) and solids (between 15 C deg. and 40 deg C.):

- Removable head packaging;
- Cans with friction closures and
- low pressure tubes and cartridges

may be used.
-
Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages *.
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In addition, where inner packagings are glass and contain liquids of packing group I and II there must be sufficient inert absorbent to absorb any spillage *.
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* unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.

Storage incompatibility

For alkyl aromatics:
The alkyl side chain of aromatic rings can undergo oxidation by several mechanisms. The most common and dominant one is the attack by oxidation at benzylic carbon as the intermediate formed is stabilised by resonance structure of the ring.

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- Following reaction with oxygen and under the influence of sunlight, a hydroperoxide at the alpha-position to the aromatic ring, is the primary oxidation product formed (provided a hydrogen atom is initially available at this position) - this product is often short-lived but may be stable dependent on the nature of the aromatic substitution; a secondary C-H bond is more easily attacked than a primary C-H bond whilst a tertiary C-H bond is even more susceptible to attack by oxygen
- Monoalkylbenzenes may subsequently form monocarboxylic acids; alkyl naphthalenes mainly produce the corresponding naphthalene carboxylic acids.
- Oxidation in the presence of transition metal salts not only accelerates but also selectively decomposes the hydroperoxides.
- Hock-rearrangement by the influence of strong acids converts the hydroperoxides to hemiacetals. Peresters formed from the hydroperoxides undergo Criegee rearrangement easily.
- Alkali metals accelerate the oxidation while CO2 as co-oxidant enhances the selectivity.
- Microwave conditions give improved yields of the oxidation products.
- Photo-oxidation products may occur following reaction with hydroxyl radicals and NOx - these may be components of photochemical smogs.

Oxidation of Alkylaromatics: T.S.S Rao and Shubhra Awasthi: E-Journal of Chemistry Vol 4, No. 1, pp 1-13 January 2007

- Vigorous reactions, sometimes amounting to explosions, can result from the contact between aromatic rings and strong oxidising agents.
- Aromatics can react exothermically with bases and with diazo compounds.
- A number of phosphate and thiophosphate esters are of limited thermal stability and undergo highly exothermic self-accelerating decomposition reactions which may be catalysed by impurities.
- The potential hazards can be reduced by appropriate thermal control measures.

BRETHERICK L.: Handbook of Reactive Chemical Hazards
Thermal decomposition of organophosphate esters, in the presence of trimethylolpropane or its homologues (common components of synthetic lubricants), may produce bicyclic phosphates and phosphites. These may occur be produced in as little as 5 minutes at 650 deg C. These bicyclic compounds are a class of materials with neurotoxic properties which produce convulsive seizures in test animals. The formation of these compounds does not occur, for example, in the presence of a pentaerythritol base (another common component of synthetic lubricants).

- Avoid strong acids, bases.
- Alkyl esters of thiophosphates are often temperature sensitive and decompose if overheated. Thermal decomposition products include highly toxic and odiferous hydrogen sulfide and extremely odourous alkyl mercaptans. Both species can be detected at extremely low concentrations and vapours may travel long distances (sometimes kilometers). These decomposition vapours are flammable and may ignite suddenly when mixed with air in the presence of an ignition source such as sparks or flames.
- Low temperature storage may produce crystallisation from solution.
- CARE: If heating to liquefy, use tepid water or oil, Avoid temperatures in excess of 50 deg. C. If heated, product temperature should be constantly monitored and product should be agitated to avoid localised temperatures in the container. Higher temperature will produce degradation of product and eventually cause decomposition.
- If the material is overheated, especially in the presence of water hydrogen sulfide may be released; this can cause rapid respiratory collapse, coma and death without necessarily any warning odour being sensed.
- If decomposition is suspected, or if the product has been overheated, do not open the container, discontinue heating and secure the unit. Initiate emergency procedures, taking all necessary precautions to avoid exposure to noxious or flammable gases (e.g. hydrogen sulfide), such as evacuation of contaminated areas, and use of suitable respiratory protection .
- Extreme caution must be used in tank gauging or similar operations as lethal concentration of hydrogen sulfide may accumulate in the head space of containers.

Incompatible materials and coatings include:

- butyl rubber
- butadiene rubber
- styrene rubber

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	chlorpyrifos	Chlorpyrifos	0.2 mg/m3	Not Available	Not Available	Not Available
Australia Workplace exposure limits for airborne contaminants (WEL list) - Appendix A - Workplace Exposure Limits	chlorpyrifos	Chlorpyrifos	0.007 ppm / 0.1 mg/m3	Not Available	Not Available	Workers exposed to this chemical may require specific health monitoring (see regulations 368-378, Schedule 14 to the model WHS Regulations).

MATERIAL DATA

for chlorpyrifos:
The recommended TLV-TWA is thought to reduce the potential for biologically significant decreases in plasma cholinesterase and to provide a wide margin of safety in preventing cholinergic symptoms or organic injury**.**
Odour threshold: 0.25 ppm.
The TLV-TWA is protective against ocular and upper respiratory tract irritation and is recommended for bulk handling of gasoline based on calculations of hydrocarbon content of gasoline vapour. A STEL is recommended to prevent mucous membrane and ocular irritation and prevention of acute depression of the central nervous system. Because of the wide variation in molecular weights of its components, the conversion of ppm to mg/m3 is approximate. Sweden recommends hexane type limits of 100 ppm and heptane and octane type limits of 300 ppm. Germany does not assign a value because of the widely differing compositions and resultant differences in toxic properties.
Odour Safety Factor (OSF)
OSF=0.042 (gasoline)

Exposure controls

Appropriate engineering controls

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

Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.
An approved self contained breathing apparatus (SCBA) may be required in some situations.

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Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity
3: Intermittent, low production.	3: High production, heavy use
4: Large hood or large air mass in motion	4: Small hood-local control only

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

Individual protection measures, such as personal protective equipment



Eye and face protection

- Safety glasses with unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient where complete eye protection is needed such as when handling bulk-quantities, where there is a danger of splashing, or if the material may be under pressure.
- Chemical goggles. Whenever there is a danger of the material coming in contact with the eyes; goggles must be properly fitted.[AS/NZS 1337.1, EN166 or national equivalent]
- Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection.
- Alternatively a gas mask may replace splash goggles and face shields.
- 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].

Skin protection

See Hand protection below

Hands/feet protection

- Elbow length PVC gloves

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:
· frequency and duration of contact,
· chemical resistance of glove material,
· glove thickness and
· dexterity
Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).
· When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
· When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
· Contaminated gloves should be replaced.
As defined in ASTM F-739-96 in any application, gloves are rated as:
· Excellent when breakthrough time > 480 min
· Good when breakthrough time > 20 min
· Fair when breakthrough time < 20 min
· Poor when glove material degrades
For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.
It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.
Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task.
Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:
· Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.
· Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential

Continued...

	Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Body protection	See Other protection below
Other protection	‣ Overalls. ‣ Eyewash unit. ‣ Barrier cream. ‣ Skin cleansing cream. ‣ Ensure that there is a supply of atropine tablets on hand ‣ Ensure all employees have been informed of symptoms of organophosphorus or carbamate poisoning and that the use of atropine in first aid is understood .

Respiratory protection

Full face respirator with supplied air.

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.
Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	A-AUS P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	-	A-AUS / Class 1 P2	-
up to 100 x ES	-	A-2 P2	A-PAPR-2 P2 ^

^ - Full-face
A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

- 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	Tan – yellow coloured liquid.

Physical state	Liquid	**Relative density (Water = 1)**	Not Available
Odour	Characteristic	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (mm2/s)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	>62	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Combustible.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Partly miscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available
Heat of Combustion (kJ/g)	Not Available	**Ignition Distance (cm)**	Not Available
Flame Height (cm)	Not Available	**Flame Duration (s)**	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	**Enclosed Space Ignition Deflagration Density (g/m3)**	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

Continued...

Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

a) Acute Toxicity	There is sufficient evidence to classify this material as acutely toxic.
b) Skin Irritation/Corrosion	There is sufficient evidence to classify this material as skin corrosive or irritating.
c) Serious Eye Damage/Irritation	There is sufficient evidence to classify this material as eye damaging or irritating
d) Respiratory or Skin sensitisation	Based on available data, the classification criteria are not met.
e) Mutagenicity	Based on available data, the classification criteria are not met.
f) Carcinogenicity	Based on available data, the classification criteria are not met.
g) Reproductivity	Based on available data, the classification criteria are not met.
h) STOT - Single Exposure	There is sufficient evidence to classify this material as toxic to specific organs through single exposure
i) STOT - Repeated Exposure	There is sufficient evidence to classify this material as toxic to specific organs through repeated exposure
j) Aspiration Hazard	There is sufficient evidence to classify this material as an aspiration hazard

Inhaled	Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may produce severely toxic effects; these may be fatal. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation. Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial 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. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Symptoms of acute exposure to cholinesterase-inhibiting compounds may include the following: numbness, tingling sensations, incoordination, headache, dizziness, tremor, nausea, abdominal cramps, sweating, blurred vision, difficulty breathing or respiratory depression, slow heartbeat. Very high doses may result in unconsciousness, incontinence, and convulsions or fatality. Some cholinesterase-inhibitors may cause delayed symptoms beginning 1 to 4 weeks after an acute exposure that may or may not have produced immediate symptoms. In such cases, numbness, tingling, weakness, and cramping may appear in the lower limbs and progress to incoordination and paralysis. Improvement may occur over months or years, but some residual impairment may remain The early warnings of poisonings associated with cholinesterase inhibition include nasal hyperaemia (localised engorgement with blood), watery discharge, chest discomfort, dyspnoea and wheezing due to increased bronchial secretions and bronchioconstriction. Other effects may include tearing, urination, chest pains, breathing difficulties, low blood pressure, irregular heartbeat, loss of reflexes, twitching, visual disturbances, dilated or pin-point pupils, convulsion, lung congestion, coma and heart-include ataxia, slurred speech, tremors of the tongue and eyelids, and eventual paralysis of the extremities and respiratory muscles. Fatalities in man are generally due to respiratory failure on the basis of central nervous system paralysis although cardiac arrest may also occur. Where cholinesterase inhibitors have been used as miotic eyedrops there has occasional evidence of toxic effects on the crystalline lens and obstruction of the nasolachrymal canals. High inhaled concentrations of mixed hydrocarbons may produce narcosis characterised by nausea, vomiting and lightheadedness. Inhalation of aerosols may produce severe pulmonary oedema, pneumonitis and pulmonary haemorrhage. Inhalation of petroleum hydrocarbons consisting substantially of low molecular weight species (typically C2-C12) may produce irritation of mucous membranes, incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and anaesthetic stupor. Massive exposures may produce central nervous system depression with sudden collapse and deep coma; fatalities have been recorded. Irritation of the brain and/or apnoeic anoxia may produce convulsions. Although recovery following overexposure is generally complete, cerebral micro-haemorrhage of focal post-inflammatory scarring may produce epileptiform seizures some months after the exposure. Pulmonary episodes may include chemical pneumonitis with oedema and haemorrhage. The lighter hydrocarbons may produce kidney and neurotoxic effects. Pulmonary irritancy increases with carbon chain length for paraffins and olefins. Alkenes produce pulmonary oedema at high concentrations. Liquid paraffins may produce anaesthesia and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce polyneuropathy. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue and vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons are cardiac sensitisers and may cause ventricular fibrillations. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal. Inhalation of aerosols containing chlorpyrifos has been shown to depress cholinesterase activity. The acute toxicity of inhaled alkylbenzene is best described by central nervous system depression. These compounds may also act as general anaesthetics. Whole body symptoms of poisoning include light-headedness, nervousness, apprehension, a feeling of well-being, confusion, dizziness, drowsiness, ringing in the ears, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness, depression of breathing, and arrest. Heart stoppage may result from cardiovascular collapse. A slow heart rate and low blood pressure may also occur. Alkylbenzenes are not generally toxic except at high levels of exposure. Their breakdown products have low toxicity and are easily eliminated from the body. Inhalation of dusts, generated by the material during the course of normal handling, may produce severe damage to the health of the individual. Relatively small amounts absorbed from the lungs may prove fatal. Inhalation of dusts, generated by the material, during the course of normal handling, may produce severely toxic effects; these may be fatal.
Ingestion	Toxic effects may result from the accidental ingestion of the material; animal experiments indicate that ingestion of less than 40 gram may be fatal or may produce serious damage to the health of the individual. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by swallowing. Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result.

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	Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis). Ingestion may produce nausea, vomiting, anorexia, abdominal cramps, and diarrhoea. Generalised symptoms produced by cholinesterase inhibitors may ensue following appreciable absorption. Some organophosphates may cause delayed symptoms beginning 1 to 4 weeks after an acute exposure which may or may not have produced immediate symptoms. In such cases, numbness, tingling, weakness, and cramping may appear in the lower limbs and progress to incoordination and paralysis. Improvement may occur over months or years, and in some cases residual impairment will remain Thiophosphates (phosphothioate esters) do not generally produce the same degree of cholinesterase inhibition associated with other organophosphates. They may however react with a range of compounds to produce such inhibitors. Ingestion of large quantities may produce severe abdominal pains, thirst, acidaemia, difficult breathing, convulsions, collapse, shock and even death. Organophosphates may suppress the immune system in some animal species. Symptoms of chlorpyrifos poisoning are cholinesterase inhibition, headache, fatigue, dizziness, blurred vision, weakness, nausea, cramps, diarrhea, chest discomfort, sweating, miosis, tearing, salivation, vomiting, cyanosis and muscle twitching. In advanced cases convulsions, coma, loss of reflexes, and loss of sphincter control may occur. [OHMTADS]. Workers exposed to a 0.5% chlorpyrifos emulsion in field trials for malaria control showed a measured decrease in plasma and red blood cell cholinesterase levels (5 of 7 workers showed more than a 50% decrease within two weeks of the start of the program). Spray workers ingesting 0.03 mg chlorpyrifos/kg body weight/day for 3 weeks did not show a statistically significant plasma cholinesterase depression. Workers exposed to a 0.5% chlorpyrifos emulsion in field trials for malaria control showed a measured decrease in plasma and red blood cell cholinesterase levels (5 of 7 workers showed more than a 50% decrease within two weeks of the start of the program). Chlorpyrifos-oxon, an active metabolite of chlorpyrifos, is 10 to 20 times more active than the parent compound against acetylcholinesterase in producing delayed polyneuropathy. In humans the enzyme that deactivates oxon-metabolites (paraoxonase) is variable both in form and in amount. Rabbit paraoxonase hydrolyses chlorpyrifos oxon at a high rate making rabbits chlorpyrifos resistant Ingestion of petroleum hydrocarbons may produce irritation of the pharynx, oesophagus, stomach and small intestine with oedema and mucosal ulceration resulting; symptoms include a burning sensation in the mouth and throat. Large amounts may produce narcosis with nausea and vomiting, weakness or dizziness, slow and shallow respiration, swelling of the abdomen, unconsciousness and convulsions. Myocardial injury may produce arrhythmias, ventricular fibrillation and electrocardiographic changes. Central nervous system depression may also occur. Light aromatic hydrocarbons produce a warm, sharp, tingling sensation on contact with taste buds and may anaesthetise the tongue. Aspiration into the lungs may produce coughing, gagging and a chemical pneumonitis with pulmonary oedema and haemorrhage.
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant 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 (nonallergic). 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. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact. The material may accentuate any pre-existing dermatitis condition Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. Localised sweating and fasciculation (small localised muscular contractions visible through the skin) may occur at sites of contact. Absorption may produce cholinesterase inhibition effects following delays of up to 2-3 hours (but generally not more than 12 hours). Toxic effects of chlorpyrifos are dose related; as four repeated doses of 25 mg/kg (i.e. 1.75 gram/70 kg adult) each applied to the skin of human volunteers for 12 hours caused depression of plasma cholinesterase; four repeated doses of 10 mg/kg (i.e. 0.7 gram/ 70 kg adult) each did not show this effect Open cuts, abraded or irritated skin should not be exposed to this material The liquid may be miscible with fats or oils and may degrease the skin, potentially producing a non-allergic contact dermatitis. The material is not classified as a skin irritant under Regulation (EC) No 1272/2008 (CLP), as amended. 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. Skin contact with the material may produce toxic effects; systemic effects may result following absorption.
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation. Direct contact with the eyes may produce lachrymation (tears), twitching of the eyelids, miosis (contraction of the pupils) and ciliary muscle spasm mydriasis (dilation of the pupils). Absorption may produce generalised cholinesterase inhibition. Petroleum hydrocarbons may produce pain after direct contact with the eyes. Slight, but transient disturbances of the corneal epithelium may also result. The aromatic fraction may produce irritation and lachrymation.
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests. Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, on the basis that similar materials tested in appropriate animal studies provide some suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects. Repeated or prolonged exposures to cholinesterase inhibitors produce symptoms similar to acute effects. In addition workers exposed repeatedly to these substances may exhibit impaired memory and loss of concentration, severe depression and acute psychosis, irritability, confusion, apathy, emotional lability, speech difficulties, headache, spatial disorientation, delayed reaction times, sleepwalking, drowsiness or insomnia. An influenza-like condition with nausea, weakness, anorexia and malaise has been described. There is a growing body of evidence from epidemiological studies and from experimental laboratory studies that short-term exposure to some cholinesterase-inhibiting insecticides may produce behavioural or neuro-chemical changes lasting for days or months, presumably outlasting the cholinesterase inhibition. Although the number of adverse effects following humans poisonings subsides, there are still effects in some workers months after cholinesterase activity returns to normal. These long-lasting effects include blurred vision, headache, weakness, and anorexia. The neurochemistry of animals exposed to chlorpyrifos or fenthion is reported to be altered permanently after a single exposure. These effects may be more severe in developing animals where both acetyl- and butyrylcholinesterase may play an integral part in the development of the nervous system. ***Padilla S., The Neurotoxicity of Cholinesterase-Inhibiting Insecticides: Past and Present Evidence Demonstrating Persistent Effects. Inhalation Toxicology 7:903-907, 1995***

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Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/or memory loss, tremor in the fingers and tongue, vertigo, olfactory disorders, constriction of visual field, paraesthesias of the extremities, weight loss and anaemia and degenerative changes in the liver and kidney. Chronic exposure by petroleum workers, to the lighter hydrocarbons, has been associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness and paraesthesias), psychological and neurophysiological deficits, bone marrow toxicities (including hypoplasia possibly due to benzene) and hepatic and renal involvement. Chronic dermal exposure to petroleum hydrocarbons may result in defatting which produces localised dermatoses. Surface cracking and erosion may also increase susceptibility to infection by microorganisms. One epidemiological study of petroleum refinery workers has reported elevations in standard mortality ratios for skin cancer along with a dose-response relationship indicating an association between routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have been unable to confirm this finding.
Hydrocarbon solvents are liquid hydrocarbon fractions derived from petroleum processing streams, containing only carbon and hydrogen atoms, with carbon numbers ranging from approximately C5-C20 and boiling between approximately 35-370 deg C. Many of the hydrocarbon solvents have complex and variable compositions with constituents of 4 types, alkanes (normal paraffins, isoparaffins, and cycloparaffins) and aromatics (primarily alkylated one- and two-ring species). Despite the compositional complexity, most hydrocarbon solvent constituents have similar toxicological properties, and the overall toxicological hazards can be characterized in generic terms. Hydrocarbon solvents can cause chemical pneumonitis if aspirated into the lung, and those that are volatile can cause acute CNS effects and/or ocular and respiratory irritation at exposure levels exceeding occupational recommendations. Otherwise, there are few toxicologically important effects. The exceptions, n-hexane and naphthalene, have unique toxicological properties

Animal studies:
No deaths or treatment related signs of toxicity were observed in rats exposed to light alkylate naphtha (paraffinic hydrocarbons) at concentrations of 668, 2220 and 6646 ppm for 6 hrs/day, 5 days/wk for 13 weeks. Increased liver weights and kidney toxicity (male rats) was observed in high dose animals. Exposure to pregnant rats at concentrations of 137, 3425 and 6850 ppm did not adversely affect reproduction or cause maternal or foetal toxicity. Lifetime skin painting studies in mice with similar naphthas have shown weak or no carcinogenic activity following prolonged and repeated exposure. Similar
naphthas/distillates, when tested at nonirritating dose levels, did not show any significant carcinogenic activity indicating that this tumorigenic response is likely related to chronic irritation and not to dose. The mutagenic potential of naphthas has been reported to be largely negative in a variety of mutagenicity tests. The exact relationship between these results and human health is not known. Some components of this product have been shown to produce a species specific, sex hormonal dependent kidney lesion in male rats from repeated oral or inhalation exposure. Subsequent research has shown that the kidney damage develops via the formation of a alpha-2u-globulin, a mechanism unique to the male rat. Humans do not form alpha-2u-globulin, therefore, the kidney effects resulting from this mechanism are not relevant in human.
Alkyl thiophosphates may degrade, under certain circumstances, to produce hydrogen sulfide and alkyl mercaptans.
Prolonged skin contact may produce irritation and slight burns. Published literature and unpublished documents by the U.S. Environmental Protection Agency contain reports of congenital defects of test animals and in children exposed to Dursban (chloropyrifos). Birth defects of an extensive and unusual patterns were noted (1) in four children exposed, in utero, to Dursban. These included defects of the brain, eyes, ears, palate, teeth, heart, feet, nipples and genitalia. Brain defects were present in the ventricles, corpus callosum, choroid plexus, and septum pellucidum. Genital defects of the testes (undescended), micro phallus and labia (fused) were noted. All children had growth retardation, and three had hypotonia and profound mental retardation.
(1): Janette D. Sherman: Archives of Environmental Health, 51, 1, pp 5-8, 1996
Children are reported (2) to have developed narrowing of the peripheral visual fields, myopia, decreased acuity and retinal damage following in utero exposure to chlorpyrifos. The basis for these eye defects may be interference with the development of acetylcholine, a retinal neurotransmitter beginning in the pre-natal period. 3,5,6-trichloropyridinol (TCP) which is both a feed-stock of the product and a metabolic breakdown product of chlorpyrifos has been found to cause central nervous system anomalies (hydrocephaly and dilated brain ventricles) and multiple anomalies in the animal foetus, including cleft palate and skull and vertebral abnormalities. It is suggested that TCP contributes to the teratogenic effect of TCP. The reported half-life for TCP ranges between 27 hours and 66.5 to 127-9 hours.
(2): Janette D Sherman: International Journal of Occupational Medicine and Toxicology, 4, 4, pp 417-431

Apparent Dingo 500 Insecticide

TOXICITY	IRRITATION
Not Available	Not Available

chlorpyrifos

TOXICITY	IRRITATION
dermal (rat) LD50: 202 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
Inhalation (Rat) LC50: >0.2 mg/L4h[2]	Skin: no adverse effect observed (not irritating)[1]
Oral (Mouse) LD50; >69 mg/kg[1]	

solvent naphtha petroleum, heavy aromatic

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >2000 mg/kg[2]	Eye (Rodent - rabbit): 100uL/24H - Moderate
Inhalation (Rat) LC50: >0.003 mg/L4h[1]	Eye: adverse effect observed (irritating)[1]
Oral (Rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Skin (Rodent - rabbit): 500uL/24H - Mild
	Skin (Rodent - rabbit): 500uL/24H - Moderate
	Skin: adverse effect observed (irritating)[1]
	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***

Apparent Dingo 500 Insecticide

Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic 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 persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. 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. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.
Data demonstrate that during inhalation exposure,aromatic hydrocarbons undergo substantial partitioning into adipose tissues. Following cessation of exposure, the level of aromatic hydrocarbons in body fats rapidly declines. Thus, the aromatic hydrocarbons are unlikely to

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	bioaccumulate in the body. Selective partitioning of the aromatic hydrocarbons into the non-adipose tissues is unlikely. No data is available regarding distribution following dermal absorption. However, distribution following this route of exposure is likely to resemble the pattern occurring with inhalation exposure. Aromatics hydrocarbons may undergo several different Phase I dealkylation, hydroxylation and oxidation reactions which may or may not be followed by Phase II conjugation to glycine, sulfation or glucuronidation. However, the major predominant biotransformation pathway is typical of that of the alkylbenzenes and consists of: (1) oxidation of one of the alkyl groups to an alcohol moiety; (2) oxidation of the hydroxyl group to a carboxylic acid; (3) the carboxylic acid is then conjugated with glycine to form a hippuric acid. The minor metabolites can be expected to consist of a complex mixture of isomeric triphenols, the sulfate and glucuronide conjugates of dimethylbenzyl alcohols, dimethylbenzoic acids and dimethylhippuric acids. Consistent with the low propensity for bioaccumulation of aromatic hydrocarbons, these substances are likely to be significant inducers of their own metabolism. The predominant route of excretion of aromatic hydrocarbons following inhalation exposure involves either exhalation of the unmetabolized parent compound, or urinary excretion of its metabolites. When oral administration occurs, there is little exhalation of unmetabolized these hydrocarbons, presumably due to the first pass effect in the liver. Under these circumstances, urinary excretion of metabolites is the dominant route of excretion.
CHLORPYRIFOS	Oral (rat) LD50: 135-163 mg/kg * Oral (rabbit) LD50: 1000-2000 mg/kg * Inhalation (rat) LC50: >0.2 mg/l(14 ppb)/ 4-6hr * NOEL (2 y) based on blood plasma cholinesterase activity, for rats 0.03, dogs 0.01 mg/kg daily * ADI 0.01 mg/kg b.w. * Toxicity Class WHO II;EPA II * Non-teratogenic * ***[* The Pesticides Manual, Incorporating The Agrochemicals Handbook, 10th Edition, Editor Clive Tomlin, 1994, British Crop Protection Council]***
Apparent Dingo 500 Insecticide & SOLVENT NAPHTHA PETROLEUM, HEAVY AROMATIC	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. For petroleum: This product contains benzene, which can cause acute myeloid leukaemia, and n-hexane, which can be metabolized to compounds which are toxic to the nervous system. This product contains toluene, and animal studies suggest high concentrations of toluene lead to hearing loss. This product contains ethyl benzene and naphthalene, from which animal testing shows evidence of tumour formation. Cancer-causing potential: Animal testing shows inhaling petroleum causes tumours of the liver and kidney; these are however not considered to be relevant in humans. Mutation-causing potential: Most studies involving gasoline have returned negative results regarding the potential to cause mutations, including all recent studies in living human subjects (such as in petrol service station attendants). Reproductive toxicity: Animal studies show that high concentrations of toluene (>0.1%) can cause developmental effects such as lower birth weight and developmental toxicity to the nervous system of the foetus. Other studies show no adverse effects on the foetus. Human effects: Prolonged or repeated contact may cause defatting of the skin which can lead to skin inflammation and may make the skin more susceptible to irritation and penetration by other materials. Animal testing shows that exposure to gasoline over a lifetime can cause kidney cancer, but the relevance in humans is questionable.
Apparent Dingo 500 Insecticide & CHLORPYRIFOS	for chlorpyrifos: prenatal exposure to the pesticide chlorpyrifos (CPF) is associated with an increased risk of neurodevelopmental problems in children, including developmental delays, attention problems, and a lower IQ. Studies have shown associations between prenatal CPF exposure and alterations in brain structure, function, and metabolism, which can lead to poorer motor skills and attention deficits later in childhood. The majority of the neurological symptoms associated with chlorpyrifos exposure result from its inhibition of acetylcholinesterase and the subsequent cholinergic over-stimulation. Common symptoms related to excessive cholinergic activity include headache, diaphoresis, nausea, vomiting, diarrhea, epigastric cramping, bradycardia, blurred vision, miosis, bronchoconstriction and excess mucous secretions, pulmonary oedema, dyspnoea, muscle fasciculations, salivation, lacrimation, and urination. In adults and children, acute-duration inhalation exposure to unspecified concentrations of chlorpyrifos is associated with paraesthesia and lightheadedness. Headache is also a common occurrence. Additionally, acute duration chlorpyrifos exposure may produce signs of neurological toxicity weeks or months after the initial symptoms have resolved. For example, a family which became ill after an unspecified concentration of chlorpyrifos was applied in their home initially presented with headaches, nausea, and muscle cramps. However, numbness, paraesthesia (most prominent in the legs), and memory impairment were reported by the family 1 month later. The children also showed a decline in scholastic performance that lasted for approximately 6 months. Neurological exams conducted 6 months post-exposure revealed mild short term memory loss on all routine mental status testing of recall of multiple objects. Nerve conduction studies revealed low amplitude sural nerve action potentials in all family members. Motor and upper-extremity sensory nerve action potentials were normal. Sural nerve amplitudes in all but one family member had returned to normal 6 months later. Although inhalation was the most likely route of exposure, the family could also have been exposed dermally. In a chronic duration exposure study, the prevalence of selected illnesses and symptoms in 175 employees involved in the production of chlorpyrifos and 335 matched controls with no history of exposure to organophosphorus chemicals were compared. Employees were further subdivided for dose response testing into three exposure intensity groups on the basis of job title and air-monitoring data. The prevalence of respiratory illness or other respiratory symptoms were compared. There were no statistically significant differences in the number of subjects with respiratory illness or other respiratory symptoms between the exposed and control groups. Exposure was assumed to be via inhalation and dermal routes. Unstable blood pressure and pulse were noted in a 33-year-old male acutely exposed to an unspecified concentration of Dursban that was accidentally sprayed into the ventilating system at his place of work. The symptoms began approximately 6 weeks after exposure and slowly resolved over 8-10 weeks. Exposure was assumed to be via inhalation and dermal routes. While cardiovascular effects are possible after acute-duration exposure to chlorpyrifos because of cholinergic over-stimulation, the 6-week interval between a single exposure and onset of symptoms raises doubts as to whether chlorpyrifos was the causative agent in this case.. Gastrointestinal effects following acute-duration exposure to chlorpyrifos have been observed in humans. A family became ill and complained of feeling nauseated after their house was sprayed with Dursban. The time from exposure to the onset of symptoms and exposure-level data were not reported. Exposure was assumed to be via inhalation and dermal routes. Intermediate-duration exposure to chlorpyrifos may be associated with diarrhea in humans. Diarrhoea developed in a 40-year-old male exterminator who was repeatedly exposed to an unknown concentration of Dursban in a closed environment over a 6-month period. Exposure was assumed to be via inhalation and dermal routes. Erythrocyte cholinesterase levels determined at the onset of symptoms were initially low (value not given). The diarrhea probably resulted from stimulation of the parasympathetic nervous system as a consequence of cholinesterase inhibition. Stimulation of the parasympathetic nervous system increases gastrointestinal motility, thereby decreasing food transit times. The net result is that there is less time for water to be absorbed by the gastrointestinal tract and diarrhoea results. For dithiophosphate alkyl esters and their (zinc) salts: **Acute toxicity:** Dithiophosphate alkyl esters consist of a phosphorodithioic acid structure with alkyl ester substituent groups. The alkyl groups are saturated hydrocarbon chains that vary in length and extent of branching. While corrosive to tissue the esters demonstrate a low concern for acute systemic toxicity. Data on acute mammalian toxicity of zinc dialkyldithiophosphates in highly refined lubricant base oil also indicate a low concern for acute toxicity. Commercial oil-based samples of the zinc dialkyldithiophosphate category have been tested for acute oral toxicity. The acute oral LD50 for these studies in rats ranged from 2000-3500 mg/kg. Clinical signs observed following treatment

Continued...

included diarrhea, lethargy, reduced food consumption, and staining about the nose and eye. Ptosis, piloerection, ataxia and salivation were occasionally observed. The incidence and severity of these symptoms were proportional to the dose. In many cases the effects were found to be reversible during observation week 2. Necropsy findings were few in number. Lung congestion, gastrointestinal irritation and a reduction in body fat were observed in some animals.
Acute dermal toxicity and irritation studies using the ester on experimental animals resulted in severe dermal irritation and corrosivity. There is minimal opportunity of human exposure to the chemicals in this category. Dithiophosphate alkyl esters exhibit extreme corrosive properties on skin.
Commercial oil-based samples of the zinc dialkyldithiophosphate category have been tested for acute dermal toxicity. The acute dermal LD50s for these studies in rabbits were greater than 2000 mg/kg (limit tests). No treatment-related mortality was observed at doses ranging from 2000-8000 mg/kg. Dermal application of the test materials to abraded skin for 24 hours typically produced moderate-to-severe erythema and edema, which in some cases persisted through the 14-day observation period. Clinical signs included varying degrees of reduced food consumption, weight loss, diarrhea, lethargy, ataxia, ptosis, motor incoordination and/or loss of righting reflex. There were no remarkable gross necropsy observations. Overall, the acute dermal LD50 for these substances were greater than 2000 mg/kg indicative of a relatively low order of lethal toxicity. Zinc dialkyldithiophosphates are high molecular weight components (average > 500 gm/mol), which generally accepted that the molecular weight limit for passive transport across biological membranes. Thus, upon exposure it is unlikely that significant amounts of these components will be absorbed for systemic distribution. In addition, these materials have a low water solubility that further inhibits absorption and distribution in the mammalian system.
The negligible vapor pressure and high viscosity at ambient temperature indicates that these materials are unlikely to represent an inhalation exposure under conditions of use
Repeat dose toxicity: Data from several repeated-dose toxicity studies using commercial samples of zinc dialkyldithiophosphates in highly refined lubricant base oil has been reviewed. Repeated dermal exposure to experimental animals resulted in moderate-to-severe dermal irritation, behavioral distress, body weight loss and emaciation, reduction in hematological parameters and adverse effects on male reproductive organs. These effects were observed across several members of the category with carbon chain lengths ranging from C4-8. There was no evidence that the incremental increase in carbon chain length or molecular weight could be correlated with significant changes in toxicity parameters.
Oral administration caused significant gastric irritation and related gastrointestinal disturbances, signs of distress but with no evidence of adverse effects on male reproductive organs.
Reproductive toxicity: An epidemiological study on workers exposed to oil-based zinc dialkyldithiophosphates (range C4-8) in an additive manufacturing plant revealed no adverse effects on worker reproductive health. Review of the available information underscores the similarity of clinical and pathological findings in repeated-dose dermal toxicity studies with C4-10 zinc dialkyldithiophosphates, as well as the absence of reproduction and developmental toxicity and the lack of untoward findings in a human epidemiological investigation.
Reproductive organ effects, following dermal application, have been observed in male rabbits; these are attributed to the stress associated with the severe dermal responses to the test material, rather than direct a systemic response to the test materials. Changes in male reproductive organs in the rabbit have been observed when other irritating substances are applied to the skin at dose levels that cause skin lesions. Thus, dermal irritation alone, or in combination with the accompanying weight loss and stress, is thought to play a role in the reproductive organ response to repeated cutaneous application of zinc dialkyldithiophosphates.
Mutagenicity: Findings indicate that commercial samples of zinc dialkyldithiophosphates in highly refined lubricant base oil have a small potential for inducing genetic toxicity. In vitro bacterial gene mutation assays, in vitro mammalian gene mutation assays, or in vivo chromosomal aberration assays have been conducted. Frequencies of reverse mutations in bacteria were not significantly changed after exposure to the zinc dialkyldithiophosphates. In vitro mutation studies in mammalian cells indicate that the zinc dialkyldithiophosphates do not consistently display mutagenic activity in the absence of metabolic activation, however, upon biotransformation, these materials showed mutagenic activity. The findings in bacterial and mammalian cells did not vary in proportion to the alkyl chain length or any other physicochemical parameter.
The results of the studies performed in the absence of hepatic microsome activation were inconsistent, but in general indicating that zinc dialkyldithiophosphates have mutagenic potential (3 studies negative, 3 studies positive in the absence of metabolic activation). However, the weight of evidence (2 studies positive, 1 study negative) indicates that metabolic activation of zinc dialkyldithiophosphates by induced hepatic microsomal enzymes results in a significant increase in the mutagenic potential of this class of chemical substances.
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Thiophosphates (or phosphorothioates, PS) are chemical compounds and anions with the general chemical formula PS
4-xO3-
x (x = 0, 1, 2, or 3) and related derivatives where organic groups are attached to one or more O or S. Thiophosphates feature tetrahedral phosphorus(V) centers.]
Organothiophosphates are a subclass of organophosphorus compounds that are structurally related to the inorganic thiophosphates. Common members have formulas of the type (RO)3-xRxPS and related compounds where RO is replaced by RS. Many of these compounds are used as insecticides, some have medical applications, and some have been used as oil additives.
number of phosphorothioates have been studied extensively for their safety profiles in a several species such as mice, rats, monkeys, and humans. The dose-dependent side effects in experimental rats and mice included thrombocytopenia, splenomegaly, and elevation of transaminases]. Histopathology changes included mononuclear cell infiltration in tissues such as liver, kidney, and spleen, and reticuloendothelial cell and lymphoid cell hyperplasia. The severity of side effects is dependent on the dose, frequency, and duration of the administration of oligonucleotides. In general, the toxicity profiles of phosphorothioate oligonucleotides are similar with various lengths and base compositions, with exceptions in the presence of certain sequence motifs such as CpG-dinucleotides] and poly-G , which contribute to the severity of toxicity
hosphates (P = O) are biologically active, whereas phosphorothioates (P = S) need bioactivation to the corresponding metabolite (oxon) before becoming so.

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 either not available or does not fill the criteria for classification***
✔ ***– Data available to make classification***

SECTION 12 Ecological information

Toxicity

Apparent Dingo 500 Insecticide

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

chlorpyrifos

Endpoint	Test Duration (hr)	Species	Value	Source

Continued...

	EC50	72h	Algae or other aquatic plants	0.58mg/L	2
	BCF	1344h	Fish	49-493	7
	EC50	48h	Crustacea	<0.001mg/L	5
	EC50	96h	Algae or other aquatic plants	0.112-0.169mg/L	4
	NOEC(ECx)	672h	Fish	<0.001mg/L	2
	LC50	96h	Fish	<0.001mg/L	4

solvent naphtha petroleum, heavy aromatic	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	<1mg/l	1
	EC50	48h	Crustacea	0.95mg/l	1
	EC50	96h	Algae or other aquatic plants	11.7mg/l	2
	EC50(ECx)	48h	Crustacea	0.95mg/l	1
	LC50	96h	Fish	0.58mg/l	2

Legend:	***Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. US EPA, Ecotox database - Aquatic Toxicity Data 4. ECETOC Aquatic Hazard Assessment Data 5. NITE (Japan) - Bioconcentration Data 6. METI (Japan) - Bioconcentration Data 7. Vendor Data 8. User defined data***

Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
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.
Toxic to flora.
Toxic to soil organisms.
Toxic to bees.
When spilled this product may act as a typical oil, causing a film, sheen, emulsion or sludge at or beneath the surface of the body of water. The oil film on water surface may physically affect the aquatic organisms, due to the interruption of the
oxygen transfer between the air and the water
Oils of any kind can cause:

- drowning of water-fowl due to lack of buoyancy, loss of insulating capacity of feathers, starvation and vulnerability to predators due to lack of mobility
- lethal effects on fish by coating gill surfaces, preventing respiration
- asphyxiation of benthic life forms when floating masses become engaged with surface debris and settle on the bottom and
- adverse aesthetic effects of fouled shoreline and beaches

In case of accidental releases on the soil, a fine film is formed on the soil, which prevents the plant respiration process and the soil particle saturation. It may cause deep water infestation.
For Aromatic Substances Series:
Environmental Fate: Large, molecularly complex polycyclic aromatic hydrocarbons, or PAHs, are persistent in the environment longer than smaller PAHs.
Atmospheric Fate: PAHs are 'semi-volatile substances" which can move between the atmosphere and the Earth's surface in repeated, temperature-driven cycles of deposition and volatilization. Terrestrial Fate: BTEX compounds have the potential to move through soil and contaminate ground water, and their vapors are highly flammable and explosive.
Ecotoxicity - Within an aromatic series, acute toxicity increases with increasing alkyl substitution on the aromatic nucleus. The order of most toxic to least in a study using grass shrimp and brown shrimp was dimethylnaphthalenes > methylnaphthalenes >naphthalenes. Anthrcene is a phototoxic PAH. UV light greatly increases the toxicity of anthracene to bluegill sunfish. Biological resources in strong sunlight are at more risk than those that are not. PAHs in general are more frequently associated with chronic risks.
For petroleum distillates:
Environmental fate:
When petroleum substances are released into the environment, four major fate processes will take place: dissolution in water, volatilization, biodegradation and adsorption. These processes will cause changes in the composition of these UVCB substances. In the case of spills on land or water surfaces, photodegradation-another fate process-can also be significant.
As noted previously, the solubility and vapour pressure of components within a mixture will differ from those of the component alone. These interactions are complex for complex UVCBs such as petroleum hydrocarbons.
Each of the fate processes affects hydrocarbon families differently. Aromatics tend to be more water-soluble than aliphatics of the same carbon number, whereas aliphatics tend to be more volatile. Thus,when a petroleum mixture is released into the environment, the principal water contaminants are likely to be aromatics, whereas aliphatics will be the principal air contaminants . The trend in volatility by component class is as follows: alkenes = alkanes > aromatics = cycloalkanes.
The most soluble and volatile components have the lowest molecular weight; thus there is a general shift to higher molecular weight components in residual materials.
Biodegradation:
Biodegradation is almost always operative when petroleum mixtures are released into the environment. It has been widely demonstrated that nearly all soils and sediments have populations of bacteria and other organisms capable of degrading petroleum hydrocarbons Degradation occurs both in the presence and absence of oxygen. Two key factors that determine degradation rates are oxygen supply and molecular structure. In general, degradation is more rapid under aerobic conditions. Decreasing trends in degradation rates according to structure are as follows:
(1) n-alkanes, especially in the C10–C25 range, which are degraded readily;
(2) isoalkanes;
(3) alkenes;
(4) benzene, toluene, ethylbenzene, xylenes (BTEX) (when present in concentrations that are not toxic to microorganisms);
(5) monoaromatics;
(6) polynuclear (polycyclic) aromatic hydrocarbons (PAHs); and
(7) higher molecular weight cycloalkanes (which may degrade very slowly.
Three weathering processes-dissolution in water, volatilization and biodegradation-typically result in the depletion of the more readily soluble,volatile and degradable compounds and the accumulation of those most resistant to these processes in residues.
When large quantities of a hydrocarbon mixture enter the soil compartment, soil organic matter and other sorption sites in soil are fully saturated and the hydrocarbons will begin to form a separate phase (a non-aqueous phase liquid, or NAPL) in the soil. At concentrations below the retention capacity for the hydrocarbon in the soil, the NAPL will be immobile this is referred to as residual NAPL . Above the retention capacity, the NAPL becomes mobile and will move within the soil
Bioaccumulation:
Bioaccumulation potential was characterized based on empirical and/or modelled data for a suite of petroleum hydrocarbons expected to occur in petroleum substances.
Bioaccumulation factors (BAFs) are the preferred metric for assessing the bioaccumulation potential of substances, as the bioconcentration factor (BCF) may not adequately account for the bioaccumulation potential of substances via the diet, which predominates for substances with log Kow > ~4.5
In addition to fish BCF and BAF data, bioaccumulation data for aquatic invertebrate species were also considered. Biota-sediment/soil accumulation factors (BSAFs), trophic magnification factors and biomagnification factors were also considered in characterizing bioaccumulation potential.
Overall, there is consistent empirical and predicted evidence to suggest that the following components have the potential for high bioaccumulation, with BAF/BCF values greater than 5000: C13–C15 isoalkanes, C12 alkenes, C12–C15 one-ring cycloalkanes, C12 and C15 two-ring cycloalkanes, C14 polycycloalkanes, C15 one-ring aromatics, C15 and C20 cycloalkane monoaromatics, C12–C13 diaromatics, C20 cycloalkane diaromatics, and C14 and C20 three-ring PAHs
These components are associated with a slow rate of metabolism and are highly lipophilic. Exposures from water and diet, when combined, suggest that the rate of uptake would exceed that of the total elimination rate. Most of these components are not expected to biomagnify in aquatic or terrestrial foodwebs, largely because a combination of metabolism, low dietary assimilation efficiency and growth dilution allows the elimination rate to exceed the uptake rate from the diet; however,
one study suggests that some alkyl-PAHs may biomagnify.While only BSAFs were found for some PAHs, it is possible that BSAFs will be > 1 for invertebrates, given that they do not have the same metabolic competency as fish.

Continued...

In general, fish can efficiently metabolize aromatic compounds. There is some evidence that alkylation increases bioaccumulation of naphthalene but it is not known if this can be generalized to larger PAHs or if any potential increase in bioaccumulation due to alkylation will be sufficient to exceed a BAF/BCF of 5000.
Some lower trophic level organisms (i.e., invertebrates) appear to lack the capacity to efficiently metabolize aromatic compounds, resulting in high bioaccumulation potential for some aromatic components as compared to fish.
This is the case for the C14 three-ring PAH, which was bioconcentrated to a high level (BCF > 5000) by invertebrates but not by fish. There is potential for such bioaccumulative components to reach toxic levels in organisms if exposure is continuous and of sufficient magnitude, though this is unlikely in the water column following a spill scenario due to relatively rapid dispersal
Bioaccumulation of aromatic compounds might be lower in natural environments than what is observed in the laboratory. PAHs may sorb to organic material suspended in the water column (dissolved humic material), which decreases their overall bioavailability primarily due to an increase in size. This has been observed with fish
Ecotoxicity:
Diesel fuel studies in salt water are available. The values varied greatly for aquatic species such as rainbow trout and Daphnia magna, demonstrating the inherent variability of diesel fuel compositions and its effects on toxicity. Most experimental acute toxicity values are above 1 mg/L. The lowest 48-hour LC50 for salmonids was 2.4 mg/L. Daphnia magna had a 24-hour LC50 of 1.8 mg/.The values varied greatly for aquatic species such as rainbow trout and Daphnia magna, demonstrating the inherent variability of diesel fuel compositions and its effects on toxicity. Most experimental acute toxicity values are above 1 mg/L. The lowest 48-hour LC50 for salmonids was 2.4 mg/L. Daphnia magna had a 24-hour LC50 of 1.8 mg/L
The tropical mysid Metamysidopsis insularis was shown to be very sensitive to diesel fuel, with a 96-hour LC50 value of 0.22 mg/L this species has been shown to be as sensitive as temperate mysids to toxicants. However,However this study used nominal concentrations,and therefore was not considered acceptable. In another study involving diesel fuel, the effect on brown or common shrimp (Crangon crangon) a 96-hour LC50 of 22 mg/L was determined. A “gas oil”was also tested and a 96-hour LC50 of 12 mg/L.was determined
The steady state cell density of marine phytoplankton decreased with increasing concentrations of diesel fuel, with different sensitivities between species . The diatom Phaeodactylum tricornutum showed a 20% decrease in cell density in 24 hours following a 3 mg/L exposure with a 24-hour no-observed effect concentration (NOEC) of 2.5 mg/L. The microalga Isochrysis galbana was more tolerant to diesel fuel, with a 24-hour lowest-observed-effect concentration (LOEC) of 26 mg/L (14% decrease in cell density), and a NOEC of 25 mg/L.
Finally, the green algae Chlorella salina was relatively insensitive to diesel fuel contamination, with a 24-hour LOEC of 170 mg/L (27% decrease in cell density), and a NOEC of 160 mg/L . All populations of phytoplankton returned to a steady state within 5 days of exposure
In sandy soils, earthworm (Eisenia fetida) mortality only occurred at diesel fuel concentrations greater than 10 000 mg/kg, which was also the concentration at which sub-lethal weight loss was recorded
Nephrotoxic effects of diesel fuel have been documented in several animal and human studies.Some species of birds (mallard ducks in particular) are generally resistant to the toxic effects of petrochemical ingestion, and large amounts of petrochemicals are needed in order to cause direct mortality

For dithiophosphate alkyl esters and their (zinc) salts:
The physicochemical properties of dithiophosphate alkyl esters parallel their structural similarity. All members of this category are within a narrow molecular weight range (256-354 daltons) and are highly acidic. In addition, modeling data indicate they have similar melting and boiling points, low water solubility, low vapor pressure, and are lipophilic in nature.
Modeling data indicates that the vapor pressure of these substances range from 3.46 x 10-3 to 1.9 x 10-5 mm Hg at 25 C and generally follow a pattern based upon their molecular weight and the extent of branching of the alkyl side chain.
Modeling data indicates that these substances have low water solubility and that the log of the octanol-water partition coefficient (log Kow) of these substances range from 4.48-7.99. The low water solubility is consistent with the high lipophilic nature of these substances.
Members of the zinc dialkyldithiophosphate category, described here, contain alkyl chain lengths that range from C3-10, or tetrapropenylphenol (range = C10-15, C12 enriched). It is common for zinc dialkyldithiophosphates to contain mixed alkyl esters (e.g., C4, C5), although derivatives with single chain lengths (e.g., C8) are included in the category . As a result of this diversity in alkyl side chain length, the molecular weight distribution for the members of the category is broad, 578 to 1303 gm/mol. Due to the predominant influence of carbon chain length on molecular weight.
Vapour pressures for the zinc dialkyldithiophosphates are thought to be less than 0.5 mm Hg.
Unpublished data for a commercial zinc dialkyldithiophosphate with an alkyl group less than C8 indicates a water solubility of 1.6 mg/L. The zinc dialkyldithiophosphates are generally regarded to be poorly soluble in water.
Unpublished company data on a commercial zinc dialkyldithiophosphate with a carbon chain length of less than eight yielded a log Kow value of 2.49. Longer chain materials are likely to have higher octanol/water partition coefficients. The log Kow is a measure of the lipophilicity of a substance and is used as a surrogate indicator of the potential of a chemical substance to bioaccumulate in aquatic organisms. While Log Kow is a good predictor of bioaccumulation for nonpolar organic compounds, the mechanisms for uptake and depuration of metals and metal compounds are very complex and variable. For metal compounds, the Log Kow data are not indicative of the bioaccumulation potential .
Fate and Transport Characteristics. Members of this category are expected to be poorly biodegradable.
The members of the category are resistant to hydrolysis at room temperature because they lack readily hydrolysable moieties. When heated hydrolytic degradation results in the formation of the phosphorothioic acid ester and hydrogen sulfide. Continued heating at high temperatures results in the formation of the mono-ester and eventually, phosphorothioic acid itself.
These materials are known to be thermally labile at temperatures >120 C. This decomposition mechanism is key to how the zinc salts provide anti-wear and anti-oxidation performance enhancements in engine oils.
Photodegradation is not expected to cause significant physical degradation of dithiophosphate alkyl esters. Category members do not contain bonds that have a high potential to absorb UV light above 290 nm. These substances have low vapor pressure, which indicates that they have a low potential to partition into the air to a significant extent where they would be subject to indirect photodegradation.
These substances are not expected to partition to water or air if released into the environment due to their low water solubility and low vapor pressure. They are also hydrophobic in nature, which suggests that any which reaches the water compartment will be immobilized through binding to the organic component of soils and sediments.
A Japanese MITI publication cited a bioaccumulation factor of less than 100 for a C4-5 ester zinc dithiophosphate indicating a low potential for bioconcentration or cumulative effects. .
The hydrocarbon portion of these compounds that is susceptible to biodegradation is present in both the zinc dialkyldithiophosphates and the dithiophosphate alkyl esters. Therefore, it is expected that the dithiophosphate alkyl esters will behave similarly. The zinc salts are poorly biodegradable.
Ecotoxicity:
The low water solubility suggests that the acute aquatic toxicity of these substances should be low due to limited bioavailability to aquatic organisms. However, the length of the alkyl side chains on these substances will influence their relative water solubility, and, hence, their relative toxicity. Diethyl dithiophosphate for example is highly toxic to Daphnids Zinc O,O-bis(isooctyl)dithiophosphate (CAS RN 28629-66-5) also appears to be harmful to aquatic organisms such as fish and Daphnids.
Studies on various thiophosphates indicated complete mineralization within three weeks by acclimation. A water stability study demonstrated the nature of hydrolysis involves the attack of water molecule on the phosphorus ester involving P-O bond fission. .
for chlorpyrifos:
.log Kow : 4.96-5.11
Koc : 4381-13600
Environmental fate:
Plants: Non-systemic in plants, not absorbed from soil via roots. residues taken up by plant tissues are metabolised to 3,5,6-trichloro-2-pyridinol which is conjugated and sequestered.
Soil and Water: In soil, chlorpyrifos is slowly degraded, with a half-life of about 60-120 days to 3,5,6-trichlorpyridin-2-ol which is degraded to organochlorine compounds and carbon dioxide.
Half-life (hr) air : 6.34
Half-life (hr) H2O surface water : 840-1872
Half-life (hr) soil : 240-1344
Henry's atm m3 /mol: 7.80E-05
BCF : 14
log BCF : 2.5-3.54
* Handbook of Pesticide Toxicology (Hayes, Wayland, J. Jr & Laws E.R), Vol.2 pg 1065, Academic Press Inc.1991

The vapor pressure of chlorpyrifos is 1.9x10-5 mm Hg at 25 C (2.5x10-8 atm). This suggests that while chlorpyrifos is in the atmosphere, it will exist primarily in the vapor phase but will also partition to available airborne particulate. The removal rate by dry deposition is low for such compounds therefore, depending on its reactivity characteristics and the amount of available airborne particulate, chlorpyrifos may travel long distances in the air. The low solubility of chlorpyrifos at 1.12 mg/L at 24 C indicates that dry deposition is a more important process than wet deposition.

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The transport of chlorpyrifos from water to air can occur due to volatilisation. Compounds with a Henry's law constant (H) of <10-5 atm-m3/mol may volatilize slowly from water (Lyman et al. 1990). Therefore, chlorpyrifos, with an H value of 6.6x10-6 atm-m3/mol at 25 C may volatilise slowly from water. The dimensionless Henry's law constant (H') or air/water partition coefficient for chlorpyrifos, as calculated from vapour pressure and solubility data, has been reported to be 5x10-4, 7.3x10-4, and 1.7 +/- 0.3x10-4 . Using these data, the estimated volatilisation half-life from a river 1 meter deep flowing 1 m/set with a wind velocity of 3 m/sec is estimated to be 9 days.
Both chlorpyrifos and its degradation product, TCP, have ultraviolet (UV) absorbencies above 295 nm, indicating their susceptibility to photodegradation by sunlight. The photodegradation half-life of chlorpyrifos in the laboratory is approximately 2.6 days. While in the atmosphere, chlorpyrifos will react with photochemically induced hydroxyl radicals. Its estimated halflife is 6.34 hours The processes primarily responsible for the transformation and degradation of chlorpyrifos in water are abiotic hydrolysis and photosensitised oxidation. Neutral hydrolysis is favored below pH 9, whereas alkaline hydrolysis dominates above pH 9. Thus, both the disappearance half-life and the products are pH-dependent. Neutral hydrolysis yields O-ethyl-O-3,5,6-trichloro-2-pyridyl phosphorothioate, while alkaline hydrolysis occurs by base-catalysed cleavage at the phosphate ester linkage to produce TCP and phosphorothioic acid.
Under field conditions, chlorpyrifos exhibits very short persistence in the water compartment of aquatic ecosystems, and half-lives as short as several hours have been observed. This is due to its considerable volatility from water (arising from low solubility and moderate vapor pressure) and its high association with sediment.
In macrophyte-dominated freshwater model ecosystems, Elodea nutalli vegetation adsorbed a large proportion of the dose of chlorpyrifos applied and hampered mixing of the insecticide in the water column. Only a relatively small proportion of the applied dose became incorporated in the sediment. In open water model ecosystems, however, mixing was rapid and the sediment compartment, particularly its upper layer, was a sink for chlorpyrifos.
The transport processes that may move chlorpyrifos from soil to other media are volatilisation, leaching, runoff, and biotransfer by plants. Post-application volatilisation of chlorpyrifos applied as an agricultural insecticide and subsequent atmospheric transport is thought to be a primary means by which chlorpyrifos is dispersed throughout the environment. Volatilisation is affected by soil cultivation practices (no-till cultivation increase losses).
Volatilisation rates, which result from the complex interplay between chlorpyrifos sorbed to soil, dissolved in the soil pore water, and present in the soil air spaces, can be quite variable.
Leaching studies have shown chlorpyrifos to have little mobility in soil. Laboratory leaching studies revealed that all the surface-applied residues of chlorpyrifos were confined to the upper 5 cm of several soils after elution with 20 cm of water. Field studies have confirmed this lack of mobility, with chlorpyrifos residues being confined to the upper 12 inches of soils in several trials.
Studies indicate that runoff of chlorpyrifos is of minor environmental significance.
Chlorpyrifos may undergo degradation on the surface of soils by photo-induced reactions. Laboratory photodegradation of chlorpyrifos on soil surfaces with UV light (254 nm from mercury lamps) demonstrated that three different photochemical processes (hydrolysis, dechlorination, and oxidation) take place simultaneously. The oxidative and dehalogenated products formed during photo-irradiation of soil undergo further photolysis to form chloropyridinols and O,O-diethyl phosphorothioic acid. The oxon is unstable; it tends to hydrolyse more rapidly than chlorpyrifos and does not accumulate in the soil. With the passage of time, the percentage of chlorinated pyridinols also decreased, suggesting that these products are mineralised in the soil under UV-photo-irradiation conditions. Under simulated sunlight conditions, the rate of photodegradation of chlorpyrifos on a leaf surface was slow. Chlorpyrifos was stable up to 10 days; then the oxon (1.5%) and the hydrolytic product, TCP (2.5%), were detected. Dehalogenated analogs of chlorpyrifos could be detected only after 15 days of constant irradiation. Under these conditions, the photo-oxidation process was more predominant than the photohydrolytic or dehalogenation process. Formation of such photoproducts on an irradiated soil surface was very fast, but the rates in the laboratory will differ from those found under environmental conditions.
Chlorpyrifos undergoes transformation in soil by the processes of abiotic hydrolysis and microbial degradation. All half-lives were shorter in the natural soils as opposed to the sterile soils, however, indicating microbial degradation in addition to abiotic chemical hydrolysis. The biotic process at work is probably co-metabolism.
In a study of persistence of chlorpyrifos in a silt loam soil, the disappearance rate was fast in the first 15 days, but slowed after that. The calculated half-lives ranged from 15.8 to 17.3 days.
Research has shown that only very small levels of chlorpyrifos are taken up by plant roots, translocated, or metabolised by plant tissues. In one study, after 7 days, <1% of the chlorpyrifos applied was found in nontreated areas of the plant. In another study however researchers have found that soil-applied doses of chlorpyrifos are transported to foliage. Chlorpyrifos is expected to have reduced bioavailability from soil and water as it is adsorbed strongly to soil.
Ecotoxicity:
Birds: Acute oral LD50 for chickens 32-102 mg/kg;
LC50 for bobwhite quail 423 ppm
LD50 for duck 76 mg/kg *
Fish LC50 (96 h) for rainbow trout 0.003, roach 0.25 mg/l
Bees: Toxic to bees. LD50 (contact) 59 ng/bee, (oral) 250 ng/bee
Daphnia EC50 (48 h) 1.7 ug/l
For organophosphorus compounds:
Environmental fate:
Organophosphorus compounds and pesticides are relatively non-persistent in the environment with half-lives ranging from hours to several weeks or months. Only rarely are pesticides found in crops beyond the growing season during which they are applied. Chemical or photochemical mechanisms may produce a leaving group which is easily degraded. As a rule these compounds do not represent a serious problem as contaminants of soil and water. Breakdown products are usually non-toxic being composed of low-molecular weight, volatile molecules that are easily degraded and utilised by micro-organisms.
Being esters they are also susceptible to hydrolysis. Most organophosphorus pesticides are stable to acid pHs but under alkaline conditions hydrolysis is rapid with the breakdown rate increasing 10-fold for each pH unit above 7. An increase of 10 deg. C of temperature will increase the hydrolysis rate approximately 4-fold. When these compounds are present in the soil their disappearance is affected by their interaction with the physical characteristics and water content of the soil, and the microflora present.
In certain types of soil strong binding may make them unavailable for biological decomposition. In such soils even running water produces little movement and thus minimal contamination of water supplies. Less tightly bound substances are similarly unlikely to produce substantial contamination because of rapid breakdown. Metallic ions in the soil interact with organophosphorus esters through hydrogen linkage whilst increased organic matter facilitates further binding.
In general only minute amounts of residue and their breakdown products are found in natural water systems. In soil however there is a greater likelihood of the presence and buildup of toxic residues.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
chlorpyrifos	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
chlorpyrifos	HIGH (BCF = 2880)
solvent naphtha petroleum, heavy aromatic	LOW (BCF = 159)

Mobility in soil

Ingredient	Mobility
chlorpyrifos	LOW (Log KOC = 6829)

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.

Continued...

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.

Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.
A Hierarchy of Controls seems to be common - the user should investigate:
- Reduction
- Reuse
- Recycling
- Disposal (if all else fails)

This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.
- **DO NOT** allow wash water from cleaning or process equipment to enter drains.
- It may be necessary to collect all wash water for treatment before disposal.
- In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.
- Where in doubt contact the responsible authority.
- Recycle wherever possible. Special hazard may exist - specialist advice may be required.
- Consult manufacturer for recycling options.
- Consult State Land Waste Management Authority for disposal.
- Bury or incinerate residue at an approved site.
- Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.
- Puncture containers to prevent re-use and bury at an authorised landfill.

SECTION 14 Transport information

Labels Required



Marine Pollutant	
HAZCHEM	2X

Land transport (ADG)

14.1. **UN number or ID number**	3018
14.2. **UN proper shipping name**	ORGANOPHOSPHORUS PESTICIDE, LIQUID, TOXIC (contains chlorpyrifos)
14.3. **Transport hazard class(es)**	Class: 6.1 Subsidiary Hazard: Not Applicable
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: 61 223 274 Limited quantity: 5 L

Air transport (ICAO-IATA / DGR)

14.1. **UN number**	3018
14.2. **UN proper shipping name**	Organophosphorus pesticide, liquid, toxic * (contains chlorpyrifos)
14.3. **Transport hazard class(es)**	ICAO/IATA Class: 6.1 ICAO / IATA Subsidiary Hazard: Not Applicable ERG Code: 6L
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: A3 A4 Cargo Only Packing Instructions: 663 Cargo Only Maximum Qty / Pack: 220 L Passenger and Cargo Packing Instructions: 655 Passenger and Cargo Maximum Qty / Pack: 60 L Passenger and Cargo Limited Quantity Packing Instructions: Y642 Passenger and Cargo Limited Maximum Qty / Pack: 2 L

Continued...

Sea transport (IMDG-Code / GGVSee)

14.1. **UN number**	3018
14.2. **UN proper shipping name**	ORGANOPHOSPHORUS PESTICIDE, LIQUID, TOXIC (contains chlorpyrifos)
14.3. **Transport hazard class(es)**	IMDG Class: 6.1 IMDG Subsidiary Hazard: Not Applicable
14.4. **Packing group**	III
14.5 **Environmental hazard**	Marine Pollutant
14.6. **Special precautions for user**	EMS Number: F-A, S-A Special provisions: 61 223 274 Limited Quantities: 5 L

14.7. Maritime transport in bulk according to IMO instruments

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

Not Applicable

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

Product name	Group
chlorpyrifos	Not Applicable
solvent naphtha petroleum, heavy aromatic	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
chlorpyrifos	Not Applicable
solvent naphtha petroleum, heavy aromatic	Not Applicable

SECTION 15 Regulatory information

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

chlorpyrifos is found on the following regulatory lists

Australia Chemicals with non-industrial uses removed from the Australian Inventory of Chemical Substances (old Inventory)
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Model Work Health and Safety Regulations - Schedule 10 - Table 14.1 Hazardous chemicals (other than lead) requiring health monitoring
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Chemical Footprint Project - Chemicals of High Concern List
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

solvent naphtha petroleum, heavy aromatic is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (chlorpyrifos; solvent naphtha petroleum, heavy aromatic)
China - IECSC	Yes
Colombia - Colombia National Industrial Chemicals Inventory	No (chlorpyrifos; solvent naphtha petroleum, heavy aromatic)
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	TSCA Inventory 'Active' substance(s) (solvent naphtha petroleum, heavy aromatic); No (chlorpyrifos)

Continued...

National Inventory	Status
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	Yes
UAE - Control List (Banned/Restricted Substances)	No (solvent naphtha petroleum, heavy aromatic)
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	16/11/2026
Initial Date	16/11/2026

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。
- 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
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration
- MARPOL: International Convention for the Prevention of Pollution from Ships
- IMSBC: International Maritime Solid Bulk Cargoes Code
- IGC: International Gas Carrier Code
- IBC: International Bulk Chemical Code

- 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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end of SDS