ERACAST RT30A PART B

Section 1 - CHEMICAL PRODUCT AND COMPANY IDENTIFICATION

PRODUCT NAME
ERACAST RT30A PART B

PRODUCT USE
Used according to manufacturer's directions.
Polyurethane curative

SUPPLIER
Company: Era Polymers Pty Ltd
Address:
25- 27 Green Street, Banksmeadow, NSW 2019, Australia

Telephone: +61 2 9666 3788
Emergency Tel:**1800 039 008 (AUS)**
Emergency Tel:**+80024362255 (INTL)**
Fax: +61 2 9666 4805
Email: erapol@erapol.com.au
Website: ~

Section 2 - HAZARDS IDENTIFICATION

GHS Classification
Acute Toxicity (Dermal) Category 4
Acute Toxicity (Inhalation) Category 4
Acute Toxicity (Oral) Category 4
Chronic Aquatic Hazard Category 3
STOT - RE Category 2

EMERGENCY OVERVIEW

continued...

HAZARD
WARNING

Determined by Chemwatch using GHS criteria

H302	Harmful if swallowed.
H312	Harmful in contact with skin.
H332	Harmful if inhaled.
H373	May cause damage to organs through prolonged or repeated exposure.
H412	Harmful to aquatic life with long lasting effects.

PRECAUTIONARY STATEMENTS

Prevention

Code	Phrase
P260	Do not breathe dust/ fume/ gas/ mist/ vapours/ spray.
P261	Avoid breathing dust/ fume/ gas/ mist/ vapours/ spray.
P264	Wash ... thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P271	Use only outdoors or in a well- ventilated area.
P273	Avoid release to the environment.
P280	Wear protective gloves/protective clothing/eye protection/face protection.

Response

Code	Phrase
P301+P312	IF SWALLOWED: Call a POISON CENTER or doctor/physician if you feel unwell.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.
P312	Call a POISON CENTER or doctor/physician if you feel unwell.
P314	Get medical advice/attention if you feel unwell.
P330	Rinse mouth.
P363	Wash contaminated clothing before reuse.

Disposal

Code	Phrase
P501	Dispose of contents/container to ...

Section 3 - COMPOSITION / INFORMATION ON INGREDIENTS

NAME	CAS RN	%
tris(2- chloroisopropyl)phosphate	13674-84-5	30-60
diethyltoluenediamine	68479-98-1	<2.5
bis(phenylmercury) dodecenylsuccinate	27236-65-3	<1
All other substances non hazardous		>60

Section 4 - FIRST AID MEASURES

SWALLOWED
• IF SWALLOWED, REFER FOR MEDICAL ATTENTION, WHERE POSSIBLE, WITHOUT DELAY.
• For advice, contact a Poisons Information Centre or a doctor.
• Urgent hospital treatment is likely to be needed.
• In the mean time, qualified first-aid personnel should treat the patient following observation and

continued...

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 MSDS 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 MSDS.

Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:
- INDUCE vomiting with fingers down the back of the throat, ONLY IF CONSCIOUS. Lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.

NOTE: Wear a protective glove when inducing vomiting by mechanical means.

EYE
■ If this product comes in contact with the eyes:
- Wash out immediately with fresh running water.
- Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.
- Seek medical attention without delay; if pain persists or recurs seek medical attention.
- Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.

SKIN
■ If skin contact occurs:
- Immediately remove all contaminated clothing, including footwear.
- Flush skin and hair with running water (and soap if available).
- Seek medical attention in event of irritation.

INHALED
- If fumes or combustion products are inhaled remove from contaminated area.
- Lay patient down. Keep warm and rested.
- Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.
- Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.
- Transport to hospital, or doctor.

NOTES TO PHYSICIAN
Treat symptomatically.
For acute and short term repeated exposures to aryl and alkylmethoxy compounds of mercury: Absorption proceeds more rapidly than its inorganic counterpart but once inside the body biotransformation releases inorganic mercury. [Ellenhorn and Barceloux: Medical Toxicology].
- Moderate adsorption of inorganic mercury compounds through the gastro-intestinal tract (7-15%) is the principal cause of poisoning. These compounds are highly concentrated (as the mercuric (Hg (2+) form) in the kidney; acute ingestion may lead to oliguric renal failure. Severe mucosal necrosis may also result from ingestion.
- Chronic effects range from proteinuria to nephrotic syndrome. Chronic presentation also involves dermatitis, gingivitis, stomatitis, tremor and neuropsychiatric symptoms of erethism.
- Absorbed inorganic mercury does not significantly cross the blood-brain barrier.
- Emesis and lavage should be initiated following acute ingestion.
- Activated charcoal interrupts absorption; cathartics should be administered when charcoal is given.
- The use of British Anti-Lewisite is indicated in severe inorganic poisoning. Newer derivatives of BAL (e.g. dimercaptosuccinic acid, [DMSA] and 2,3-dimercapto-1-propanesulfate [DMPS]) may prove more effective. [Ellenhorn and Barceloux: Medical Toxicology]

BIOLOGICAL EXPOSURE INDEX - BEI

continued...

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

Determinant	Index	Sampling Time	Comments
1. Total inorganic mercury in urine	35 ug/gm creatinine	Preshift	B
2. Total inorganic mercury in blood	15 ug/L	End of shift at end of workweek	B

B: Background levels occur in specimens collected from subjects NOT exposed.
All persons handling organic phosphorus ester materials regularly should undergo regular medical examination with special stress on the central nervous systems. Whilst atropine or pyridine-2-aldoxime methiodide (PAM) are beneficial antidotes for acute phosphate ester poisonings, they are of little value in reversing acute or chronic neurological damage due to phosphites and some types of aryl phosphate.

Section 5 - FIRE FIGHTING MEASURES

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

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 water delivered as a fine spray to control fire and cool adjacent area.
- Avoid spraying water onto liquid pools.
- 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.

FIRE/EXPLOSION HAZARD
■ Combustion products include: carbon dioxide (CO_2), hydrogen chloride, phosgene, phosphorus oxides (POx), other pyrolysis products typical of burning organic material.
The most important route of thermal degradation of the chlorinated trisphosphates is elimination of phosphoric acid, with consequent introduction of double bonds into the aliphatic moiety (such as vinyl chloride from tris(chloroethyl)phosphate and dichloropropenes from tris(dichloropropyl)phosphate)).
In a real situation, where oxygen is present, such as in combustion of materials into which the triphosphate has been incorporated, there will be many products of thermal degradation and partial combustion including hydrogen chlorides, oxides of carbon and oxidised carbon compounds such as ketones.
Flame retardants may not themselves be immune from combustion but will quickly self-extinguish under fire normal conditions. Their thermal degradation products may be be required to break the combustion cycle of materials in which they are found. When materials burn they introduce flammable gases into the immediate environment, The gas flame itself is maintained by the action of high energy "radicals" (that is H+ and OH- in the gas phase) which decompose molecules to give free carbon. This free carbon may react with oxygen in air to " burn" to CO_2, generating heat energy.
Halogenated flame retardants act by effectively removing the H+ and OH- radicals in the gas flame phase. This considerable slows or prevents the burning process, thus reducing heat generation and, as a result, th production of further gaseous material. The halogenated flame retardants release bromine or chlorine as free radicals (Br- or Cl- as appropriate) which react with the flammable gases to give off HBr or HCl. These then react with the high energy H+ or OH- radicals to give water and the much lower energy Br- or Cl- radicals

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which then become available to begin a new cycle of H+ and OH- radical removal.
Because chlorine (from chorinated retardants) is released over a wider range of temperatures than bromine, it is present in the flame zone at lower concentrations and is thus less effective.
Phosphorus-containing flame retardants effectively work in the solid phase of burning materials (as distinct from the burning gas above them). When heated the phosphorus reacts to give a polymeric form of phosphoric acid. This acid causes the material to char, forming a glassy layer, and thus inhibits the "pyrolysis" process (which causes breakdown of the solid to release flammable gases which further fuel the fire).
May emit poisonous fumes.

FIRE INCOMPATIBILITY
• Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result.

Section 6 - ACCIDENTAL RELEASE MEASURES

MINOR SPILLS
• 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
Chemical Class: organophosphates
For release onto land: recommended sorbents listed in order of priority.

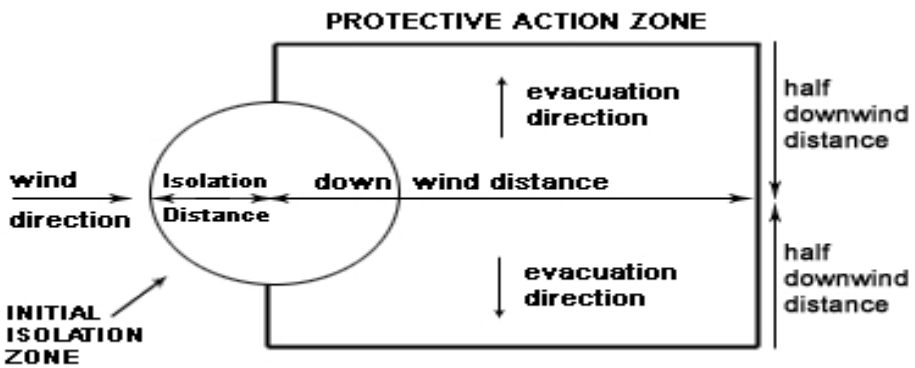
SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS
LAND SPILL - SMALL				
cross- linked polymer - particulate	1	shovel	shovel	R, W, SS
cross- linked polymer - pillow	1	throw	pitchfork	R, DGC, RT
wood fiber - pillow	1	throw	pitchfork	R, P, DGC, RT
foamed glass - pillow	2	shovel	shovel	R, W, P, DGC
sorbent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

continued...

sorbent clay - particulate	2	blower	skiploader	R, I, P
polypropylene - particulate	2	blower	skiploader	R, SS, DGC
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.
Moderate hazard.
• Clear area of personnel and move upwind.
• Alert Fire Brigade and tell them location and nature of hazard.
• Wear breathing apparatus plus protective gloves.
• Prevent, by any means available, spillage from entering drains or water course.
• No smoking, naked lights or ignition sources.
• Increase ventilation.
• Stop leak if safe to do so.
• Contain spill with sand, earth or vermiculite.
• Collect recoverable product into labelled containers for recycling.
• Absorb remaining product with sand, earth or vermiculite.
• Collect solid residues and seal in labelled drums for disposal.
• Wash area and prevent runoff into drains.
• If contamination of drains or waterways occurs, advise emergency services.

PROTECTIVE ACTIONS FOR SPILL



From US Emergency Response Guide 2000
Guide

SMALL SPILLS

Name	Isolation Distance ft (m)	Downwind Day mile (km)	Protection Night mile (km)

LARGE SPILLS

Name	Isolation Distance ft (m)	Downwind Day mile (km)	Protection Night mile (km)
From IERG (Canada/Australia)			
Isolation Distance		-	
Downwind Protection Distance		-	
IERG Number		None	

FOOTNOTES

1 PROTECTIVE ACTION ZONE is defined as the area in which people are at risk of harmful exposure. This zone assumes that random changes in wind direction confines the vapour plume to an area within 30 degrees on either side of the predominant wind direction, resulting in a crosswind protective action distance equal to the downwind protective action distance.

2 PROTECTIVE ACTIONS should be initiated to the extent possible, beginning with those closest to the spill and working away from the site in the downwind direction. Within the protective action zone a level of vapour concentration may exist resulting in nearly all unprotected persons becoming incapacitated and unable to take protective action and/or incurring serious or irreversible health effects.

3 INITIAL ISOLATION ZONE is determined as an area, including upwind of the incident, within which a high probability of localised wind reversal may expose nearly all persons without appropriate protection to life-threatening concentrations of the material.

4 SMALL SPILLS involve a leaking package of 200 litres (55 US gallons) or less, such as a drum (jerrican or box with inner containers). Larger packages leaking less than 200 litres and compressed gas leaking from a small cylinder are also considered "small spills".

LARGE SPILLS involve many small leaking packages or a leaking package of greater than 200 litres, such as a cargo tank, portable tank or a "one-tonne" compressed gas cylinder.

5 Guide is taken from the US DOT emergency response guide book.

6 IERG information is derived from CANUTEC - Transport Canada.

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

Section 7 - HANDLING AND STORAGE

PROCEDURE FOR HANDLING

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

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conditions are maintained.

SUITABLE CONTAINER
• Metal can or drum
• Packaging as recommended by manufacturer.
• Check all containers are clearly labelled and free from leaks.

STORAGE INCOMPATIBILITY
• 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 reaction with oxidising agents.

STORAGE REQUIREMENTS
• Store in original containers.
• Keep containers securely sealed.
• No smoking, naked lights or ignition sources.
• 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 MSDS.

SAFE STORAGE WITH OTHER CLASSIFIED CHEMICALS

+	X	+	X	X	+

+: May be stored together
O: May be stored together with specific preventions
X: Must not be stored together

Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

EXPOSURE CONTROLS
The following materials had no OELs on our records
• tris(2- chloroisopropyl)phosphate: CAS:13674- 84- 5 CAS:16839- 32- 0 CAS:98112- 32- 4
• diethyltoluenediamine: CAS:68479- 98- 1
• bis(phenylmercury) dodecenylsuccinate: CAS:27236- 65- 3

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EMERGENCY EXPOSURE LIMITS

Material	Revised IDLH Value (mg/m3)	Revised IDLH Value (ppm)
bis(phenylmercury) dodecenylsuccinate|35331	10	

MATERIAL DATA

BIS(PHENYLMERCURY) DODECENYLSUCCINATE:
ERACAST RT30A PART B:

TRIS(2-CHLOROISOPROPYL)PHOSPHATE:
No exposure limits set by NOHSC or ACGIH.

DIETHYLTOLUENEDIAMINE:
Sensory irritants are chemicals that produce temporary and undesirable side-effects on the eyes, nose or throat. Historically occupational exposure standards for these irritants have been based on observation of workers' responses to various airborne concentrations. Present day expectations require that nearly every individual should be protected against even minor sensory irritation and exposure standards are established using uncertainty factors or safety factors of 5 to 10 or more. On occasion animal no-observable-effect-levels (NOEL) are used to determine these limits where human results are unavailable. An additional approach, typically used by the TLV committee (USA) in determining respiratory standards for this group of chemicals, has been to assign ceiling values (TLV C) to rapidly acting irritants and to assign short-term exposure limits (TLV STELs) when the weight of evidence from irritation, bioaccumulation and other endpoints combine to warrant such a limit. In contrast the MAK Commission (Germany) uses a five-category system based on intensive odour, local irritation, and elimination half-life. However this system is being replaced to be consistent with the European Union (EU) Scientific Committee for Occupational Exposure Limits (SCOEL); this is more closely allied to that of the USA.
OSHA (USA) concluded that exposure to sensory irritants can:
• cause inflammation
• cause increased susceptibility to other irritants and infectious agents
• lead to permanent injury or dysfunction
• permit greater absorption of hazardous substances and
• acclimate the worker to the irritant warning properties of these substances thus increasing the risk of overexposure.
CEL TWA: 0.02 ppm, 0.145 mg/m3 [BAYER]

BIS(PHENYLMERCURY) DODECENYLSUCCINATE:
It is the goal of the ACGIH (and other Agencies) to recommend TLVs (or their equivalent) for all substances for which there is evidence of health effects at airborne concentrations encountered in the workplace.
At this time no TLV has been established, even though this material may produce adverse health effects (as evidenced in animal experiments or clinical experience). Airborne concentrations must be maintained as low as is practically possible and occupational exposure must be kept to a minimum.
NOTE: The ACGIH occupational exposure standard for Particles Not Otherwise Specified (P.N.O.S) does NOT apply.
Exposure limits with "skin" notation indicate that vapour and liquid may be absorbed through intact skin. Absorption by skin may readily exceed vapour inhalation exposure. Symptoms for skin absorption are the same as for inhalation. Contact with eyes and mucous membranes may also contribute to overall exposure and may also invalidate the exposure standard.

PERSONAL PROTECTION

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EYE

- Safety glasses with side shields
- Chemical goggles.
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lens or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent].

HANDS/FEET

■ The selection of the suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and
has to be observed when making a final choice.
Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:

- frequency and duration of contact,
- chemical resistance of glove material,
- glove thickness and
- dexterity

Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).

- When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- Contaminated gloves should be replaced.

Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly.
Application of a non-perfumed moisturiser is recommended.
WARNING: Do NOT use latex or PVC gloves

- In 1997, a researcher (Dr. Karen E. Wetterhahn) died from organic mercury poisoning, resulting from a single exposure to dimethylmercury almost a year before.
- Heavy metals and organic metal compounds, in particular, have posed special hazards in worker protection. At the time of diagnosis and before she lapsed into a vegetative state, Dr. Wetterhahn asked that her case be made known to others.

Permeation testing of the potential of transdermal exposure to dimethylmercury produced the following results*.

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Glove material	Thickness in mm*	Breakthrough Time
Nitrile	0.2	0.25 minutes
Neoprene	0.8	<10 mins.
Butyl	0.33	<15 mins.
Viton	0.28	<15 mins.
Silver Shield	0.13	>240 mins.
Silver Shield & Neoprene Pair	0.7	>240 mins.

*Michael B Blayney:
Applied Occupational and Environmental Hygiene: 16, pp 233-236, 2001
* Originally quoted as mil (one mil = 0.001 inches).

OTHER
• Overalls.
• P.V.C. apron.
• Barrier cream.
• Skin cleansing cream.
• Eye wash unit.

RESPIRATOR
•Type AK-P Filter of sufficient capacity. (AS/NZS 1716 & 1715, EN 143:2000 & 149:2001, ANSI Z88 or national equivalent)
■ 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.
Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half- face Respirator	Full- Face Respirator
up to 10	1000	a- AUS / Class1	-
up to 50	1000	-	a- AUS / Class 1
up to 50	5000	Airline *	-
up to 100	5000	-	a- 2
up to 100	10000	-	a- 3
100+			Airline**

* - Continuous Flow ** - Continuous-flow or positive pressure demand
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).

The local concentration of material, quantity and conditions of use determine the type of personal protective equipment required. For further information consult site specific CHEMWATCH data (if available), or your

continued...

Occupational Health and Safety Advisor.

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.
Provide adequate ventilation in warehouse or closed storage area.

Section 9 - PHYSICAL AND CHEMICAL PROPERTIES

APPEARANCE

Brown Colour

PHYSICAL PROPERTIES

Liquid.

State	Liquid	Molecular Weight	Not Available
Melting Range (°C)	Not Available	Viscosity	Not Available
Boiling Range (°C)	Not Available	Solubility in water (g/L)	Not Available
Flash Point (°C)	Not Available	pH (1% solution)	Not Available
Decomposition Temp (°C)	Not Available	pH (as supplied)	Not Available
Autoignition Temp (°C)	Not Available	Vapour Pressure (kPa)	Not Available
Upper Explosive Limit (%)	Not Available	Specific Gravity (water=1)	1.07
Lower Explosive Limit (%)	Not Available	Relative Vapour Density (air=1)	Not Available
Volatile Component (%vol)	Not Available	Evaporation Rate	Not Available

Section 10 - CHEMICAL STABILITY

CONDITIONS CONTRIBUTING TO INSTABILITY

• Presence of incompatible materials.
• Product is considered stable.
• Hazardous polymerisation will not occur.
For incompatible materials - refer to Section 7 - Handling and Storage.

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Section 11 - TOXICOLOGICAL INFORMATION

Health hazard summary table:

Acute toxicity	Acute Tox. (dermal) 4 Acute Tox. (inhal) 4 Acute Tox. (oral) 4
Skin corrosion/irritation	Not applicable
Serious eye damage/irritation	Not applicable
Respiratory or skin sensitization	Not applicable
Germ cell mutagenicity	Not applicable
Carcinogenicity	Not applicable
Reproductive toxicity	Not applicable
STOT- single exposure	Not applicable
STOT- repeated exposure	STOT RE 2
Aspiration hazard	Not applicable

POTENTIAL HEALTH EFFECTS

ACUTE HEALTH EFFECTS

SWALLOWED
■ Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.

EYE
■ Although the liquid is not thought to be an irritant (as classified by EC Directives), direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn).

SKIN
■ Skin contact with the material may be harmful; systemic effects may result following absorption.
■ The material is not thought to be a skin irritant (i.e. is unlikely to produce irritant dermatitis as described in EC Directives using animal models). Temporary discomfort, however, may result from prolonged dermal exposures. Good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting.
■ Open cuts, abraded or irritated skin should not be exposed to this material.
■ Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.

INHALED
■ Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful.
■ The material is not thought to produce respiratory irritation (as classified by EC Directives using animal models). Nevertheless inhalation of vapours, fumes or aerosols, especially for prolonged periods, may produce respiratory discomfort and occasionally, distress.
■ Inhalation hazard is increased at higher temperatures.
■ Chlorinated phosphate esters are distinguished from their non-halogenated congeners by possessing anaesthetic-like and muscle-relaxant properties. Even at high doses, however, they do not appear to produce pathological side-effects.

CHRONIC HEALTH EFFECTS
Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.

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Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects.
On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.

TOXICITY AND IRRITATION
■ unless otherwise specified data extracted from RTECS - Register of Toxic Effects of Chemical Substances.

BIS(PHENYLMERCURY) DODECENYLSUCCINATE:
ERACAST RT30A PART B:
■ No significant acute toxicological data identified in literature search.

TRIS(2-CHLOROISOPROPYL)PHOSPHATE:
ERACAST RT30A PART B:
■ For non-polymeric chlorinated trisphosphates (typically (tris(chloroethyl)phosphate (TCEP), tris(chloropropyl)phosphate (TCPP) and tris(dichoropropyl)phosphate (TDCPP)

Chlorinated trisphosphates do not necessarily have similar chemical, physical, toxicological or environmental properties.
Blooming has been identified as a source of potential exposure (human and environmental) to trisphosphate plasticers/ flame retardants. Blooming is defined as the migration (or more appropriately, diffusion) of an ingredient in rubber or plastic to the outer surface after curing. Thus is generally a slow process.
Increased temperature may accelerate the rate of migration. For example trisphosphates are know to bloom from car interior plastics, TVs and computer VDUs
Acute toxicity:
In rats, oral doses of TCEP are absorbed and distributed around the body to various organs, particularly the liver and kidney, but also the brain. Metabolites in rats and mice include bis(2-chloroethyl) carboxymethyl phosphate; bis(2-chloroethyl) hydrogen phosphate; and bis(2-chloroethyl)-2-hydroxyethyl phosphate glucuronide. Excretion is rapid, nearly complete and mainly via the urine. TCEP is of low to moderate acute oral toxicity (oral LD50 in the rat = 1150 mg/kg body weight). In repeat dose studies, TCEP caused adverse effects on the brain (hippocampal lesions in rats), liver and kidneys. The NOEL was 22 mg/kg body weight per day and the LOEL 44 mg/kg body weight per day for increased weights of liver and kidneys in rats
TCPP is of low to moderate acute toxicity by the oral (LD50 in rats = 1017-4200 mg/kg body weight), dermal (LD50 in rats and rabbits is > 5000 mg/kg body weight) and inhalation routes (LC50 in rats is > 4.6 mg/litre).
TDCPP is of low to moderate acute toxicity by the oral route (LD50 in rats = 2830 mg/kg body weight) and of low acute toxicity by the dermal route (dermal LD50 in rats is > 2000 mg/kg body weight). In a 3-month study in mice, an exposure of approximately 1800 mg/kg body weight per day caused death within one month. The no-observed-effect level (NOEL) for the study was 15.3 mg/kg body weight per day; the lowest-observed level (LOEL) for increased liver weight was 62 mg/kg body weight per day.
Irritation studies: TCEP is non-irritant to skin and eyes, but has not been tested for sensitization potential.
Rabbit eye and skin irritancy studies have indicated that TCPP is either non-irritant or mildly irritant.
Sensitisation studies: A skin sensitization study showed that TCPP has no sensitizing properties. The sensitization potential of TDCPP has not been investigated
Neurotoxicity: A very high oral dose of TCEP caused some inhibition of plasma cholinesterase and brain neuropathy target esterase in hens, but did not cause delayed neurotoxicity. In rats, a high dose of TCEP caused convulsions, brain lesions and impaired performance in a water maze.
Developmental toxicity: TCEP is not teratogenic
A TDCPP teratology study on rats showed foetotoxicity at an oral dose of 400 mg/kg body weight per day; there was maternal toxicity at doses of 100 and 400 mg/kg body weight per day. No teratogenicity was seen

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Reproductive toxicity: TCEP adversely affects the fertility of male rats and mice. Effects on the reproductive system (i.e. effects on testes) were noted in a reproduction study in mice.
The potential for TDCPP to affect human male reproductive ability is unclear in view of testicular toxicity in rats but a lack of effect on male reproductive performance in rabbits. The possible effect on female reproduction has not been investigated.
In a 2-year carcinogenicity study in rats, using tris(dichloroisopropyl)phosphate (TDCiPP), effects wer observed on the reproductive system of male rats (i.e. effects on testes). The effects were not confirmed in a fertility study in male rabbits. However, the nature of the reproductive toxicity of TDCiPP has not been sufficiently investigated in a well-designed study.
Histological abnormalities were identified in the testes and seminal vesicles in male rats. A LOAEL of 5 mg/kg is derived from this study. An LOAEL of 5 mg/kg has been proposed
Mutagenicity: No conclusions can be drawn about the mutagenicity of TCEP as in vitro test results were inconsistent and an in vivo bone marrow micronucleus test gave equivocal results.
The results of in vitro and in vivo mutagenicity studies investigating an appropriate range of end-points indicate that TCPP is not genotoxic. TCPP has been investigated for potential delayed neurotoxicity in hens. There was no evidence of delayed neurotoxicity when two oral doses (each of 13 230 mg/kg body weight) were given 3 weeks apart.
Overall, the mutagenicity data show that TDCPP is not genotoxic in vivo.
Carcinogenicity: TCEP causes benign and malignant tumours at various organ sites in rats and mice.
The carcinogenicity of TDCPP has been investigated in a single 2-year feeding study. It was carcinogenic (increased occurrence of liver carcinomas) at all exposure levels that were tested (5-80 mg/kg body weight per day) in both male and female rats. Kidney, testicular and brain tumours were also found. In addition, there were non-neoplastic adverse effects in bone marrow, spleen, testis, liver and kidney. The effects in the kidney and testis occurred at all exposure levels. Only animals in the highest dose and control groups were evaluated for effects in the bone marrow and spleen. It was impossible, therefore, to determine whether there was a dose-response relationship for these effects in these organs.
TDCiPP produces liver tumours in rats.
Immunotoxicity: TDCPP exposure produced some indications of immunotoxicity in mice but only at high doses. Limited human studies following occupational exposure are available but they add little to the knowledge of the safety aspects of TDCPP.

ERACAST RT30A PART B:
~OTHER

TRIS(2-CHLOROISOPROPYL)PHOSPHATE:

TOXICITY	IRRITATION
Dermal (rabbit) LD50:>5000 mg/kg*	Eye (rabbit):non- irritating*
Inhalation (rat) LC50:>4.6 mg/kl/4H*	Skin (rabbit):Mild (24 h):
Oral (Rat) LD50:1500 mg/kg *[Akzo Nobel]	
Intravenous (Mouse) LD50:56 mg/kg	

■ For tris(2-chloro-1-methylethyl)phosphate (TCPP)
The flame retardant product supplied in the EU, marketed as TCPP, is actually a reaction mixture containing four isomers. The individual isomers in this reaction mixture are not separated or marketed. The individual components are never produced as such. These data are true for TCPP produced by all EU manufacturers. The other isomers in the mixture include bis(1-chloro-2-propyl)-2-chloropropyl phosphate (CAS 76025-08-6); bis(2-chloropropyl)-1-chloro-2-propyl phosphate (CAS 76649-15-5) and tris(2-chloropropyl) phosphate (CAS 6145-73-9). The assumption is made that all isomers have identical properties in respect of risk assessment. The assumption is justified in part by the fact that they exhibit very similar chromatographic properties, even under conditions optimised to separate them. Predicted physicochemical properties differ to only a small extent.
Chlorinated alkyl phosphate esters (particularly TCPP) were identified as possible substitutes for the fire retardant pentabromodiphenyl ether They appear to be relatively persistent substances, and there is some human health concern. Three substances in this group have been characterised to a degree and serve as a read across reference for TCPP. They include tris(2-chloroethyl)phosphate (TCEP, CAS 115-96-8), tris[2-(chloro-

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chloromethyl)ethyl]phosphate (TDCP, CAS 13674-87-8) and 2,2-bis(chloromethyl)trimethylene bis[bis(2-chloroethyl)phosphate] (V6, CAS 38051-10-4). Other flame retardants in this family, which do not appear as EU HPV (High Production Volume) substances, include tetrakis[2-(chloroethyl)ethylene)diphosphate (CAS 33125-86-9), tris (2,3-dichloro-1-propyl)phosphate (CAS 78-43-3, an isomer of TDCP))
Acute toxicity: The inhalation exposure studies in animals were somewhat equivocal and in general lacking in detailed information. One study yielded an LC50 of > 7 mg/L/4 hr. A limit test yielded an acute LC50 value of >4.6 mg/L/4h. No deaths occurred at this concentration. Toxic signs observed in this study, and in 2 further poorly reported studies, included mild lethargy, matted fur, acute bodyweight depression and convulsions.
From the studies, it appears that TCPP is more toxic when administered whole body as aerosol than by nose-only exposure. This suggests that some of the systemic toxicity observed when TCPP is administered whole body may result from dermal or oral uptake, rather than inhalation. Therefore, it is concluded that TCPP is of low toxicity via the inhalation route.
Studies in rats indicated that TCPP is of moderate toxicity via the oral route of exposure, with LD50 values from the better quality studies ranging from 632 mg/kg up to 4200 mg/kg, with the majority of values determined to be <2000 mg/kg. Common clinical and macroscopic signs of toxicity observed on nearly all studies included depression, ataxia, hunched posture, lethargy, laboured respiration, increased salivation, partially closed eyelids, body tremors, pilo-erection, ptosis, haemorrhagic lungs and dark liver and/or kidneys. A NOAEL of 200 mg/kg can be identified for acute oral toxicity. This is taken from a 1996 study, in which no clinical signs of toxicity were observed in animals dosed with 200 mg/kg TCPP. Based on the results of the acute oral studies, TCPP should be classified with R22, harmful if swallowed.
In a delayed neurotoxicity study conducted in hens, TCPP showed moderate toxicity. The principle effects were reduced mean body weight and food consumption, feather loss and cessation of laying. There was no evidence of inhibited plasma acetylcholinesterase or brain neurotoxic esterase enzyme levels. Therefore, there is no concern for acute delayed neurotoxicity for TCPP.
Studies in rats and rabbits indicated that TCPP is of low toxicity via the dermal route of exposure with LD50 values of >2000mg/kg.
There is an extensive database in animals, indicating that TCPP is non-irritant in the rabbit eye and skin. The lack of any substantial skin or eye irritation and the lack of irritation observed in the acute inhalation studies suggest that TCPP would be unlikely to produce significant respiratory tract irritation.
Evidence from a guinea pig study as well as from a local lymph node assay, indicates that TCPP does not possess significant skin sensitisation potential. No information is available on the respiratory sensitisation potential of TCPP.
Repeat dose toxicity: A study is available in which male and female rats were fed diets containing TCPP for 13 weeks at concentrations corresponding to mean substance intake values of up to 1349 mg/kg/day and 1745 mg/kg/day for males and females respectively. This study indicated the liver and thyroid to be the main target organs affected by TCPP. Effects observed included statistically significant increases in absolute and relative liver weights in males at all doses and females at the two highest doses, periportal hepatocyte swelling in high dose groups and mild thyroid follicular cell hyperplasia in males at all doses and females at the highest dose.Based on the increase in both absolute and relative liver weights, accompanied by mild thyroid follicular cell hyperplasia observed in males of all dose groups, a LOAEL of 52 mg/kg/day is derived and taken forward to risk characterisation. This LOAEL is taken forward in preference to the NOAEL which was identified in a 4-week study in which rats were dosed with TCPP at concentrations of 0, 10, 100 and 1000 mg/kg/day, as it was derived from a study of longer duration. The 4-week study also showed the liver as the target organ, with increased liver weight changes observed in the high dose groups, accompanied by hepatocyte hypertrophy in all high-dose males and one mid-dose male and changes in ALAT activity in high-dose animals.
A two-week study in which rats were fed diets of TCPP at concentrations corresponding to mean substance intake values of up to 1636 mg/kg/day for males and 1517 mg/kg/day for females showed no major clinical signs of toxicity. There was a significant reduction in weight gain and food consumption in high dose males during week 2, but there were no other significant findings.
In a 2-generation reproductive toxicity study in which rats were fed TCPP in the diet over two successive generations, the low-dose of 99 mg/kg for females is considered to be the LOAEL for parental toxicity. This is based on decreased body weight and food consumption seen in mid and high dose parental animals and the effects on uterus weight seen in all dosed animals. For males, a NOAEL of approximately 85 mg/kg is derived for parental toxicity, based on decreased body weights, food consumption and organ weight changes observed at

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mid and high dose groups.
No data are available on inhalation and dermal repeated dose toxicity.
Genotoxicity: The mutagenic potential of TCPP has been well investigated in vitro. Evidence from several bacterial mutagenicity studies shows that TCPP is not a bacterial cell mutagen. TCPP was also shown to be non-mutagenic in fungi. In mammalian cell studies, TCPP did not induce forward mutations at the TK locus in L5178Y mouse lymphoma cells in one study, but in a second study, the result was considered equivocal (in the presence of rat liver S9 fraction). A confirmatory mouse lymphoma was conducted in accordance with the relevant regulatory guidelines. The results of the assay indicate that TCPP shows clastogenic activity in vitro in the presence of metabolic activation.
The main concern for TCPP is clastogenicity, owing to the clearly positive in vitro mouse lymphoma study. In vivo, TCPP was not clastogenic in a mouse bone marrow micronucleus test. TCPP did not induce an increase in chromosomal aberrations in a rat bone marrow cytogenetics assay. In order to further investigate the potential for TCPP to induce DNA damage, an in vivo Comet assay in the rat liver was conducted. The liver was chosen for comet analysis as TCPP caused an increased mutation frequency in the mouse lymphoma assay in the presence of S9 and also induced liver enlargement in repeat dose studies. Under the conditions of this study, TCPP did not induce DNA damage in the liver of rats treated with either 750 or 1500 mg/kg TCPP.
Overall, it is considered that TCPP is not genotoxic in vivo.
Carcinogenicity: TCPP is structurally similar to two other chlorinated alkyl phosphate esters, TDCP (tris [2-chloro-1-(chloromethyl)ethyl] phosphate) and TCEP (tris (2-chloroethyl) phosphate). TDCP and TCEP are non-genotoxic carcinogens, in vivo, and have agreed classifications of Carc Cat 3 R40. Based on the available repeat dose toxicity data for TCPP, supported by a qualitative read-across from TDCP and TCEP, there is a potential concern for carcinogenicity for TCPP by a nongenotoxic mechanism. No quantitative read-across can be performed since there are no insights into an underlying mode of action for TCEP and TDCP which would make a prediction on a relatively potency of TCPP possible. Therefore, as a reasonable worst case approach, a risk characterisation will be carried out for this end-point.
It is proposed that the effects observed in the 90-day study for TCPP are taken as a starting point for risk characterisation. If these effects were to progress to cancer, they would do so by a non-genotoxic mechanism. Therefore, it is proposed that the LOAEL of 52 mg/kg/day, identified from the 90-day study with TCPP, should be used as a basis for risk characterisation of the carcinogenicity endpoint.
Reproductive toxicity: In a two-generation reproductive toxicity study with TCPP, there were no treatment related effects in pre-coital time, mating index, female fecundity index, male and female fertility index, duration of gestation and post-implantation loss. There was no effect on sperm parameters at necropsy. In females, the length of the longest oestrus cycle and the mean number of cycles per animal were statistically significantly increased in high dose animals of both generations. A decrease in uterus weight was observed in all dosed females in F0 and in high dose females in F1. Effects were also noted on pituitary weights, significant in high dose females of both generations. A LOAEL of 99 mg/kg is derived for effects on fertility. This is based on effects on the effect on uterus weight seen in all dosed females in F0 and high dose females in F1.
Developmental toxicity: From the same study, a LOAEL of 99 mg/kg is derived for developmental toxicity. This is based on a treatment related effect on the number of runts observed in all TCPP-treated groups of the F0 generation.
In a separate study, no treatment-related effects on foetal mortality, implantation number, resorption or foetal weight were observed following treatment of pregnant dams with TCPP. Cervical ribs and missing 13th ribs were noted at a low incidence in all treatment groups, but not in the control group. However, as a specific rib count undertaken in the 2-generation study did not reveal an increase in this effect, it is concluded that this is not toxicologically significant. Weaning rate and rearing condition were unaffected by treatment and there was no evidence of any abnormality.
for alkyl esters of phosphoric acid:
The chemicals in this category exhibit a low to moderate order of acute toxicity. The rat oral LD50 values ranged from 500-1000 mg/kg with 2-ethylhexyl phosphate to >36,800 mg/kg for tris(2-ethylhexyl) phosphate. The dermal LD50 values ranged from 1200 to > 2000 mg/kg (rat) with bis(2-ethylhexyl) hydrogen phosphate to > 20,000 mg/kg (rabbit) with tris(2- ethylhexyl) phosphate. The inhalation LC50 values ranged from > 0.447 mg/l (4 hr. rat) with tris(2-ethylhexyl) phosphate to > 5.14 mg/l (4 hr. rat) with triisobutyl phosphate.
Metabolism: Phosphoric acid esters are metabolized via dealkylation. Metabolism studies conducted on the

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tributyl phosphate indicate that dealkylation to form the alkyl alcohol is the primary route of metabolism Phosphoric acid tri-esters are rapidly metabolised to di-esters with mono-diesters also being produced. Studies of tributyl phosphate show that 40-64% of the parent compound is metabolised to dibutyl dihydrogen phosphate and that 1.1-2.1 % is metabolised to the monobutyl species. Therefore, tris(2-ethylhexyl) phosphate is expected to be metabolised to bis(2-ethylhexyl) phosphate (CAS RN: 298-07-7) and mono(2-ethylhexyl) phosphate (CAS RN 1070-03-7). Based on the evidence for dealkylation as the primary metabolic pathway, 2-ethylhexanol is the expected metabolite of tris(2-ethylhexyl) phosphate (CAS RN: 78-42-2) and 2-ethylhexyl phosphate (CAS RN: 12645-31-7). Triisobutyl phosphate is expected to be metabolised similarly as tributyl phosphate, with methoxypropanol as the alcohol metabolite

Oral repeat dose NOAEL's in rats for dibutyl hydrogen phosphate, tributyl phosphate, ethylhexanol, 2-ethylhexanoic acid, bis(2-ethylhexyl) hydrogen phosphate, tris(2-ethylhexyl) phosphate, and triisobutyl phosphate were 30 mg/kg/day (44 days), 75 mg/kg/day (90 days), 125 mg/kg/day (90 days), 100 mg/kg/day (90 days), 250 mg/kg/day (5 days), and 1000 mg/kg/day (90 days), and 68.4-84.3 mg/kg (90 days), respectively.

The weight of the evidence indicates that the members of this category are not genotoxic. Tris(2-ethylhexyl) phosphate, bis(2-ethylhexyl) hydrogen phosphate, 2-ethylhexyl phosphate, dibutyl hydrogen phosphate, tributyl phosphate, triisobutyl phosphate, 2-ethylhexanol, 2- ethylhexanoic acid, and phosphoric acid were negative in the Ames assay. Tris(2-ethylhexyl) phosphate, bis(2-ethylhexyl) phosphate, 2-ethylhexyl phosphate, and 2-ethylhexanol also were negative in the mouse lymphoma assay. Furthermore, tris(2-ethylhexyl) phosphate, dibutyl hydrogen phosphate, tributyl phosphate, and 2-ethylhexanol were negative in the chromosomal aberration assays (in vitro and/ or in vivo). Tris(2-ethylhexyl) phosphate was negative in a sister chromatid exchange assay while 2-ethylhexanoic acid was positive. Triisobutyl phosphate was negative in the in vivo mouse micronucleus assay.

Reproductive toxicity was evaluated with a number of the members of this category. No effects on reproductive organs were observed in repeat dose studies with tris(2-ethylhexyl) phosphate, dibutyl hydrogen phosphate, tributyl phosphate, 2-ethylhexanol, or 2-ethylhexanoic acid. A two generation reproduction study with tributyl phosphate did not find any reproductive effects in rats at the highest dose tested (225 mg/kg/day). No significant effects on reproduction were seen in rats with an oral OECD 422 combined repeat dose toxicity and reproductive/developmental toxicity screen with dibutyl hydrogen phosphate (NOAEL = 1000 mg/kg). Reproductive effects were reported in rats at 300 mg/kg/day and 600 mg/kg/day in a one generation study with 2-ethylhexanoic acid.

Developmental toxicity: The developmental toxicity of tributyl phosphate was evaluated in both rats and rabbits. Tributyl phosphate and triisobutyl phosphate were determined not to be teratogenic. 2-Ethylhexanol was found to cause developmental toxicity only at doses that were maternally toxic. Drinking water and gavage developmental toxicity studies have also been conducted with 2-ethylhexanoic acid in rats and rabbits. Developmental effects in rats at concentrations as low as 100 mg/kg administered in drinking water have been reported. Developmental studies with rats and rabbits concluded that 2-ethylhexanoic acid did not produce developmental effects in rats or rabbits under the conditions of these tests. The authors noted that the rat NOAEL was 100 mg/kg/day based on slight foetotoxicity at 250 mg/kg/day and that the rabbit NOAEL was 250 mg/kg/day (highest dose). The maternal NOAEL's for rats and rabbits were 250 mg/kg/day and 25 mg/kg/day, respectively.

DIETHYLTOLUENEDIAMINE:

TOXICITY	IRRITATION
Oral (rat) LD50:470- 540 mg/kg	Skin (rabbit):slight
Dermal (rabbit) LD50:>700 mg/kg	Eye (rabbit):moderate- SEVERE
Inhalation (rats) LD50:>2.45 mg/l [Manufacturer]	

■ The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

p-Phenylenediamines are oxidised by the liver microsomal enzymes (S9). Pure p-phenylenediamine is non-mutagenic in but becomes mutagenic after it is oxidized. Azo dyes containing phenylenediamine are mutagenic in certain assay most likely due to the formation of oxidized p-phenylenediamine. Modification of the moieties that can be metabolized to p-phenylenediamine by sulfonation, carboxylation or copper complexation eliminated the mutagenic responses.

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Section 12 - ECOLOGICAL INFORMATION

BIS(PHENYLMERCURY) DODECENYLSUCCINATE:
DIETHYLTOLUENEDIAMINE:
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.
Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

DIETHYLTOLUENEDIAMINE:
BIS(PHENYLMERCURY) DODECENYLSUCCINATE:
TRIS(2-CHLOROISOPROPYL)PHOSPHATE:
DO NOT discharge into sewer or waterways.

TRIS(2-CHLOROISOPROPYL)PHOSPHATE:
For non-polymeric chlorinated trisphosphates (typically tris(chloroethyl)phosphates (TCEP), tris(chloropropyl)phosphates (TCPP) and tris(dichloropropyl)phosphates (TDCPP)

Chlorinated trisphosphates are clear liquids or low-melting solids, with little or no odour. They:
• are denser than water, the density increases with the number of chlorines in the molecule
• have low vapour pressure (typically >1 Pa at ambient temperatures and as a consequence have high boiling
• points (>200 C); they are considered to exhibit some volatility (the literature sometimes describes "appreciable" volatility but this is a description relative to other fire retardants)
• cannot be distilled at atmospheric pressures with decomposition
• have high flash points
• are soluble to a limited extent in water (but readily soluble in ketones, alcohols and chlorinated hydrocarbons); solubilities range from 0.053 g/l (TDCPP) to 8 g/l TCEP)
• have moderate log Kow values, increasing with increasing levels of chlorination (1.7 - 3.8)
• are hydrolysed to produce phosphoric acid and a chlorinated alcohol - there may also be hydrolytic attack on aliphatic chlorines to produce ethylene glycol, propylene glycol or glycerol

Environmental fate:
Trisphosphates are somewhat volatile and are likely to be slowly released to the atmosphere from the surfaces of solid articles containing these compounds during normal use. Some may be released to waste water during washing of fabrics containing these substances.
Atmospheric fate: Once in the atmospheric compartment the compounds are destroyed through reaction with atmospheric hydroxyl radicals. Generally th primary pathway for degradation in the atmosphere will be through hydrogen abstraction by OH radicals. Assuming the accepted global average atmospheric concentrations of hydroxyl radicals to be 5 x 10+5 moles/cm3, the atmospheric half-lives range from 35 hrs (TCPP) to 102 hours (TCEP). Degradation of the compounds in the atmosphere is expected to lead to ultimate destruction with formation of HCl, water and carbon dioxde. The phosphorus component is likely to be converted to phosphoric acid and precipitated (along with HCl) to the surface with rain
Aquatic fate: Trisphosphates are normally not readily biodegradable under aerobic conditions and due to their relatively high water solubility are not expected to bioaccumulate
Chlorinated trisphosphates may be released to the water compartment primarily in landfill leachate resulting from degradation of polyurethane foam and polyester.
Generally these compounds show some water solubility and modest log Kows. Even where the log Kow is relatively high (as with TDCPP, log Kow 3.4) environmental models indicate that only around 25% of the compound becomes associated with sludge while 5% may be released into the air - the rest dissolves.
Terrestrial fate: It is estimated that these compounds have an affinity for the organic components of soils and sediments (log Koc >2) and are expected to have low mobility in these media. The relatively high water solubility indicates that partitioning from soil to water phase is possible and that mobility in and from

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soil media may be quite high.
TDCPP is most likely to become associated with soil and sediments and given its apparent resistance to aerobic bacterial degradation is also likely to be persistant in aerobic soils and sediments.
Abiotic degradation: Hydrolysis of the compounds in aqueous solution is likely to be slow in the normal environmental range 4<pH<9. However at elevated temperatures and/ or under extreme pH conditions hydrolysis of the C-Cl bonds may be more rapid
Biodegradation: Chlorinated trisphosphates are generally not readily biodegradable according to OECD criteria but the lower chlorinated species may be "ultimately" or inherently biodegradable Species with higher chlorination seem resistant to biodegradation.
While refractory to aerobic bacterial biodegradation these compounds may be metabolised by higher organisms.
TDCPP had a half-life of 31 hours in water containing killifish (Oryzias latipes) and 42 hours in water containing goldfish (Carassius auratus)
Bioaccumulation : Relatively high water solubilities and modest values of log Kow indicate a low potential for bioaccumulation. Measured bioconcentration factors (BCF) are low. The largest measured BCF is 107 found for killifish (Oryzias latipes) exposed to 0.3-1.2 mg/l TDCPP for 96 hours. Similar low values were found for TCEP. On transfer of fish to clean water elimination of TDCPP from fish tissues was rapid with a half-life for elimination of 1.65 hours
Ecotoxicity
TCEP and TCPP are slightly toxic to aquatic organisms at all trophic levels and TDCPP is moderately toxic to fish. These compounds are slightly toxic to terrestrial species,, aquatic green algae but are non-toxic to sewage bacteria.
for alkyl esters of phosphoric acid:
Environmental fate:
The physicochemical properties and environmental fate of the chemicals in this category are similar. They have a low melting point, a high boiling point or decomposition temperature, and low vapor pressure. The tri-esters are slightly soluble and the others are moderately soluble to soluble in water. The results of the hydrolysis studies with 2-ethylhexyl phosphate (CAS RN: 12645-31-7), and triisobutyl phosphate (CAS RN: 126-71-6), and tributyl phosphate (CAS RN: 126-73-8) indicate that the mono-, di-, and tri-esters all are hydrolytically stable. Fugacity Level III calculations indicate that if they are released into the environment, they will exist predominantly in the soil and/ or soil or the aquatic environment depending on the environmental compartment that they first contact. The log Kow, indicates that they will not bioconcentrate. They exhibit appreciable biodegradation in 28 days or sooner indicating that they are moderately degradable if soluble and will not persist in the environment Tris(2-ethylhexyl) phosphate, which has limited solubility in water, exhibited 0% biodegradation after 28 days in the OECD 301D closed bottle test.
Biodegradation of phosphoric acid esters involves stepwise hydrolysis to ortho-phosphate and alcohol moieties. The alcohol would then be expected to undergo further degradation
Ecotoxicity:
Studies of the ecotoxicity of the chemicals in this category indicate that none of the members are highly toxic to aquatic species. The fish 96-hour LC50 values ranged from >500 mg/l in 0. latipes and >I00 mg/l in 0. mykiss for 2-ethylhexyl phosphate to 23 mg/l in 0. mykiss for triisobutyl phosphate. The invertebrate 48-hour ECso values with Daphnia ranged from 110 mg/l for 2-ethylhexyl phosphate to 11 mg/l for triisobutyl phosphate. The algal 96-hour EC50 values ranged from 4.4 mg/l with tributyl phosphate in S. capricornutum and to 161 mg/l with 2-ethylhexylphosphate in S. capricornutum.
The principal problems of phosphate contamination of the environment relates to eutrophication processes in lakes and ponds. Phosphorus is an essential plant nutrient and is usually the limiting nutrient for blue-green algae. A lake undergoing eutrophication shows a rapid growth of algae in surface waters. Planktonic algae cause turbidity and flotation films. Shore algae cause ugly muddying, films and damage to reeds. Decay of these algae causes oxygen depletion in the deep water and shallow water near the shore. The process is self-perpetuating because anoxic conditions at the sediment/water interface causes the release of more adsorbed phosphates from the sediment. The growth of algae produces undesirable effects on the treatment of water for drinking purposes, on fisheries, and on the use of lakes for recreational purposes.
Aquatic toxicity:
96h EC50: 47 mg/l

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(Daphnia magna) 21 day NOEC: 32 mg/l
Not readily biodegradable. [Akzo Nobel]

DIETHYLTOLUENEDIAMINE:
Marine Pollutant Yes
Phenylenediamines are not readily biodegradable via CO2 evolution, but they are susceptible to both hydrolysis and photodegradation. These materials have been shown not to partition to water or air if released into the environment due to their low water solubility and low vapor pressure. Analytical studies of hydrolysis products indicate that the molecule cleaves at the aromatic carbon-nitrogen bond.
It is difficult to define clearly the ways in which phenylenediamines are eliminated from the hydrosphere. Elimination processes such as oxidation reactions, adsorption, and stripping effects can only be conjectured.
It is impossible to say with any degree of certainty for any of the three isomers what proportion of their elimination is accounted for by biodegradation. The following elimination rates have been found: between 0 and 69 % for o-phenylenediamine, between 0 and 60 % for m-phenylenediamine and between 0 and 100 % for p-phenylenediamine. It is assumed that any phenylenediamines released into the atmosphere are destroyed by photodegradation. The calculated half-life is less than 2 hours. The low POW values indicate that bioaccumulation is unlikely to occur to any significant degree. Only one study has dealt with the behaviour of phenylenediamines in soil, in respect to their soil sorption and geoaccumulation. According to this study, adsorption is relatively strong at low concentrations and expandable clay minerals
but quite weak at higher concentrations. No information is available on the sorption behaviour against organic material.
The substituted p-phenylenediamines and presumably the other isomers, in general, are very toxic to aquatic organisms.
Aromatic amines (arylamines), particularly primary aromatic amines, covalently and irreversibly bind to humic substances present in most natural waters.
All metabolites with moieties of: anilines, benzidines and toluidines are of environmental concern. Anilines and benzidines are both acutely toxic and toxic depending on the specific aquatic species (except algae). Toluidines represent a similar concern, It has been speculated that aqueous solutions of aromatic amines can be oxidised by organic radicals, but there are no actual data on reaction rates. Based on a study of reaction rate data for these compounds an estimate of the half-life of aromatic amines in water is approximately 100 days, assuming a peroxy radical concentration of 10-10 mole/L in sunlit, oxygenated water.

BIS(PHENYLMERCURY) DODECENYLSUCCINATE:
Marine Pollutant Yes
Mercury may occur in the environment as free mercury, Hg(0), mercury ions in salts and complexes, Hg+ and (Hg2)2+ and as organic mercury compounds. Each species has its own set of physical, chemical and toxicologic properties.
In natural systems a dynamic equilibrium between soil and water mercury occurs determined largely by the physicochemical and biological conditions which pertain. Mercury ion is transported to aquatic ecosystems via surface run-off and from the atmosphere. It is complexed or tightly bound to both inorganic and organic particles, particularly sediments with high sulfur content. Organic acids such as fulvic and humic acids are often associated with mercury not bound to particles. Methyl mercury is produced by sediment micro-organisms, nonbiologically in sediments and by certain species of fish. The methylation of mercury by micro-organisms is the detoxification response that allows the organism to dispose of the heavy metal ions as small organometallic complexes. Methylation occurs only within a narrow pH range in which the micro-organism might exist and the rate of synthesis depends on the redox potential, composition of the microbial population, availability of Hg2+ and temperature. Vitamin B12 derivatives are thought to be the methylating agents, because they are the only methyl carbanion- or methyl radical-donating coenzymes known. In addition it has been demonstrated that the livers of yellow-fin tuna and albacore produce methyl mercury results in its desorption at relatively high rates thus little methyl mercury is found in sediments. Demethylation by sediment micro-organisms also occurs at a rapid rate compared with methylation. The best conversion rate for inorganic mercury to methyl mercury under ideal conditions is less than 1.5% per month. Methyl mercury released into surface waters may also undergo photodecomposition into mercury.
Methyl mercury can be bioaccumulated by planktonic algae and fish. In fish, the rate of absorption of methyl

continued...

mercury is faster than that of inorganic mercury and the clearance rate is slower resulting in high concentrations of methyl mercury in muscle tissue. The ratio of organic mercury to total mercury is generally high in fish compared with other aquatic organisms. Selenium which is also present in seawater and other seafoods readily complexes with methyl mercury and is thought to have a protective effect against the toxic action of methyl mercury. The danger of methyl mercury poisoning has been illustrated in Minimata, Japan in the late 1950s following industrial release of mercury into the bay which subsequently resulted in at least 1200 cases of poisoning, some fatal.

Ecotoxicity

Ingredient	Persistence: Water/Soil	Persistence: Air	Bioaccumulation	Mobility
tris(2-chloroisopropyl)phosphate	HIGH	No Data Available	LOW	MED
diethyltoluenediamine	No Data Available	No Data Available	No Data Available	No Data Available
bis(phenylmercury) dodecenylsuccinate	No Data Available	No Data Available	No Data Available	No Data Available

Section 13 - DISPOSAL CONSIDERATIONS

- Containers may still present a chemical hazard/ danger when empty.
- Return to supplier for reuse/ recycling if possible.

Otherwise:

- If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.
- Where possible retain label warnings and MSDS and observe all notices pertaining to the product.

Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.

A Hierarchy of Controls seems to be common - the user should investigate:

- Reduction
- Reuse
- Recycling
- Disposal (if all else fails)

This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.

- DO NOT allow wash water from cleaning or process equipment to enter drains.
- It may be necessary to collect all wash water for treatment before disposal.
- In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.
- Where in doubt contact the responsible authority.
- Recycle wherever possible or consult manufacturer for recycling options.
- Consult State Land Waste Authority for disposal.
- Bury or incinerate residue at an approved site.
- Recycle containers if possible, or dispose of in an authorised landfill.

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Section 14 - TRANSPORTATION INFORMATION

HAZCHEM:
None

NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS: UN, IATA, IMDG

Section 15 - REGULATORY INFORMATION

Indications of Danger:
Xn Harmful

POISONS SCHEDULE S5

REGULATIONS

Regulations for ingredients

tris(2-chloroisopropyl)phosphate (CAS: 13674-84-5, 16839-32-0, 98112-32-4) is found on the following regulatory lists;
"OECD List of High Production Volume (HPV) Chemicals", "OSPAR National List of Candidates for Substitution – United Kingdom"

diethyltoluenediamine (CAS: 68479-98-1) is found on the following regulatory lists;
"OECD List of High Production Volume (HPV) Chemicals"

bis(phenylmercury) dodecenylsuccinate (CAS: 27236-65-3) is found on the following regulatory lists;
"OSPAR List of Chemicals for Priority Action", "United Nations Consolidated List of Products Whose Consumption and/or Sale Have Been Banned, Withdrawn, Severely Restricted or Not Approved by Governments"

No data for ERACAST RT30A PART B (CW: 9-47791)

Section 16 - OTHER INFORMATION

Denmark Advisory list for selfclassification of dangerous substances

Substance	CAS	Suggested codes
tris(2- chloroisopropyl)phosphate	13674- 84- 5	Mut3; R68 Rep3; R63 Xn; R22 Xi; R38

INGREDIENTS WITH MULTIPLE CAS NUMBERS

Ingredient Name	CAS		
tris(2-chloroisopropyl)phosphate	13674-84-5,	16839-32-0,	98112-32-4

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■ Classification of the preparation and its individual components has drawn on official and authoritative sources using available literature references.

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

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