



ALL PURPOSE CHEMICAL RELEASE AGENT FOR CONCRETE FORMWORK

Chemwatch Hazard Alert Code: 2

Chemwatch: 81-8831 Version No: 2.1.1.1

Safety Data Sheet according to HSNO Regulations

Issue Date: 22/06/2017 Print Date: 19/08/2017 L.GHS.NZL.EN

SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier		
Product name	Cemix APRA	
Synonyms	All purpose chemical release agent for concrete formwork, All purpose release agent, A.P.R.A	
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains diesel)	
Other means of identification	Not Available	
Relevant identified uses o	f the substance or mixture and uses advised against	
Relevant identified uses	Air entertaining agent for concrete mortar.	
• • • • • • • • • • • • • • • • • • • •	Details of the supplier of the safety data sheet	
Registered company name	Cemix (a part of Ardex NZ)	
Address	19 Alfred Street Onehunga Auckland 1061 New Zealand	
Telephone	+64 9 636 1000	
Fax	+64 9 636 0000	
Website	www.cemix.co.nz	
Email	Not Available	
Emergency telephone nun	Emergency telephone number	
Association / Organisation	Not Available	
Emergency telephone numbers	0800 ASK CEMIX	
Other emergency telephone	Not Available	

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

Considered a Hazardous Substance according to the criteria of the New Zealand Hazardous Substances New Organisms legislation. Classified as Dangerous Goods for transport purposes.

Classification [1]	Flammable Liquid Category 4, Skin Corrosion/Irritation Category 2, Respiratory Sensitizer Category 1, Skin Sensitizer Category 1, Carcinogenicity Category 2, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Aspiration Hazard Category 1, Acute Aquatic Hazard Category 2, Chronic Aquatic Hazard Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI
Determined by Chemwatch using GHS/HSNO criteria	3.1D, 6.1E (aspiration), 6.3A, 6.5A (respiratory), 6.5B (contact), 6.7B, 6.9 (narcotic), 9.1B, 9.1D

Label elements

Hazard pictogram(s)







SIGNAL WORD	

DANGER

Hazard statement(s)

H227	Combustible liquid
H315	Causes skin irritation.
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H317	May cause an allergic skin reaction.
H351	Suspected of causing cancer.
H336	May cause drowsiness or dizziness.
H304	May be fatal if swallowed and enters airways.

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H411	Toxic to aquatic life with long lasting effects.
Precautionary statement(s) Prevention
P201	Obtain special instructions before use.
P210	Keep away from heat/sparks/open flames/hot surfaces No smoking.
P261	Avoid breathing mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P281	Use personal protective equipment as required.
P285	In case of inadequate ventilation wear respiratory protection.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER or doctor/physician.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.
P308+P313	IF exposed or concerned: Get medical advice/attention.
P331	Do NOT induce vomiting.
P342+P311	If experiencing respiratory symptoms: Call a POISON CENTER or doctor/physician.
P362	Take off contaminated clothing and wash before reuse.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam for extinction.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P312	Call a POISON CENTER or doctor/physician if you feel unwell.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P391	Collect spillage.

Precautionary statement(s) Storage

P403+P235	Store in a well-ventilated place. Keep cool.
P405	Store locked up.

Precautionary statement(s) Disposal

P501 Dispose of contents/container in accordance with local regulations.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
68334-30-5	80-90	<u>diesel</u>
122-80-5	4-5	4'-aminoacetanilide
Not avail.	4-5	mineral oil

SECTION 4 FIRST AID MEASURES

NZ Poisons Centre 0800 POISON (0800 764 766) | NZ Emergency Services: 111

Description of first aid measures

Eye Contact	If this product comes in contact with eyes: • Wash out immediately with water. • If irritation continues, seek medical attention. • Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: Immediately remove all contaminated clothing, including footwear. Flush skin and hair with running water (and soap if available). Seek medical attention in event of irritation.
Inhalation	 If furnes, aerosols or combustion products are inhaled remove from contaminated area. Other measures are usually unnecessary.
Ingestion	 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.

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Indication of any immediate medical attention and special treatment needed

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.

Treat symptomatically

- Heavy and persistent skin contamination over many years may lead to dysplastic changes. Pre-existing skin disorders may be aggravated by exposure to this product.
- ▶ In general, emesis induction is unnecessary with high viscosity, low volatility products, i.e. most oils and greases
- High pressure accidental injection through the skin should be assessed for possible incision, irrigation and/or debridement.

NOTE: Injuries may not seem serious at first, but within a few hours tissue may become swollen, discoloured and extremely painful with extensive subcutaneous necrosis. Product may be forced through considerable distances along tissue planes.

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

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

Special hazards arising from the substrate or mixture

Eiro I	Incompatibility

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

Advice for firefighters	
Fire Fighting	 Alert Fire Brigade and tell them location and nature of hazard. Wear full body protective clothing with breathing apparatus. Prevent, by any means available, spillage from entering drains or water course. Use 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. ▶ Heating may cause expansion or decomposition leading to violent rupture of containers. ▶ On combustion, may emit toxic fumes of carbon monoxide (CO). ▶ May emit acrid smoke. ▶ Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO2) other pyrolysis products typical of burning organic material. CARE: Water in contact with hot liquid may cause foaming and a steam explosion with wide scattering of hot oil and possible severe burns. Foaming may cause overflow of containers and may result in possible fire.

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	 Environmental hazard - contain spillage. Clean up all spills immediately. Avoid breathing vapours and contact with skin and eyes. Control personal contact with the substance, by using protective equipment. Contain and absorb spill with sand, earth, inert material or vermiculite. Wipe up. Place in a suitable, labelled container for waste disposal.
Major Spills	Environmental hazard - contain spillage. 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 vermiculitie. 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.

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

▶ Avoid all personal contact, including inhalation. Wear protective clothing when risk of exposure occurs. Use in a well-ventilated area. Prevent concentration in hollows and sumps. DO NOT enter confined spaces until atmosphere has been checked. Avoid smoking, naked lights or ignition sources. Avoid contact with incompatible materials. Safe handling 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. Use good occupational work practice. Observe manufacturer's storage and handling recommendations contained within this SDS. Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions. Store in original containers. Keep containers securely sealed Store in a cool, drv. well-ventilated area. Other information ▶ Store away from incompatible materials and foodstuff containers. Protect containers against physical damage and check regularly for leaks.

Conditions for safe storage, including any incompatibilities

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Suitable container	 Metal can or drum Packaging as recommended by manufacturer. Check all containers are clearly labelled and free from leaks. 			
Storage incompatibility	CARE: Water in contact with heated material may cause foaming or a steam explosion with possible severe burns from wide scattering of hot material. Resultant overflow of containers may result in fire. Avoid reaction with oxidising agents			

▶ Observe manufacturer's storage and handling recommendations contained within this SDS

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
New Zealand Workplace Exposure Standards (WES)	mineral oil	Oil mist, mineral	5 mg/m3	10 mg/m3	Not Available	(om) - Sampled by a method that does not collect vapour.

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
diesel	Diesel fuels; (inlcudes diesel fuel No. 4 (68476-31-3), fuel oil No.2 (68476-30-2), fuel oil residual (68476-33-5)	300 mg/m3	3,300 mg/m3	20,000 mg/m3

Ingredient	Original IDLH	Revised IDLH
diesel	Not Available	Not Available
4'-aminoacetanilide	Not Available	Not Available
mineral oil	Not Available	Not Available

MATERIAL DATA

for fuels, diesel

TLV TWA: 15 ppm (vapour); 100 mg/m3 (inhalable fraction and vapour) (skin)

OEL TWA: 5 mg/m3 (stable aerosol) Exxon Mobil 2009

OEL TWA: 200 mg/m3 (vapour) Exxon Mobil 2009

for fuels, diesel, no. 2 [inhalable total hydrocarbon, vapour and aerosol] TLV TWA 100 mg/m3 (skin) $\,$

for kerosine (petroleum), hydrosulfurized

TLV TWA: 200 mg/m3 (skin)

Vapour concentrations above the recommended exposure levels are irritating to the eyes and respiratory tract, may cause headaches and dizziness, are anaesthetic and may have other central nervous system effects.

Diesel fuel is carcinogenic in animal tests and causes mutations in vitro. Repeated dermal exposure to high concentrations in test animals resulted in reduced litter size and litter weight, and increased foetal resorptions at maternally toxic doses. Dermal exposure to high concentrations resulted in severe skin irritation with weight loss and some mortality. Inhalation exposure to high concentrations resulted in respiratory tract irritation, lung changes/ infiltration/ accumulation, and reduction in lung function.

For diesel engine exhaus

WARNING: This is classified by IARC as Group 1: CARCINOGENIC TO HUMANS

Diesel exhaust fumes are carcinogenic in animal tests. Inhalation exposures to exhaust for 2 years in test animals resulted in lung tumours and lymphoma. Extract of particulate produced skin tumours in tests animals and caused mutations in-vitro.

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Odour threshold: 0.7 ppm Odour Safety Factor (OSF) OSF=0.00025 (diesel exhaust)

Note: The conventional combustible gas detector will not measure diesel vapour at concentrations low enough to determine employee exposures with acceptable sensitivity and accuracy. In addition, available hydrocarbon detectors are not sensitive to measure these levels. Currently there are several instrument methods available to monitor diesel vapour at the concentration necessary to determine employee exposure. The two most readily available are the photo-ionisation detector (PID) and the colourimetric detector tube specifically produced for this purpose.

The requirement ensuring that the atmosphere remains at less than 10% of the LEL (approximately 600 ppm diesel fuel) is not adequate for worker protection.

Toxicity and Irritation data for petroleum-based mineral oils are related to chemical components and vary as does the composition and source of the original crude.

A small but definite risk of occupational skin cancer occurs in workers exposed to persistent skin contamination by oils over a period of years. This risk has been attributed to the presence of certain polycyclic aromatic hydrocarbons (PAH) (typified by benz[a]pyrene).

Petroleum oils which are solvent refined/extracted or severely hydrotreated, contain very low concentrations of both.

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.

for kerosene CAS 8008-20-6

TLV TWA: 100 mg/m3 as total hydrocarbon vapour Skin A3

OEL TWA: 14 ppm, 100 mg/m3 [NIOSH, 1985]

REL TWA: 150 ppm [Shell] CEL TWA: 300 ppm, 900 mg/m3 (CEL = Chemwatch Exposure Limit)

for petroleum distillates:

CEL TWA: 500 ppm, 2000 mg/m3 (compare OSHA TWA)

(CEL = Chemwatch Exposure Limit)

NOTE M: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.005% w/w benzo[a]pyrene (EINECS No 200-028-5). This note applies only to certain complex oil-derived substances in Annex IV.

European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

NOTE N: The classification as a carcinogen need not apply if the full refining history is known and it can be shown that the substance from which it is produced is not a carcinogen. This note applies only to certain complex oil-derived substances in Annex VI.

European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

Exposure controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.

The basic types of engineering controls are:

Process controls which involve changing the way a job activity or process is done to reduce the risk.

Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.

Employers may need to use multiple types of controls to prevent employee overexposure.

General exhaust is adequate under normal operating conditions. If risk of overexposure exists, wear SAA approved respirator, Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

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

Appropriate engineering controls

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.

Personal protection









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Eye and face protection

- ► Safety glasses with side shields.
- Chemical goggles.

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

Skin protection

See Hand protection below

- ▶ Wear chemical protective gloves, e.g. PVC.
- ▶ Wear safety footwear or safety gumboots, e.g. Rubber

NOTE:

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

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.

The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.

Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturizer is recommended.

Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:

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

Hands/feet protection

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

- When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
- Contaminated gloves should be replaced.

For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.

It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.

Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task.

Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:

- Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.
- Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential

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

Body protection

See Other protection below

Other protection

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

Not Available

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

The effect(s) of the following substance(s) are taken into account in the computergenerated selection:

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Material	CPI
NITRILE	С

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

C: Poor to Dangerous Choice for other than short term immersion

NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -

* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.

Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

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

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

Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content. The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.

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SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Brown liquid; does not mix with water.		
Physical state	Liquid	Relative density (Water = 1)	0.8-0.82
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	~180	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	>75	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Combustible.	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	0.07 @ 25C	Gas group	Not Available
Solubility in water (g/L)	Immiscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

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

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

The material is not thought to produce adverse health effects or irritation of the respiratory tract (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.

Fumes from diesel combustion are extremely variable in composition, may contain particulates, unburnt components and may be extremely irritating. Vapour or mist may produce respiratory tract irritation. Human exposure may produce immediate cough, dyspnea, cyanosis and unconsciousness. A productive cough with sputtum smelling of diesel fuel may persist for many days. Chest X-rays have shown diffuse shadowing most prominent at the base of the lungs; this resolved slowly with treatment. High vapour levels may produce central nervous system excitation followed by depression; symptoms include restlessness, confusion, ataxia, headache, dizziness, anorexia, nausea, vomiting, weakness, incoordination, stupor, delirium and coma Inhalation hazard is increased at higher temperatures.

Inhaled

High inhaled concentrations of mixed hydrocarbons may produce narcosis characterised by nausea, vomiting and lightheadedness. Inhalation of aerosols may produce severe pulmonary oedema, pneumonitis and pulmonary haemorrhage. Inhalation of petroleum hydrocarbons consisting substantially of low molecular weight species (typically C2-C12) may produce irritation of mucous membranes, incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and anaesthetic stupor. Massive exposures may produce central nervous system depression with sudden collapse and deep coma; fatalities have been recorded. Irritation of the brain and/or apnoeic anoxia may produce convulsions. Although recovery following overexposure is generally complete, cerebral micro-haemorrhage of focal post-inflammatory scarring may produce epileptiform seizures some months after the exposure. Pulmonary episodes may include chemical pneumonitis with oedema and haemorrhage. The lighter hydrocarbons may produce kidney and neurotoxic effects. Pulmonary irritancy increases with carbon chain length for paraffins and olefins. Alkenes produce pulmonary oedema at high concentrations. Liquid paraffins may produce anaesthesia and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce polyneuropathy. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue and vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons are cardiac sensitisers and may cause ventricular fibrillations.

Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal. Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination

Ingestion

Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result.

Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).

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> Ingestion of petroleum hydrocarbons may produce irritation of the pharynx, oesophagus, stomach and small intestine with oedema and mucosal ulceration resulting; symptoms include a burning sensation in the mouth and throat. Large amounts may produce narcosis with nausea and vomiting, weakness or dizziness, slow and shallow respiration, swelling of the abdomen, unconsciousness and convulsions. Myocardial injury may produce arrhythmias, ventricular fibrillation and electrocardiographic changes. Central nervous system depression may also occur. Light aromatic hydrocarbons produce a warm, sharp, tingling sensation on contact with taste buds and may anaesthetise the tongue. Aspiration into the lungs may produce coughing, gagging and a chemical pneumonitis with pulmonary oedema and haemorrhage.

The material produces severe skin irritation; evidence exists, or practical experience predicts, that the material either:

- ▶ produces severe inflammation of the skin in a substantial number of individuals following direct contact, and/or
- produces significant and severe inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period.
- ▶ Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.

NOTE: Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.

Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.

When applied under a patch for 24 hours to rabbit skin, diesel produced extreme irritation, severe erythema and oedema with blistering and open sores. Topical application has produced acute renal failure and gastrointestinal syndromes in humans.

Many phenylenediamine derivatives are suspected of producing occupational dermatoses with clinical course of the condition closely related to exposure; the dermatoses generally disappear when exposure ceases and reappears if exposure reoccurs

Oxidation of the phenylenediamines reduces dermal absorption. All three isomers are absorbed following ingestion. m- and p-phenylenediamine are metabolised rapidly and excreted predominantly in acetylated form in the urine. There is no selective accumulation of p-phenylenediamine in target organs; corresponding studies have not been carried out with o- or m-phenylenediamine. In contrast to m-phenylenediamine, for which binding to DNA in the kidney and liver has been described, p-phenylenediamine was found to bind to protein in the liver but not to DNA. Oedema, possibly caused by increased vascular permeability, is the dominant symptom of intoxication with p-phenylenediamine, while oedema occurs rarely, if ever, following intoxication with p-phenylenediamine, while oedema occurs rarely, if ever, following intoxication with p-phenylenediamine, while oedema occurs rarely, if ever, following intoxication with p-phenylenediamine, while oedema occurs rarely, if ever, following intoxication with p-phenylenediamine, while oedema occurs rarely, if ever, following intoxication with p-phenylenediamine, while oedema occurs rarely, if ever, following intoxication with p-phenylenediamine, while oedema occurs rarely, if ever, following intoxication with p-phenylenediamine, while oedema occurs rarely, if ever, following intoxication with p-phenylenediamine, while oedema occurs rarely, if ever, following intoxication with p-phenylenediamine, while oedema occurs rarely, if ever, following intoxication with p-phenylenediamine, while oedema occurs rarely, if ever, following intoxication with p-phenylenediamine, while oedema occurs rarely into the occurs rarely into the occurs rarely into the occurs rate of the occurs rate m-phenylenediamine.

Skin Contact

o-, m- and p-Phenylenediamine cause gene mutation in bacteria following metabolic activation. Additionally, o-phenylenediamine has been observed to damage bacterial DNA in the repair test. All three isomers had predominantly no effect on gene mutation in fungi, even with metabolic activation, while positive results were recorded with o-, m- and p-phenylenediamine in mammalian cells. Studies of the damaging effect of o-phenylenediamine on DNA and chromosomes in mammalian cells produced predominantly positive results, even without metabolic activation. The damaging effects of m- and p-phenylenediamine on DNA and chromosomes, however, vary according to the test system, and both positive and negative findings have been obtained. The three phenylenediamine isomers have been observed to form strongly mutagenic oxidation products which influence test results.

There are a few studies on the carcinogenic effect of m- and p-phenylenediamine using various methods of administration in which only subcutaneous injection produced localised tumours, o-Phenylenediamine, on the other hand, produced liver tumours in the rat and mouse only after oral administration. No short-term carcinogenicity studies have been carried out with o-phenylenediamine. m- and p-phenylenediamine led to cell transformations in vitro, in vivo studies of tumour promotion in the liver were negative.

o-, m- and p-Phenylenediamine do not impair fertility in spite of the fact that o-phenylenediamine was observed to have embryotoxic and sperm-damaging effects in unvalidated studies. p-Phenylenediamine is not teratogenic. Embryotoxic and slight teratogenic effects were observed with m-phenylenediamine at clearly maternotoxic doses, possibly as a result of a deficiency of nutrient supply to the fetus. No studies have been carried out on the teratogenic effect of o-phenylenediamine.

o-, m- and p-phenylenediamine all form methaemoglobin. The highest level of methaemoglobin formation was observed with m-phenylenediamine. There are only isolated reports of human sensitization by o- and m-phenylenediamine.

p-Phenylenediamine, on the other hand, is a very common allergen in man because of allergy to the para-group. Cases of photosensitisation induced by p-phenylenediamine have also been recorded.

Open cuts, abraded or irritated skin should not be exposed to this material

Toxic effects may result from skin absorption

The material may accentuate any pre-existing dermatitis condition

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.

Aromatic hydrocarbons may produce skin irritation, vasodilation with erythema and changes in endothelial cell permeability. Systemic intoxication, resulting from contact with the light aromatics, is unlikely due to the slow rate of permeation. Branching of the side chain appears to increase percutaneous absorption.

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).

Petroleum hydrocarbons may produce pain after direct contact with the eyes. Slight, but transient disturbances of the corneal epithelium may also result. The aromatic fraction may produce irritation and lachrymation.

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.

Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial number of individuals at a greater frequency than would be expected from the response of a normal population.

Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking.

Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.

Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.

Prolonged or repeated skin contact with diesel fuel may cause defatting and irritation of follicles with blocked sebaceous glands resulting in pimples and spots appearing on arms and legs. Hyperkeratosis has been described in engine drivers exposed occupationally to diesel fuels. Repeated application to rabbit skin produces mortalities (8 ml/kg). The primary cause of death was depression and anorexia which were induced by dermal irritation followed by infection; systemic intoxication did not appear to be a factor. Autopsy showed liver and kidney effects.

Chronic

Long term exposure to mist / furnes or ingestion may cause severe central nervous system deficiencies. Chronic exposure or voluntary sniffing can lead to bone marrow deficiencies. [CCINFO]

Animal studies confirm an association between cancer, primarily of the lung and inhalation of whole diesel exhaust

Principal route of exposure is by skin contact; lesser exposures include inhalation of fumes from hot oils, oil mists or droplets. Prolonged contact with mineral oils carries with it the risk of skin conditions such as oil folliculitis, eczematous dermatitis, pigmentation of the face (melanosis) and warts on the sole of the foot (plantar warts). With highly refined mineral oils no appreciable systemic effects appear to result through skin absorption.

Exposure to oil mists frequently elicits respiratory conditions, such as asthma; the provoking agent is probably an additive. High oil mist concentrations may produce lipoid pneumonia although clinical evidence is equivocal. In animals exposed to concentrations of 100 mg/m3 oil mist, for periods of 12 to 26 months, the activity of lung and serum alkaline phosphatase enzyme was raised; 5 mg/m3 oil mist did not produce this response. These enzyme changes are sensitive early indicators of lung damage. Workers exposed to vapours of mineral oil and kerosene for 5 to 35 years showed an increased prevalence of slight basal lung

Many studies have linked cancers of the skin and scrotum with mineral oil exposure. Contaminants in the form of additives and the polycyclic aromatic hydrocarbons (PAHs - as in the crude base stock) are probably responsible. PAH levels are higher in aromatic process oils/used/reclaimed motor oils

Subchronic 90-day feeding studies conducted on male and female rats on highly refined white mineral oils and waxes found that higher molecular-weight

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hydrocarbons (microcrystalline waxes and the higher viscosity oils) were without biological effects. Paraffin waxes and low- to mid viscosity oils produced biological effects that were inversely proportional to molecular weight, viscosity and melting point: oil-type and processing did not appear to be determinants. Biological effects were more pronounced in females than in males. Effects occurred mainly in the liver and mesenteric lymph nodes and included increased organ weights, microscopic inflammatory changes, and evidence for the presence of saturated mineral hydrocarbons in affected tissues. Inflammation of the cardiac mitral valve was also observed at high doses in rats treated with paraffin waxes.

Smith J.H., et al: Toxicologic Pathology: 24, 2, 214-230, 1996

Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/or memory loss, tremor in the fingers and tongue, vertigo, olfactory disorders, constriction of visual field, paraesthesias of the extremities, weight loss and anaemia and degenerative changes in the liver and kidney. Chronic exposure by petroleum workers, to the lighter hydrocarbons, has been associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness and paraesthesias), psychological and neurophysiological deficits, bone marrow toxicities (including hypoplasia possibly due to benzene) and hepatic and renal involvement. Chronic dermal exposure to petroleum hydrocarbons may result in defatting which produces localised dermatoses. Surface cracking and erosion may also increase susceptibility to infection by microorganisms. One epidemiological study of petroleum refinery workers has reported elevations in standard mortality ratios for skin cancer along with a dose-response relationship indicating an association between routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have been unable to confirm this finding.

Many phenylenediamine derivatives are suspected of producing occupational dermatoses with clinical course of the condition closely related to exposure; the dermatoses generally disappear when exposure ceases and reappears if exposure reoccurs.

Oxidation of the phenylenediamines reduces dermal absorption. All three isomers are absorbed following ingestion. m- and p-phenylenediamine are metabolised rapidly and excreted predominantly in acetylated form in the urine. There is no selective accumulation of p-phenylenediamine in target organs; corresponding studies have not been carried out with o- or m-phenylenediamine. In contrast to m-phenylenediamine, for which binding to DNA in the kidney and liver has been described, p-phenylenediamine was found to bind to protein in the liver but not to DNA. Oedema, possibly caused by increased vascular permeability, is the dominant symptom of intoxication with p-phenylenediamine, while oedema occurs rarely, if ever, following intoxication with o- or m-phenylenediamine.

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Cemix APRA	TOXICITY Not Available	IRRITATION Not Available
diesel	TOXICITY Dermal (rabbit) LD50: >1800 mg/kg ^[1] Oral (rat) LD50: >5000 mg/kg ^[1]	IRRITATION Skin (rabbit): 500 uL/24h SEVERE
4'-aminoacetanilide	TOXICITY Oral (rat) LD50: 2500 mg/kg ^[2]	IRRITATION Eye (rabbit): 100 mg/24h - mod
mineral oil	TOXICITY Not Available	IRRITATION Not Available
Legend:	Value obtained from Europe ECHA Registered Substances - Acute toxicity 2 extracted from RTECS - Register of Toxic Effect of chemical Substances	2.* Value obtained from manufacturer's SDS. Unless otherwise specified data

The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis.

Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.

For "kerosenes"

Acute toxicity: Oral LD50s for three kerosenes (Jet A, CAS No. 8008-20-6 and CAS No. 64742-81-0) ranged from > 2 to >20 g/kg. The dermal LD50s of the same three kerosenes were all >2.0 g//kg. Inhalation LC50 values in Sprague-Dawley rats for straight run kerosene (CAS No. 8008-20-6) and hydrodesulfurised kerosene (CAS No. 64742-81-0) were reported to be > 5 and > 5.2 mg/l, respectively. No mortalities in rats were reported in rats when exposed for eight hours to saturated vapor of deodorised kerosene (probably a desulfurised kerosene). Six hour exposures of cats to the same material produced an LC50 of >6.4 mg/l

DIESEL

When tested in rabbits for skin irritation, straight run kerosene (CAS No. 8008-20-6) produced "moderate" to "severe" irritation. Six additional skin irritation studies on a range of kerosenes produced "mild" to "severe" irritation.

An eye irritation in rabbits of straight run kerosene (CAS No. 8008-20-6) produced Draize scores of 0.7 and 2.0 (unwashed and washed eyes) at 1 hour. By 24 hours, the Draize scores had returned to zero. Eye irritation studies have also been reported for hydrodesulfurized kerosene and jet fuel. These materials produced more irritation in the unwashed eyes at 1 hour than had the straight run kerosene. The eye irritation persisted longer than that seen with straight run kerosene, but by day 7 had resolved.

Straight run kerosene (CAS No. 8008-20-6), Jet A, and hydrodesulfurized kerosene (CAS No. 64742-81-0) have not produced sensitisation when tested in quinea pigs

Repeat-Dose toxicity: Multiple repeat-dose toxicity studies have been reported on a variety of kerosenes or jet fuels. When applied dermally, kerosenes and jet fuels have been shown to produce dermal and systemic effects

Dose levels of 200, 1000 and 2000 mg/kg of a straight run kerosene (CAS No. 8008-20-6) were applied undiluted to the skin of male and female New Zealand white rabbits The test material was applied 3x/week for 28 days. One male and one female in the 2000 mg/kg dose group found dead on days 10 and 24

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respectively were thought to be treatment-related. Clinical signs that were considered to be treatment-related included: thinness, nasal discharge, lethargy, soiled anal area, anal discharge, wheezing. The high dose group appeared to have a treatment related mean body weight loss when compared to controls. Dose-related skin irritation was observed, ranging from "slight" to "moderate" in the low and high dose groups, respectively. Other treatment-related dermal findings included cracked, flaky and/or leathery skin, crusts and/or hair loss, Reductions in RBC, haemoglobin and haematocrit were seen in the male dose groups. There were no treatment related effects on a variety of clinical chemistry values. Absolute and relative weights for a number of organs were normal, with the following exceptions that were judged to be treatment-related:

- increased relative heart weights for the mid- and high- dose males and females,
- increased absolute and relative spleen weights in treated females, and
- differences in absolute and relative adrenal weights in both male and female treated animals (considered to be stress-related and therefore, indirectly related to treatment)

Gross necropsy findings were confined largely to the skin. Enlarged spleens were seen in the female groups. Microscopic examination of tissues taken at necropsy found proliferative inflammatory changes in the treated skin of all male and female animals in the high dose group. These changes were, in the majority of animals, accompanied by an increase in granulopoiesis of the bone marrow. Four of six high dose males had testicular changes (multifocal or diffuse tubular hypoplasia) that were considered by the study authors to be secondary to the skin and/or weight changes.

In a different study, hydrodesulfurised kerosene was tested in a thirteen-week dermal study using Sprague-Dawley rats. Test material was applied 5x/week to the skin of male and female rats at dose levels of 165, 330 and 495 mg/kg. Aside from skin irritation at the site of application, there were no treatment-related clinical signs during the study. Screening of all animals using a functional observation battery (FOB) did not find any substance-related effects. Opthalomological examination of all animals also found no treatment-related effects. There were no treatment-related effects on growth rates, hematological or clinical chemical values, or absolute or relative organ weights. Microscopic examination of tissues from animals surviving to termination found no treatment-related changes, with the exception of a minimal degree of a proliferative and inflammatory changes in the skin.

A hydrodesulfurised middle distillate (CAS no. 64742-80-9) has also been tested in a four week inhalation study. In the study, Sprague-Dawley rats were exposed to a nominal concentration of 25mg/m3 kerosene. Exposures were for approximately 6 hr/day, five days each week for four consecutive weeks. There were no treatment-related effects on clinical condition, growth rate, absolute or relative organ weights, or any of the hematological or clinical chemistry determinations. Microscopic examination found no treatment-related changes observed in any tissues.

Carcinogenicity: In addition to the repeat-dose studies discussed above, a number of dermal carcinogenicity studies have been performed on kerosenes or jet fuels. .Following the discovery that hydrodesulfurised (HDS) kerosene caused skin tumors in lifetime mouse skin painting studies, the role of dermal irritation in tumor formation was extensively studied. HDS kerosene proved to be a mouse skin tumor promoter rather than initiator, and this promotion required prolonged dermal irritation. If the equivalent dose of kerosene was applied to the skin in manner that did not cause significant skin irritation (eg, dilution with a mineral oil) no skin tumors occurred . Dermal bioavailability studies in mice confirmed that the reduced irritation seen with samples in mineral oil was not due to decreased skin penetration. The effect of chronic acanthosis on the dermal tumorigenicity of a hydrodesulfurised kerosene was studied and the author concluded that hyperplasia was essential for tumor promotion. However, the author also concluded that subacute inflammation did not appear to be a significant factor A sample of a hydrodesulfurised kerosene has been tested in an initiation-promotion assay in male CD-1 mice. Animal survivals were not effected by exposure to the kerosene. The study's authors concluded that the kerosene was not an initiator but it did show tumor promoting activity.

In-Vitro (Genotoxicity): The potential in vitro genotoxicities of kerosene and jet fuel have been evaluated in a variety of studies. Standard Ames assays on two kerosene samples and a sample of Jet A produced negative results with/without activation . Modified Ames assays on four kerosenes also produced negative results (with/without activation) except for one positive assay that occurred with activation. The testing of five kerosene and jet fuel samples in mouse lymphoma assays produced a mixture of negative and positive results. Hydrodesulfurized kerosene tested in a sister chromatid exchange assay produced negative results (with/without activation)

In-Vivo Genotoxicity: Multiple in vivo genotoxicity studies have been done on a variety of kerosene-based materials. Four samples of kerosene were negative and a sample of Jet A was positive in in vivo bone marrow cytogenetic tests in Sprague-Dawley rats. One of the kerosene samples produced a positive response in male mice and negative results in females when tested in a sister chromatid exchange assay . Both deodorised kerosene and Jet A samples produced negative results in dominant lethal assays. The kerosene was administered to both mice and rats intraperitoneally, while the jet fuel was administered only to mice via inhalation.

Reproductive/Developmental Toxicity Either 0, 20, 40 or 60% (v/v) kerosene in mineral oil was applied to the skin of the rats. The dose per body weight equivalents were 0, 165, 330 and 494 mg/kg. Test material was applied daily, 7 days/week from 14 days premating through 20 days of gestation. There were no treatment-related effects on mortality and no clinical signs of toxicity were observed. There were no compound-related effects on any of the reproductive/developmental parameters. The authors concluded that the no observable effect level (NOEL) for reproductive/developmental toxicity of HDS kerosene under the treatment conditions of the study was 494 mg/kg/day.

Developmental toxicity screening studies on a kerosene and a sample of Jet A have been reported . There were no compound-related deaths in either study. While kerosene produced no clinical signs, the jet fuel produced a dose-related eye irritation (or infection). The signs of irritation lasted from 2 to 8 days with most animals showing signs for 3 days. Neither of the test materials had an effect on body weights or food consumption. Examination of offspring at delivery did not reveal any treatment-related abnormalities, soft tissue changes or skeletal abnormalities. The sex ratio of the fetuses was also unaffected by treatment with either of the compounds.

The substance is classified by IARC as Group 3:

NOT classifiable as to its carcinogenicity to humans.

Evidence of carcinogenicity may be inadequate or limited in animal testing.

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 which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens).

4'-AMINOACETANILIDE

Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis

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

The following information refers to contact allergens as a group and may not be specific to this product.

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

The material may produce moderate eye irritation leading to 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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MINERAL OIL	Toxicity and Irritation data for petroleum-based mineral oils are related to chemical components and vary as does the composition and source of the original crude. A small but definite risk of occupational skin cancer occurs in workers exposed to persistent skin contamination by oils over a period of years. This risk has been attributed to the presence of certain polycyclic aromatic hydrocarbons (PAH) (typified by benz[a]pyrene). Petroleum oils which are solvent refined/extracted or severely hydrotreated, contain very low concentrations of both.		
Acute Toxicity	\otimes	Carcinogenicity	❤
Skin Irritation/Corrosion	✓	Reproductivity	0
Serious Eye Damage/Irritation	0	STOT - Single Exposure	✓
Respiratory or Skin sensitisation	~	STOT - Repeated Exposure	0
Mutagenicity	0	Aspiration Hazard	✓

Legend:

X - Data available but does not fill the criteria for classification

✓ – Data available to make classification

O - Data Not Available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Cemix APRA	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
dia a d	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
diesel	NOEC	3072	Fish	=1mg/L	1
4'-aminoacetanilide	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
4-aminoacetaniilde	LC50	96	Fish	>500mg/L	6
mineral oil	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available

Legend:

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

Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
4'-aminoacetanilide	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
diesel	LOW (BCF = 159)
4'-aminoacetanilide	LOW (BCF = 7.2)

Mobility in soil

Ingredient	Mobility
4'-aminoacetanilide	LOW (KOC = 61.72)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging

disposal

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

Otherwise:

Fig container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.

▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product.

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

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

- ► Reduction
- ► Reuse
- ▶ Recycling

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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.

Ensure that the disposal of material is carried out in accordance with Hazardous Substances (Disposal) Regulations 2001.

SECTION 14 TRANSPORT INFORMATION

Labels Required



Marine Pollutant



HAZCHEM •3Z

Land transport (UN)

UN number	3082		
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains diesel)		
Transport hazard class(es)	Class 9 Subrisk Not Applicable		
Packing group			
Environmental hazard	Environmentally hazardous		
Special precautions for user	Special provisions 274; 331; 335; 375 Limited quantity 5 L		

Air transport (ICAO-IATA / DGR)

UN number	3082		
UN proper shipping name	Environmentally hazardous substance, liquid, n.o.s. * (contains diesel)		
Transport hazard class(es)	ICAO/IATA Class 9 ICAO / IATA Subrisk Not Applicable ERG Code 9L		
Packing group	III		
Environmental hazard	Environmentally hazardous		
	Special provisions	A97 A158 A197	
	Cargo Only Packing Instructions	964	
Special precautions for user	Cargo Only Maximum Qty / Pack	450 L	
	Passenger and Cargo Packing Instructions	964	
	Passenger and Cargo Maximum Qty / Pack	450 L	
	Passenger and Cargo Limited Quantity Packing Instructions	Y964	
	Passenger and Cargo Limited Maximum Qty / Pack	30 kg G	

Sea transport (IMDG-Code / GGVSee)

UN number	3082	
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains diesel)	
Transport hazard class(es)	IMDG Class 9 IMDG Subrisk Not Applicable	
Packing group	III	
Environmental hazard	Marine Pollutant	

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EMS Number F-A, S-F 274 335 969 Special precautions for user Special provisions Limited Quantities 5 L

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

This substance is to be managed using the conditions specified in an applicable Group Standard

HSR Number	Group Standard
HSR002544	Construction Products (Subsidiary Hazard) Group Standard 2006

DIESEL(68334-30-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

New Zealand Inventory of Chemicals (NZIoC) International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of

Chemicals

4'-AMINOACETANILIDE(122-80-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Not Applicable

MINERAL OIL(NOT AVAIL.) IS FOUND ON THE FOLLOWING REGULATORY LISTS

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

New Zealand Workplace Exposure Standards (WES)

Location Test Certificate

Subject to Regulation 55 of the Hazardous Substances (Classes 1 to 5 Controls) Regulations, a location test certificate is required when quantity greater than or equal to those indicated below are present.

Hazard Class	Quantity beyond which controls apply for closed containers	Quantity beyond which controls apply when use occurring in open containers
Not Applicable	Not Applicable	Not Applicable

Approved Handler

Subject to Regulation 56 of the Hazardous Substances (Classes 1 to 5 Controls) Regulations and Regulation 9 of the Hazardous Substances (Classes 6, 8, and 9 Controls) Regulations, the substance must be under the personal control of an Approved Handler when present in a quantity greater than or equal to those indicated below.

Class of substance	Quantities
Not Applicable	Not Applicable

Refer Group Standards for further information

Tracking Requirements

Not Applicable

National Inventory	Status		
Australia - AICS	N (mineral oil)		
Canada - DSL	N (mineral oil)		
Canada - NDSL	N (4'-aminoacetanilide; mineral oil; diesel)		
China - IECSC	N (mineral oil)		
Europe - EINEC / ELINCS / NLP	N (mineral oil)		
Japan - ENCS	N (mineral oil)		
Korea - KECI	N (mineral oil)		
New Zealand - NZIoC	N (4'-aminoacetanilide; mineral oil)		
Philippines - PICCS	N (4'-aminoacetanilide; mineral oil)		
USA - TSCA	N (mineral oil)		
Legend:	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)		

SECTION 16 OTHER INFORMATION

Other information

Ingredients with multiple cas numbers

Name	CAS No
diesel	68334-30-5, 68512-90-3, 64742-81-0, 68476-30-2

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available literature references.

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

Definitions and abbreviations

PC – TWA: Permissible Concentration-Time Weighted Average

PC-STEL: Permissible Concentration-Short Term Exposure Limit

IARC: International Agency for Research on Cancer

ACGIH: American Conference of Governmental Industrial Hygienists

STEL: Short Term Exposure Limit

TEEL: Temporary Emergency Exposure Limit。

IDLH: Immediately Dangerous to Life or Health Concentrations

OSF: Odour Safety Factor

NOAEL :No Observed Adverse Effect Level

LOAEL: Lowest Observed Adverse Effect Level

TLV: Threshold Limit Value

LOD: Limit Of Detection

OTV: Odour Threshold Value

BCF: BioConcentration Factors BEI: Biological Exposure Index

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