300 MULTI FOUNT

Hazard Alert Code: MODERATE

Chemwatch Material Safety Data Sheet (REVIEW) | Revision No: 2.0 | Chemwatch 22-0062

Issue Date: 14-Dec-2009 | CD 2009/3

Section 1 - CHEMICAL PRODUCT AND COMPANY IDENTIFICATION

PRODUCT NAME
300 Multi Fount

SYNONYMS
"Aqueous, electrolyte solution, fountain solution"

PRODUCT USE
■ Used according to manufacturer's directions. Water treatment additive for lithographic printing.

SUPPLIER
Company: Hurst Australia Pty Ltd
Address:
10 Bellona Avenue
Regents Park
NSW, 2143
AUS
Telephone: +61 2 9644 6888
Emergency Tel: +61 2 9644 6888
Fax: +61 2 9644 6534
Email: info@hurst.com.au

HAZARD RATINGS

	Min	Max
Flammability:	1	
Toxicity:	2	
Body Contact:	2	
Reactivity:	2	
Chronic:	2	

Min/Nil=0
Low=1
Moderate=2
High=3
Extreme=4

Section 2 - HAZARDS IDENTIFICATION

STATEMENT OF HAZARDOUS NATURE
HAZARDOUS SUBSTANCE. NON-DANGEROUS GOODS. According to the Criteria of NOHSC, and the ADG Code.

COMBUSTIBLE LIQUID, regulated under AS1940 for Bulk Storage purposes only.

POISONS SCHEDULE
None

RISK
■ Irritating to eyes and skin.
■ May cause SENSITISATION by skin contact.
■ Toxic to aquatic organisms.
■ Inhalation and/or ingestion may produce health damage*.
■ Cumulative effects may result following exposure*.
■ May produce discomfort of the respiratory system*.
■ Limited evidence of a carcinogenic effect*.
■ Possible respiratory sensitiser*.
■ May be harmful to the foetus/ embryo*.
■ May possibly affect fertility*.
* (limited evidence).

SAFETY
■ Do not breathe gas/ fumes/ vapour/ spray.
■ Use only in well ventilated areas.
■ Keep container in a well ventilated place.
■ Avoid exposure - obtain special instructions before use.
■ To clean the floor and all objects contaminated by this material use water.
■ Keep container tightly closed.
■ In case of contact with eyes rinse with plenty of water and contact Doctor or Poisons Information Centre.
■ If swallowed IMMEDIATELY contact Doctor or Poisons Information Centre (show this container or label).

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Section 3 - COMPOSITION / INFORMATION ON INGREDIENTS

NAME	CAS RN	%
ethylene glycol monobutyl ether	111-76-2	<10
gum arabic	9000-01-5	<10
non-ionic surfactants		<10
phosphoric acid	7664-38-2	<10
5-chloro-2-methyl-4-isothiazolin-3-one	26172-55-4	<10
water	7732-18-5	>60

Section 4 - FIRST AID MEASURES

SWALLOWED

■

- If swallowed do NOT induce vomiting.
- If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.
- Observe the patient carefully.
- Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.
- Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.
- Seek medical advice.

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.
- 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 or short term repeated exposures to ethylene glycol:

- Early treatment of ingestion is important. Ensure emesis is satisfactory.
- Test and correct for metabolic acidosis and hypocalcaemia.
- Apply sustained diuresis when possible with hypertonic mannitol.
- Evaluate renal status and begin haemodialysis if indicated. [I.L.O]
- Rapid absorption is an indication that emesis or lavage is effective only in the first few hours. Cathartics and charcoal are generally not effective.
- Correct acidosis, fluid/electrolyte balance and respiratory depression in the usual manner. Systemic acidosis (below 7.2) can be treated with intravenous sodium bicarbonate solution.
- Ethanol therapy prolongs the half-life of ethylene glycol and reduces the formation of toxic metabolites.
- Pyridoxine and thiamine are cofactors for ethylene glycol metabolism and should be given (50 to 100 mg respectively) intramuscularly, four times per day for 2 days.
- Magnesium is also a cofactor and should be replenished. The status of 4-methylpyrazole, in the treatment regime, is still uncertain. For clearance of the material and its metabolites, haemodialysis is much superior to peritoneal dialysis.

[Ellenhorn and Barceloux: Medical Toxicology] It has been suggested that there is a need for establishing a new biological exposure limit before a workshift that is clearly below 100 mmol ethoxy-acetic acids per mole creatinine in morning urine of people occupationally exposed to ethylene glycol ethers. This arises from the finding that an increase in urinary stones may be associated with such exposures.

Laitinen J., et al: Occupational & Environmental Medicine 1996; 53, 595-600.

Section 5 - FIRE FIGHTING MEASURES

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EXTINGUISHING MEDIA

■ The product contains a substantial proportion of water, therefore there are no restrictions on the type of extinguishing media which may be used. Choice of extinguishing media should take into account surrounding areas.
Though the material is non-combustible, evaporation of water from the mixture, caused by the heat of nearby fire, may produce floating layers of combustible substances.
In such an event consider:

- foam.
- dry chemical powder.
- carbon dioxide.

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

■

- Combustible.
- Slight fire hazard when exposed to heat or flame.
- Acids may react with metals to produce hydrogen, a highly flammable and explosive gas.
- Heating may cause expansion or decomposition leading to violent rupture of containers.
- May emit acrid smoke and corrosive fumes.

Combustion products include: carbon dioxide (CO_2), other pyrolysis products typical of burning organic material.
May emit poisonous fumes.
May emit corrosive fumes.

FIRE INCOMPATIBILITY

■

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

HAZCHEM

None

Personal Protective Equipment

Gas tight chemical resistant suit.

Section 6 - ACCIDENTAL RELEASE MEASURES

EMERGENCY PROCEDURES

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 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: ester and ethers
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
sorbent clay - particulate	2	shovel	shovel	R,I, P
wood fiber - particulate	3	shovel	shovel	R, W, P, DGC
wood fiber - pillow	3	throw	pitchfork	R, P, DGC, RT
treated wood fiber - pillow	3	throw	pitchfork	DGC, RT
LAND SPILL - MEDIUM				
cross-linked polymer - particulate	1	blower	skiploader	R,W, SS
cross-linked polymer -	2	throw	skiploader	R, DGC, RT

pillow				
sorbent clay - particulate	3	blower	skiploader	R, I, P
polypropylene - particulate	3	blower	skiploader	W, SS, DGC
expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC
wood fiber - particulate	4	blower	skiploader	R, W, P, DGC

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.

- Absorb or contain isothiazolinone liquid spills with sand, earth, inert material or vermiculite.
- The absorbent (and surface soil to a depth sufficient to remove all of the biocide) should be shovelled into a drum and treated with an 11% solution of sodium metabisulfite (Na2S2O5) or sodium bisulfite (NaHSO3), or 12% sodium sulfite (Na2SO3) and 8% hydrochloric acid (HCl).
- Glutathione has also been used to inactivate the isothiazolinones.
- Use 20 volumes of decontaminating solution for each volume of biocide, and let containers stand for at least 30 minutes to deactivate microbicide before disposal.
- If contamination of drains or waterways occurs, advise emergency services.
- After clean up operations, decontaminate and launder all protective clothing
- and equipment before storing and re-using.

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

Section 7 - HANDLING AND STORAGE

PROCEDURE FOR HANDLING

■

- DO NOT allow clothing wet with material to stay in contact with skin
- 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 storing and handling recommendations.
- Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.

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

STORAGE INCOMPATIBILITY

■

- Avoid reaction with oxidising agents

STORAGE REQUIREMENTS

■

- Store in original containers.
- Keep containers securely sealed.
- No smoking, naked lights or ignition sources.

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- 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 storing and handling recommendations.

SAFE STORAGE WITH OTHER CLASSIFIED CHEMICALS

\+ X + X X +

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

Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

EXPOSURE CONTROLS

Source	Material	TWA ppm	TWA mg/m³	STEL ppm	STEL mg/m³	Peak ppm	Peak mg/m³	TWA F/CC	Notes
Australia Exposure Standards	ethylene glycol monobutyl ether (2-Butoxyethanol)	20	96.9	50	242				Sk
Australia Exposure Standards	phosphoric acid (Phosphoric acid)		1		3				

The following materials had no OELs on our records

- gum arabic: CAS:9000-01-5
- 5-chloro-2-methyl-4-isothiazolin-3-one: CAS:26172-55-4
- water: CAS:7732-18-5

EMERGENCY EXPOSURE LIMITS

Material	Revised IDLH Value (mg/m3)	Revised IDLH Value (ppm)
ethylene glycol monobutyl ether		700 [Unch]
phosphoric acid	1,000	

MATERIAL DATA

300 MULTI FOUNT:
■ CEL TWA: 0.1 mg/m3; STEL 0.3 mg/m3 total isothiazolinones (Rohm and Haas).
For ethylene glycol monobutyl ether (2-butoxyethanol)
Odour Threshold Value: 0.10 ppm (detection), 0.35 ppm (recognition)
Although rats appear to be more susceptible than other animals anaemia is not uncommon amongst humans following exposure. The TLV reflects the need to maintain exposures below levels found to cause blood changes in experimental animals. It is concluded that this limit will reduce the significant risk of irritation, haematologic effects and other systemic effects observed in humans and animals exposed to higher vapour concentrations. The toxic effects typical of some other glycol ethers (pancytopenia, testis atrophy and teratogenic effects) are not found with this substance.
Odour Safety Factor (OSF)
OSF=2E2 (2-BUTOXYETHANOL).
The saturated vapour concentration of phosphoric acid exceeds the TLV. The TLV-TWA is based by analogy from comparable experience and data for sulfuric acid. Exposure at or below this limit is thought to prevent throat irritation amongst unacclimatised workers.
Fumes of phosphorus pentoxide at concentrations between 0.8 and 5.4 mg/m3 were reported to be noticeable but not uncomfortable whilst concentrations between 3.6 and 11.3 mg/m3 produced coughing in unacclimatised workers but were tolerable. Concentrations of 100 mg/m3 were unbearable except in inured workers.
ETHYLENE GLYCOL MONOBUTYL ETHER:
■ For ethylene glycol monobutyl ether (2-butoxyethanol)
Odour Threshold Value: 0.10 ppm (detection), 0.35 ppm (recognition)
Although rats appear to be more susceptible than other animals anaemia is not uncommon amongst humans following exposure. The TLV reflects the need to maintain exposures below levels found to cause blood changes in experimental animals. It is concluded that this limit will reduce the significant risk of irritation, haematologic effects and other systemic effects observed in humans and animals exposed to higher vapour concentrations. The toxic effects typical of some other glycol ethers (pancytopenia, testis atrophy and teratogenic effects) are not found with this substance.
Odour Safety Factor (OSF)
OSF=2E2 (2-BUTOXYETHANOL).
Exposed individuals are reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.
Odour Safety Factor (OSF) is determined to fall into either Class A or B.
The Odour Safety Factor (OSF) is defined as:
OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm

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Classification into classes follows:

Class	OSF	Description
A	550	Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by working activities
B	26-550	As "A" for 50-90% of persons being distracted
C	1-26	As "A" for less than 50% of persons being distracted
D	0.18-1	10-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached
E	<0.18	As "D" for less than 10% of persons aware of being tested

.

GUM ARABIC:

■ 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.

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.

PHOSPHORIC ACID:

■ The saturated vapour concentration of phosphoric acid exceeds the TLV. The TLV-TWA is based by analogy from comparable experience and data for sulfuric acid. Exposure at or below this limit is thought to prevent throat irritation amongst unacclimatised workers.

Fumes of phosphorus pentoxide at concentrations between 0.8 and 5.4 mg/m3 were reported to be noticeable but not uncomfortable whilst concentrations between 3.6 and 11.3 mg/m3 produced coughing in unacclimatised workers but were tolerable. Concentrations of 100 mg/m3 were unbearable except in inured workers.

5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE:

■ CEL TWA: 0.1 mg/m3; STEL 0.3 mg/m3 total isothiazolinones (Rohm and Haas).

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WATER:

■ No exposure limits set by NOHSC or ACGIH.

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]

HANDS/FEET

■

- Wear chemical protective gloves, eg. PVC.
- Wear safety footwear or safety gumboots, eg. Rubber

NOTE:

- The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.
- Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.

Suitability and durability of glove type is dependent on usage. Factors such as:

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

are important in the selection of gloves.

- Butyl rubber gloves
- Nitrile rubber gloves

OTHER

■

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

RESPIRATOR

■ 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.

Breathing Zone Level ppm (volume)	Maximum Protection Factor	Half-face Respirator	Full-Face Respirator
1000	10	ABK-AUS P	-
1000	50	-	ABK-AUS P
5000	50	Airline *	-
5000	100	-	ABK-2 P
10000	100	-	ABK-3 P
	100+		Airline**

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

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 Occupational Health and Safety Advisor.

ENGINEERING CONTROLS

■ 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. 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	0.5-1 m/s (100-200 f/min.)

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acid fumes, pickling (released at low velocity into zone of active generation)	
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.

Section 9 - PHYSICAL AND CHEMICAL PROPERTIES

APPEARANCE

Light green liquid with a characteristic odour; miscible with water.

PHYSICAL PROPERTIES

Liquid.
Mixes with water.

Molecular Weight: Not Available	Boiling Range (°C): 100	Melting Range (°C): Not Available
Specific Gravity (water=1): 1.05	Solubility in water (g/L): Miscible	pH (as supplied): 4.8-4.9
pH (1% solution): Not Available	Vapour Pressure (kPa): Error	Volatile Component (%vol): 70
Evaporation Rate: Fast	Relative Vapour Density (air=1): Not Available	Flash Point (°C): >61
Lower Explosive Limit (%): Not Available	Upper Explosive Limit (%): Not Available	Autoignition Temp (°C): Not Available
Decomposition Temp (°C): Not Available	State: Liquid	Viscosity: Not Available

Material	Value
log Kow	0.76-0.83

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.

Section 11 - TOXICOLOGICAL INFORMATION

POTENTIAL HEALTH EFFECTS

ACUTE HEALTH EFFECTS

SWALLOWED

■ Accidental ingestion of the material may be damaging to the health of the individual.

EYE

■ This material can cause eye irritation and damage in some persons.

SKIN

■ The material may accentuate any pre-existing dermatitis condition.
Open cuts, abraded or irritated skin should not be exposed to this material.
Entry into the blood-stream, through, for example, cuts, abrasions 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.

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There is some evidence to suggest that this material can cause inflammation of the skin on contact in some persons.

INHALED

■ Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.
There is some evidence to suggest that the material can cause respiratory irritation in some persons. The body's response to such irritation can cause further lung damage.

CHRONIC HEALTH EFFECTS

■ Skin contact with the material is more likely to cause a sensitisation reaction in some persons compared to the general population.
Substance accumulation, in the human body, may occur and may cause some concern following repeated or long-term occupational exposure.
There is some evidence that inhaling this product is more likely to cause a sensitisation reaction in some persons compared to the general population.
There is some evidence from animal testing that exposure to this material may result in toxic effects to the unborn baby.
Based on experience with similar materials, there is a possibility that exposure to the material may reduce fertility in humans at levels which do not cause other toxic effects.
The isothiazolinones are known contact sensitisers. Sensitisation is more likely with the chlorinated species as opposed to the non-chlorinated species. Risk of sensitization depends on how contact occurs – it is higher when the skin has been damaged. Skin specialist studies have shown sensitisation has occurred with concentrations of 0.02% or less, and allergic reactions can occur in sensitized people with even lower concentrations. There is immune cross reaction between chlorinated isothiazolinones, but not between the non-chlorinated species or between non-chlorinated and chlorinated species. More experience is needed before conclusion of the safety of non-chlorinated species can be made.
There are conflicting reports in the literature, but isothiazolinones have been reported to cause mutations in certain bacteria. This effect has not been been shown to occur in mammal cells. Animal testing showed no reproductive or tumour-inducing effects.
There has been concern that this material can cause cancer or mutations, but there is not enough data to make an assessment.
Ethylene glycol esters and their ethers cause wasting of the testicles, reproductive changes, infertility and changes to kidney function. Shorter chain compounds are more dangerous. They are also associated with the formation of stones in the urine.

TOXICITY AND IRRITATION

■ unless otherwise specified data extracted from RTECS - Register of Toxic Effects of Chemical Substances.
■ Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.
Allergic reactions involving the respiratory tract are usually due to interactions between IgE antibodies and allergens and occur rapidly. Allergic potential of the allergen and period of exposure often determine the severity of symptoms. Some people may be genetically more prone than others, and exposure to other irritants may aggravate symptoms. Allergy causing activity is due to interactions with proteins.
Attention should be paid to atopic diathesis, characterised by increased susceptibility to nasal inflammation, asthma and eczema.
Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure.

For ethylene glycol monoalkyl ethers and their acetates (EGMAEs):

Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates
EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers.
Acute Toxicity: Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m3) for EGHE, LC50 > 400ppm (2620 mg/m3) for EGBEA to LC50 > 2132 ppm (9061 mg/m3) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitisers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE in vitro than those of rats.
Repeat dose toxicity: The fact that the NOAEL for repeated dose toxicity of EGBE is less than that of EGPE is consistent with red blood cells being more sensitive to EGBE than EGPE. Blood from mice, rats, hamsters, rabbits and baboons were sensitive to the effects of BAA in vitro and displayed similar responses, which included erythrocyte swelling (increased haematocrit and mean corpuscular hemoglobin), followed by hemolysis. Blood from humans, pigs, dogs, cats, and guinea pigs was less sensitive to haemolysis by BAA in vitro.
Mutagenicity: In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in S. typhimurium strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. In vitro cytogenicity and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and in vivo micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic.
Carcinogenicity: In a 2-year inhalation chronic toxicity and carcinogenicity study with EGBE in rats and mice a significant increase in the incidence of liver haemangiosarcomas was seen in male mice and forestomach tumours in female mice. It was decided that

300 MULTI FOUNT

Hazard Alert Code: MODERATE

based on the mode of action data available, there was no significant hazard for human carcinogenicity
Reproductive and developmental toxicity. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes).
Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m3 and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m3), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m3), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m3) indicate that the members of the category are not teratogenic.
The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m3 (rabbit-EGPE), 100 ppm or 425 mg/m3 (rat-EGPE), 50 ppm or 241 mg/m3 (rat EGBE) and 100 ppm or 483 mg/m3 (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m3 (rat and rabbit-EGHE).

ETHYLENE GLYCOL MONOBUTYL ETHER:

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

TOXICITY	IRRITATION
Oral (rat) LD50: 470 mg/kg	Skin (rabbit): 500 mg, open; Mild
Dermal (rabbit) LD50: 220 mg/kg	Eye (rabbit): 100 mg/24h-Moderate
Inhalation (human) TCLo: 100 ppm	Eye (rabbit): 100 mg SEVERE
Inhalation (human) TCLo: 195 ppm/8h	* [Union Carbide]
Inhalation (Rat) LC50: 450 ppm *	

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

The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.

For ethylene glycol:

Ethylene glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol.

dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO_2, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO_2, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested.

Respiratory Effects. Respiratory system involvement occurs 12-24 hours after ingestion of sufficient amounts of ethylene glycol and is considered to be part of a second stage in ethylene glycol poisoning	The symptoms include hyperventilation, shallow rapid breathing, and generalized pulmonary edema with calcium oxalate crystals occasionally present in the lung parenchyma. Respiratory system involvement appears to be dose-dependent and occurs concomitantly with cardiovascular changes. Pulmonary infiltrates and other changes compatible with adult respiratory distress syndrome (ARDS) may characterise the second stage of ethylene glycol poisoning Pulmonary oedema can be secondary to cardiac failure, ARDS, or aspiration of gastric contents. Symptoms related to acidosis such as hyperpnea and tachypnea are frequently observed; however, major respiratory morbidities such as pulmonary edema and bronchopneumonia are relatively rare and usually only observed with extreme poisoning (e.g., in only 5 of 36 severely poisoned cases).
Cardiovascular Effects. Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12- 24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop	and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol . As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of ethylene glycol.

Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases .Therefore, it appears that acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown.

Gastrointestinal Effects. Nausea, vomiting with or without blood, pyrosis, and abdominal cramping and pain are common early effects of acute ethylene glycol ingestion. Acute effects of ethylene glycol ingestion in one patient included intermittent diarrhea and abdominal pain, which were attributed to mild colonic ischaemia; severe abdominal pain secondary to colonic stricture and perforation developed 3 months after ingestion, and histology of the resected colon showed birefringent crystals highly suggestive of oxalate deposition.

Musculoskeletal Effects. Reported musculoskeletal effects in cases of acute ethylene glycol poisoning have included diffuse muscle tenderness and myalgias associated with elevated serum creatinine phosphokinase levels, and myoclonic jerks and tetanic contractions associated with hypocalcaemia.

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Hazard Alert Code: MODERATE

Hepatic Effects. Central hydropic or fatty degeneration, parenchymal necrosis, and calcium oxalate crystals in the liver have been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol.

Renal Effects. Adverse renal effects after ethylene glycol ingestion in humans can be observed during the third stage of ethylene glycol toxicity 24-72 hours after acute exposure. The hallmark of renal toxicity is the presence of birefringent calcium oxalate monohydrate crystals deposited in renal tubules and their presence in urine after ingestion of relatively high amounts of ethylene glycol. Other signs of nephrotoxicity can include tubular cell degeneration and necrosis and tubular interstitial inflammation. If untreated, the degree of renal damage caused by high doses of ethylene glycol progresses and leads to haematuria, proteinuria, decreased renal function, oliguria, anuria , and ultimately renal failure. These changes in the kidney are linked to acute tubular necrosis but normal or near normal renal function can return with adequate supportive therapy.

Metabolic Effects. One of the major adverse effects following acute oral exposure of humans to ethylene glycol involves metabolic changes. These changes occur as early as 12 hours after ethylene glycol exposure. Ethylene glycol intoxication is accompanied by metabolic acidosis which is manifested by decreased pH and bicarbonate content of serum and other bodily fluids caused by accumulation of excess glycolic acid. Other characteristic metabolic effects of ethylene glycol poisoning are increased serum anion gap, increased osmolal gap, and hypocalcaemia. Serum anion gap is calculated from concentrations of sodium, chloride, and bicarbonate, is normally 12-16 mM, and is typically elevated after ethylene glycol ingestion due to increases in unmeasured metabolite anions (mainly glycolate).

Neurological Effects: Adverse neurological reactions are among the first symptoms to appear in humans after ethylene glycol ingestion. These early neurotoxic effects are also the only symptoms attributed to unmetabolised ethylene glycol. Together with metabolic changes, they occur during the period of 30 minutes to 12 hours after exposure and are considered to be part of the first stage in ethylene glycol intoxication. In cases of acute intoxication, in which a large amount of ethylene glycol is ingested over a very short time period, there is a progression of neurological manifestations which, if not treated, may lead to generalized seizures and coma. Ataxia, slurred speech, confusion, and somnolence are common during the initial phase of ethylene glycol intoxication as are irritation, restlessness, and disorientation. Cerebral edema and crystalline deposits of calcium oxalate in the walls of small blood vessels in the brain were found at autopsy in people who died after acute ethylene glycol ingestion.

Effects on cranial nerves appear late (generally 5-20 days post-ingestion), are relatively rare, and according to some investigators constitute a fourth, late cerebral phase in ethylene glycol intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar nerves and are reversible over many months.

Reproductive Effects: Reproductive function after intermediate-duration oral exposure to ethylene glycol has been tested in three multi-generation studies (one in rats and two in mice) and several shorter studies (15-20 days in rats and mice). In these studies, effects on fertility, foetal viability, and male reproductive organs were observed in mice, while the only effect in rats was an increase in gestational duration.

Developmental Effects: The developmental toxicity of ethylene glycol has been assessed in several acute-duration studies using mice, rats, and rabbits. Available studies indicate that malformations, especially skeletal malformations occur in both mice and rats exposed during gestation; mice are apparently more sensitive to the developmental effects of ethylene glycol. Other evidence of embyrotoxicity in laboratory animals exposed to ethylene glycol exposure includes reduction in foetal body weight.

Cancer: No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol.

Genotoxic Effects: Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available in vivo and in vitro laboratory studies provide consistently negative genotoxicity results for ethylene glycol.

For ethylene glycol monoalkyl ethers and their acetates (EGMAEs):

Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates

EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers.

Acute Toxicity: Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m3) for EGHE, LC50 > 400ppm (2620 mg/m3) for EGBEA to LC50 > 2132 ppm (9061 mg/m3) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitisers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE in vitro than those of rats.

Repeat dose toxicity: The fact that the NOAEL for repeated dose toxicity of EGBE is less than that of EGPE is consistent with red blood cells being more sensitive to EGBE than EGPE. Blood from mice, rats, hamsters, rabbits and baboons were sensitive to the effects of BAA in vitro and displayed similar responses, which included erythrocyte swelling (increased haematocrit and mean corpuscular hemoglobin), followed by hemolysis. Blood from humans, pigs, dogs, cats, and guinea pigs was less sensitive to haemolysis by BAA in vitro.

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Hazard Alert Code: MODERATE

Mutagenicity: In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in S. typhimurium strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. In vitro cytogenicity and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and in vivo micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic.

Carcinogenicity: In a 2-year inhalation chronic toxicity and carcinogenicity study with EGBE in rats and mice a significant increase in the incidence of liver haemangiosarcomas was seen in male mice and forestomach tumours in female mice. It was decided that based on the mode of action data available, there was no significant hazard for human carcinogenicity

Reproductive and developmental toxicity. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes).

Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on	EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m3 and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m3), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m3), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m3) indicate that the members of the category are not teratogenic.

The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m3 (rabbit-EGPE), 100 ppm or 425 mg/m3 (rat-EGPE), 50 ppm or 241 mg/m3 (rat EGBE) and 100 ppm or 483 mg/m3 (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m3 (rat and rabbit-EGHE).

Exposure of pregnant rats to ethylene glycol monobutyl ether (2-butoxyethanol) at 100 ppm or rabbits at 200 ppm during organogenesis resulted in maternal toxicity and embryotoxicity including a decreased number of viable implantations per litter. Slight foetoxicity in the form of poorly ossified or unossified skeletal elements was also apparent in rats. Teratogenic effects were not observed in other species.

At least one researcher has stated that the reproductive effects were less than that of other monoalkyl ethers of ethylene glycol.

Chronic exposure may cause anaemia, macrocytosis, abnormally large red cells and abnormal red cell fragility.

Exposure of male and female rats and mice for 14 weeks to 2 years produced a regenerative haemolytic anaemia and subsequent effects on the haemopoietic system in rats and mice. In addition, 2-butoxyethanol exposures caused increases in the incidence of neoplasms and nonneoplastic lesions (1). The occurrence of the anaemia was concentration-dependent and more pronounced in rats and females. In this study it was proposed that 2-butoxyethanol at concentrations of 500 ppm and greater produced an acute disseminated thrombosis and bone infarction in male and female rats as a result of severe acute haemolysis and reduced deformability of erythrocytes or through anoxic damage to endothelial cells that compromise blood flow. In two-year studies, 2-butoxyethanol continued to affect circulating erythroid mass, inducing a responsive anaemia. Rats showed a marginal increase in the incidence of benign or malignant pheochromocytomas (combined) of the adrenal gland. In mice, 2-butoxyethanol exposure resulted in a concentration dependent increase in the incidence of squamous cell papilloma or carcinoma of the forestomach. It was hypothesised that exposure-induced irritation produced inflammatory and hyperplastic effects in the forestomach and that the neoplasia were associated with a continuation of the injury/ degeneration process. Exposure also produced a concentration -dependent increase in the incidence of haemangiosarcoma of the liver of male mice and hepatocellular carcinoma.

1: NTP Toxicology Program Technical report Series 484, March 2000.

NOTE: Changes in kidney, liver, spleen and lungs are observed in animals	exposed to high concentrations of this substance by all routes.

GUM ARABIC:

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

Oral (rabbit) LD50: 8000 mg/kg	Eye (rabbit): 36 mg/5h SEVERE

■ Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

Allergic reactions involving the respiratory tract are usually due to interactions between IgE antibodies and allergens and occur rapidly. Allergic potential of the allergen and period of exposure often determine the severity of symptoms. Some people may be genetically more prone than others, and exposure to other irritants may aggravate symptoms. Allergy causing activity is due to interactions with proteins.

300 MULTI FOUNT

Hazard Alert Code: MODERATE

Attention should be paid to atopic diathesis, characterised by increased susceptibility to nasal inflammation, asthma and eczema.

Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure.

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

PHOSPHORIC ACID:

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

Unreported (human) LDLo: 220 mg/kg	Skin (rabbit):595 mg/24h - SEVERE
Oral (rat) LD50: 1530 mg/kg	Eye (rabbit): 119 mg - SEVERE
Oral (rat) LD50: 3500 mg/kg*	[Monsanto]*
Dermal (rabbit) LD50: 1260 mg/kg*	
Inhalation (Rat) LC50: 25.5 mg/m³/4h	
Inhalation (Mouse) LC50: 25.5 mg/m³/4h	

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

The material may cause severe skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin. Repeated exposures may produce severe ulceration.

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

phosphoric acid (85%)

5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE:

■ Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.
The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.
Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.
NOTE: Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.
No significant acute toxicological data identified in literature search.
Considered to be the major sensitiser in Kathon CG (1)
(1). Bruze etal - Contact Dermatitis 20: 219-39, 1989

WATER:

■ No significant acute toxicological data identified in literature search.

CARCINOGEN

2-Butoxyethanol	International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs	Group 3

SKIN

ethylene glycol monobutyl ether	Australia Exposure Standards - Skin	Notes Sk

Section 12 - ECOLOGICAL INFORMATION

300 MULTI FOUNT

Hazard Alert Code: MODERATE

Refer to data for ingredients, which follows:
300 MULTI FOUNT:
5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE:
■ The isothiazolinones are very toxic to marine organisms (fish, Daphnia magna and algae)
The high water solubility and low log Kow values of several chlorinated and non-chlorinated indicate a low potential for bioaccumulation.
Studies of 5-chloro-2-methyl-4-isothiazolin-3-one (CMI) in bluegill sunfish (Lepornis machrochirus) show BCF values of 102, 114 and 67 at nominal concentrations of 0.02, 0.12 and 0.8 mg/l. The BCF for 2-methyl-4-isothiazolin-3-one (MI) was determined at 2.3 at a nominal concentration of 0.12 mg/l
Primary biodegradation of MI and CMI occurred with half-lives of less than 24 hours in aerobic and anoxic sediments, and within a period of less than one week the parent compounds were depleted to very low levels that could not be clearly distinguished from analytical artifacts. The ultimate aerobic biodegradability of both MI and CMI attained levels of > 55% within 29 days. Furthermore, the proposed metabolites of MI and CMI are considered to have a low aquatic toxicity on the basis of QSAR estimates and the measured toxicity of the structurally related N-(n-octyl) malonamic acid.
PHOSPHORIC ACID:
5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE:
■ Prevent, by any means available, spillage from entering drains or water courses.
300 MULTI FOUNT:
5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE:
■ Very toxic to aquatic organisms.
300 MULTI FOUNT:
5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE:
PHOSPHORIC ACID:
■ 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.
300 MULTI FOUNT:
5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE:
PHOSPHORIC ACID:
GUM ARABIC:
ETHYLENE GLYCOL MONOBUTYL ETHER:
■ DO NOT discharge into sewer or waterways.
300 MULTI FOUNT:
PHOSPHORIC ACID:
■ On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and or observed environmental fate and behaviour, the material may present a danger, immediate or long-term and /or delayed, to the structure and/ or functioning of natural ecosystems.
300 MULTI FOUNT:
ETHYLENE GLYCOL MONOBUTYL ETHER:
■ For ethylene glycol monoalkyl ethers and their acetates:
Members of this category include ethylene glycol propyl ether (EGPE), ethylene glycol butyl ether (EGBE)r and ethylene glycol hexyl ether (EGHE)
Environmental fate:
The ethers, like other simple glycol ethers possess no functional groups that are readily subject to hydrolysis in the presence of waters. The acetates possess an ester group that hydrolyses in neutral ambient water under abiotic conditions.
Level III fugacity modeling indicates that category members, when released to air and water, will partition predominately to water and, to a lesser extent, to air and soil. Estimates of soil and sediment partition coefficients (Kocs ranging from 1- 10) suggest that category members would exhibit high soil mobility. Estimated bioconcentration factors (log BCF) range from 0.463 to 0.732. Biodegradation studies indicate that all category members are readily biodegradable. The physical chemistry and environmental fate properties indicate that category members will not persist or bioconcentrate in the environment.
Ecotoxicity:
Glycol ether acetates do not hydrolyse rapidly into their corresponding glycol ethers in water under environmental conditions. The LC50 or EC50 values for EGHE are lower than those for EGPE and EGBE (which have shorter chain lengths and lower log Kow values). Overall, the LC50 values for the glycol ethers in aquatic species range from 94 to > 5000 mg/L. For EGHE, the 96-hour LC50 for Brachydanio rerio (zebra fish) is between 94 and mg/L, the 48-hour EC50 for Daphnia magna was 145 mg/L and the 72-hour EC50 values for biomass and growth rate of algae (Scenedesmus subspicatus) were 98 and 198 mg/L, respectively. LC50/EC50 values for EGPE and EGBE in aquatic species are 835 mg/l or greater.
Aquatic toxicity data for EGBEA show a 96-hour LC50 of 28.3 mg/L for rainbow trout (Oncorhynchus mykiss), a 48-hour LC50 of 37-143 mg/L for Daphnia magna, a 72-hour EC50 of greater than 500 mg/L for biomass or growth rate of algae (Scenedesmus subspicatus and Pseudokirchneriella subcapitata, respectively), and a 7-day EC10 of 30.4 mg/L and a NOEC of 16.4 mg/L for reproduction in Ceriodaphnia dubia.
300 MULTI FOUNT:
ETHYLENE GLYCOL MONOBUTYL ETHER:
■ For glycol ethers:
Environmental fate:
Ether groups are generally stable to hydrolysis in water under neutral conditions and ambient temperatures. OECD guideline studies indicate ready biodegradability for several glycol ethers although higher molecular weight species seem to biodegrade at a slower rate. No glycol ethers that have been tested demonstrate marked resistance to biodegradative processes. Upon release to the atmosphere by evaporation, high boiling glycol ethers are estimated to undergo photodegradation (atmospheric half lives = 2.4-2.5 hr). When released to water, glycol ethers undergo biodegradation (typically 47-92% after 8-21 days) and have a low potential for bioaccumulation (log Kow ranges from -1.73 to +0.51).
Ecotoxicity:
Aquatic toxicity data indicate that the tri- and tetra ethylene glycol ethers are "practically non-toxic" to aquatic species. No major differences are observed in the order of toxicity going from the methyl- to the butyl ethers.
Glycols exert a high oxygen demand for decomposition and once released to th environments cause the death of aquatic organisms if dissolved oxygen is depleted.
300 MULTI FOUNT:

300 MULTI FOUNT

Hazard Alert Code: MODERATE

ETHYLENE GLYCOL MONOBUTYL ETHER:

■ Fish LC50 (96hr.) (mg/l):	1490
■ BCF<100:	0.4
■ log Kow (Prager 1995):	0.83
■ log Kow (Sangster 1997):	0.8
■ Half- life Soil - High (hours):	672
■ Half- life Soil - Low (hours):	168
■ Half- life Air - High (hours):	32.8
■ Half- life Air - Low (hours):	3.28
■ Half- life Surface water - High (hours):	672
■ Half- life Surface water - Low (hours):	168
■ Half- life Ground water - High (hours):	1344
■ Half- life Ground water - Low (hours):	336
■ Aqueous biodegradation - Aerobic - High (hours):	672
■ Aqueous biodegradation - Aerobic - Low (hours):	168
■ Aqueous biodegradation - Anaerobic - High (hours):	2688
■ Aqueous biodegradation - Anaerobic - Low (hours):	672
■ Photooxidation half- life air - High (hours):	32.8
■ Photooxidation half- life air - Low (hours):	3.28
■ Fish LC50 (96hr.) (mg/l):	1250- 1650
■ Daphnia magna EC50 (48hr.) (mg/l):	600- 1000

log Kow: 0.76-0.83
Koc: 67
Half-life (hr) air: 17
Henry's atm m^3 /mol: 2.08E-08
BOD 5 if unstated: 0.71
COD: 2.2
Log BCF: 0.4
Fish LC50 (24 h): 983-1650 mg/L
Fish LC50 (96 h): fathead minnow 1700 mg/L **
Invertebrate toxicity:
cell mult. inhib.91-900mg/L
(Daphnia) 48h LC50: >1000 mg/L **
Bioaccumulation: not sig
Effects on algae and plankton: cell mult. inhib.35-900mg/L
Degradation Biological: rapid
processes Abiotic: no hydrol&photol,RxnOH* ** [Union Carbide]
GUM ARABIC:
■ Sugar-based compounds (saccharides), including polysaccharides are generally easily decomposed by biodegradation. Not all polysaccharides decompose with equal rapidity, and polysaccharides are also synthesised by microorganisms during, for example, the compost maturation phases. Water-insoluble species such as cellulose take longer to decompose and those with a significant degree of branching also take longer.
PHOSPHORIC ACID:

■ Fish LC50 (96hr.) (mg/l):	138

■ May cause long-term adverse effects in the aquatic environment.
■ Ecotoxicity:
The tolerance of water organisms towards pH margin and variation is diverse. Recommended pH values for test species listed in OECD guidelines are between 6.0 and almost 9. Acute testing with fish showed 96h-LC50 at about pH 3.5.
■ 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.
5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE:
Octanol/water Coefficient = 0.401 (log P)
Biodegradation (aquatic metabolism)
half life t1/2 anerobic = 4.8 hours
half life t1/2 aerobic = 17.3 hours
as mixed isothiazolinones
Rainbow trout LC50(96 hr) = 0.19 mg/L.
Bluegill Sunfish LC50(96hr) = 0.28 mg/L.
Daphnia EC50(48hr) = 0.16 mg/L.
Algal Selenastrum EC50: 0.018 mg/L.
WATER:

300 MULTI FOUNT

Hazard Alert Code: MODERATE

Ecotoxicity

Ingredient	Persistence: Water/Soil	Persistence: Air	Bioaccumulation	Mobility
300 Multi Fount		No data		
ethylene glycol monobutyl ether	LOW	LOW	LOW	HIGH
gum arabic		No data		
phosphoric acid	HIGH	No data	LOW	HIGH
5-chloro-2-methyl-4-isothiazolin-3-one	HIGH	No data	LOW	HIGH
water	LOW	No data	LOW	HIGH

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.

Section 14 - TRANSPORTATION INFORMATION

Labels Required: COMBUSTIBLE LIQUID, regulated under AS1940 for Bulk Storage purposes only.
HAZCHEM: None (ADG7)
NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS: ADG7, UN, IATA, IMDG

Section 15 - REGULATORY INFORMATION

POISONS SCHEDULE
None

300 MULTI FOUNT

Hazard Alert Code: MODERATE

REGULATIONS

Regulations for ingredients

ethylene glycol monobutyl ether (CAS: 111-76-2) is found on the following regulatory lists;

"Australia Exposure Standards","Australia Hazardous Substances","Australia High Volume Industrial Chemical List (HVICL)","Australia Inventory of Chemical Substances (AICS)","IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances","International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs","OECD Representative List of High Production Volume (HPV) Chemicals"

gum arabic (CAS: 9000-01-5) is found on the following regulatory lists;

"Australia Inventory of Chemical Substances (AICS)","Australia Therapeutic Goods Administration (TGA) Substances that may be used as active ingredients in Listed medicines","CODEX General Standard for Food Additives (GSFA) - Additives Permitted for Use in Food in General, Unless Otherwise Specified, in Accordance with GMP"

phosphoric acid (CAS: 7664-38-2,16271-20-8) is found on the following regulatory lists;

"Australia Exposure Standards","Australia Hazardous Substances","Australia High Volume Industrial Chemical List (HVICL)","Australia Inventory of Chemical Substances (AICS)","Australia National Pollutant Inventory","Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix E (Part 2)","Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix F (Part 3)","Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Schedule 5","Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Schedule 6","GESAMP/EHS Composite List of Hazard Profiles - Hazard evaluation of substances transported by ships","IMO IBC Code Chapter 17: Summary of minimum requirements","IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk","International Council of Chemical Associations (ICCA) - High Production Volume List","OECD Representative List of High Production Volume (HPV) Chemicals"

5-chloro-2-methyl-4-isothiazolin-3-one (CAS: 26172-55-4) is found on the following regulatory lists;

"Australia Inventory of Chemical Substances (AICS)","OECD Representative List of High Production Volume (HPV) Chemicals"

water (CAS: 7732-18-5) is found on the following regulatory lists;

"Australia Inventory of Chemical Substances (AICS)","GESAMP/EHS Composite List of Hazard Profiles - Hazard evaluation of substances transported by ships","IMO IBC Code Chapter 18: List of products to which the Code does not apply","OECD Representative List of High Production Volume (HPV) Chemicals"

No data for 300 Multi Fount (CW: 22-0062)

Section 16 - OTHER INFORMATION

Denmark Advisory list for selfclassification of dangerous substances

Substance CAS Suggested codes 5- chloro- 2- methyl- 4- isothiazolin- 26172- 55- R43 3- one 4

Ingredients with multiple CAS Nos

Ingredient Name	CAS
phosphoric acid	7664-38-2, 16271-20-8

REPRODUCTIVE HEALTH GUIDELINES

Ingredient	ORG	UF	Endpoint	CR	Adeq TLV
ethylene glycol monobutyl ether	3.6 mg/m3	100	D	NA	-

■ These exposure guidelines have been derived from a screening level of risk assessment and should not be construed as unequivocally safe limits. ORGS represent an 8-hour time-weighted average unless specified otherwise.
CR = Cancer Risk/10000; UF = Uncertainty factor:
TLV believed to be adequate to protect reproductive health:
LOD: Limit of detection
Toxic endpoints have also been identified as:
D = Developmental; R = Reproductive; TC = Transplacental carcinogen
Jankovic J., Drake F.: A Screening Method for Occupational Reproductive
American Industrial Hygiene Association Journal 57: 641-649 (1996).

■ Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.
A list of reference resources used to assist the committee may be found at:
www.chemwatch.net/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.