

AC Menace 960 Herbicide

AXICHEM Pty Ltd

Chemwatch: 5239-58

Version No: 5.1

Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Chemwatch Hazard Alert Code: 2

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L.GHS.AUS.EN

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

Product Identifier

Product name	AC Menace 960 Herbicide
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains metolachlor)
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Selective herbicide for pre-emergence and preplant incorporated weed control.
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Details of the supplier of the safety data sheet

Registered company name	AXICHEM Pty Ltd
Address	9 Palings Court Nerang QLD 4211 Australia
Telephone	07 5596 1736
Fax	Not Available
Website	www.axichem.com.au
Email	msds@axichem.com.au

Emergency telephone number

Association / Organisation	CHEMWATCH EMERGENCY RESPONSE
Emergency telephone numbers	+61 1800 951 288
Other emergency telephone numbers	+61 2 9186 1132

Once connected and if the message is not in your preferred language then please dial 01

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	S5
Classification ^[1]	Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 2A, Acute Toxicity (Inhalation) Category 4, Reproductive Toxicity Category 2, Hazardous to the Aquatic Environment Acute Hazard Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 1
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	
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Signal word **Warning****Hazard statement(s)**

H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H319	Causes serious eye irritation.
H332	Harmful if inhaled.
H361fd	Suspected of damaging fertility. Suspected of damaging the unborn child.
H401	Toxic to aquatic life.
H410	Very toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P302+P352	IF ON SKIN: Wash with plenty of water.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Collect spillage.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.
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Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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SECTION 3 Composition / information on ingredients**Substances**

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
51218-45-2	96	<u>metolachlor</u>
64742-94-5	<4	<u>solvent naphtha petroleum, heavy aromatic</u>
37251-69-7	<4	<u>nonylphenol, ethoxylated, propoxylated</u>
61791-26-2	<4	<u>tallow alkylamine, ethoxylated</u>
Not Available	balance	Ingredients determined not to be hazardous

Legend: 1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> Wash out immediately with fresh running water. Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. Seek medical attention without delay; if pain persists or recurs seek medical attention. Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> 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	<ul style="list-style-type: none"> If fumes, aerosols or combustion products are inhaled remove from contaminated area. Other measures are usually unnecessary.
Ingestion	<ul style="list-style-type: none"> 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.

Indication of any immediate medical attention and special treatment needed

Rapidly metabolised by rat liver microsomal oxygenases via dechlorination, O-methylation, N-dealkylation and side-chain oxidation.

The material may induce methaemoglobinaemia following exposure.

- Initial attention should be directed at oxygen delivery and assisted ventilation if necessary. Hyperbaric oxygen has not demonstrated substantial benefits.
- Hypotension should respond to Trendelenburg's position and intravenous fluids; otherwise dopamine may be needed.
- Symptomatic patients with methaemoglobin levels over 30% should receive methylene blue. (Cyanosis, alone, is not an indication for treatment). The usual dose is 1-2 mg/kg of a 1% solution (10 mg/ml) IV over 50 minutes; repeat, using the same dose, if symptoms of hypoxia fail to subside within 1 hour.
- Thorough cleansing of the entire contaminated area of the body, including the scalp and nails, is of utmost importance.

BIOLOGICAL EXPOSURE INDEX - BEI

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

Determinant	Index	Sampling Time	Comment
1. Methaemoglobin in blood	1.5% of haemoglobin	During or end of shift	B, NS, SQ

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

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

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

SECTION 5 Firefighting measures

Extinguishing media

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

Special hazards arising from the substrate or mixture

Fire Incompatibility	<ul style="list-style-type: none"> Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> 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 courses. Use water delivered as a fine spray to control fire and cool adjacent area. DO NOT approach containers suspected to be hot. Cool fire exposed containers with water spray from a protected location. If safe to do so, remove containers from path of fire. Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	<ul style="list-style-type: none"> Combustible. Slight fire hazard when exposed to heat or flame. Heating may cause expansion or decomposition leading to violent rupture of containers.

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	<ul style="list-style-type: none"> ▶ On combustion, may emit toxic fumes of carbon monoxide (CO). ▶ May emit acrid smoke. ▶ Mists containing combustible materials may be explosive. <p>Combustion products include: carbon dioxide (CO₂) hydrogen chloride phosgene nitrogen oxides (NO_x) other pyrolysis products typical of burning organic material.</p>
HAZCHEM	•3Z

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	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> ▶ 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	<p>Environmental hazard - contain spillage. Moderate hazard.</p> <ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ No smoking, naked lights or ignition sources. ▶ Increase ventilation. ▶ Stop leak if safe to do so. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Absorb remaining product with sand, earth or vermiculite. ▶ Collect solid residues and seal in labelled drums for disposal. ▶ Wash area and prevent runoff into drains. ▶ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ 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. ▶ Avoid smoking, naked lights or ignition sources. ▶ 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. ▶ 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.
Other information	<ul style="list-style-type: none"> ▶ Store in original containers. ▶ Keep containers securely sealed.

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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 storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	<p>Corrodes mild steel.</p> <ul style="list-style-type: none"> ▶ DO NOT use unlined steel containers ▶ Metal can or drum ▶ Packaging as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<ul style="list-style-type: none"> ▶ Many arylamines (aromatic amines such as aniline, N-ethylaniline, o-toluidine, xyldine etc. and their mixtures) are hypergolic (ignite spontaneously) with red fuming nitric acid. When the amines are dissolved in triethylamine, ignition occurs at -60 deg. C. or less. ▶ Various metal oxides and their salts may promote ignition of amine-red fuming nitric acid systems. Soluble materials such as copper(I) oxide, ammonium metavanadate are effective; insoluble materials such as copper(II) oxide, iron(II) oxide, potassium dichromate are also effective. ▶ Avoid oxidising agents, acids, acid chlorides, acid anhydrides, chloroformates.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
AC Menace 960 Herbicide	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
metolachlor	Not Available	Not Available
solvent naphtha petroleum, heavy aromatic	Not Available	Not Available
nonylphenol, ethoxylated, propoxylated	Not Available	Not Available
tallow alkylamine, ethoxylated	Not Available	Not Available

Occupational Exposure Banding


Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
metolachlor	D	> 0.1 to ≤ 1 ppm
nonylphenol, ethoxylated, propoxylated	E	≤ 0.1 ppm
tallow alkylamine, ethoxylated	C	> 1 to ≤ 10 parts per million (ppm)

Notes:

Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.

MATERIAL DATA

Exposure controls

Appropriate engineering controls	General exhaust is adequate under normal operating conditions.
Personal protection	

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Eye and face protection	<ul style="list-style-type: none"> ▸ Safety glasses with side shields. ▸ Chemical goggles. ▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	<ul style="list-style-type: none"> ▸ Wear chemical protective gloves, e.g. PVC. ▸ Wear safety footwear or safety gumboots, e.g. Rubber <p>NOTE:</p> <ul style="list-style-type: none"> ▸ 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. <p>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.</p> <p>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.</p> <p>Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> · 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. <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>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.</p> <p>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.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> · 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 <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p>
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▸ Overalls. ▸ P.V.C apron. ▸ Barrier cream. ▸ Skin cleansing cream. ▸ Eye wash unit.

Respiratory protection

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

Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator
up to 10	1000	AK-AUS / Class1 P3	-

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up to 50	1000	-	AK-AUS / Class 1 P3
up to 50	5000	Airline *	-
up to 100	5000	-	AK-2 P3
up to 100	10000	-	AK-3 P3
100+			Airline**

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

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Clear, yellowish to brown liquid with weak odour; mixes with water.		
Physical state	Liquid	Relative density (Water = 1)	1.085-1.125
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	430
pH (as supplied)	Not Applicable	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	120	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	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)	4.2 mPa (25 C)	Gas group	Not Available
Solubility in water	Miscible	pH as a solution (Not Available%)	4-8
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ▸ Unstable in the presence of incompatible materials. ▸ Product is considered stable. ▸ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	The material is not thought to produce either adverse health effects or irritation of the respiratory tract following inhalation (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.
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Ingestion	<p>Accidental ingestion of the material may be damaging to the health of the individual.</p> <p>The substance and/or its metabolites may bind to haemoglobin inhibiting normal uptake of oxygen. This condition, known as "methaemoglobinemia", is a form of oxygen starvation (anoxia).</p> <p>Symptoms include cyanosis (a bluish discolouration skin and mucous membranes) and breathing difficulties. Symptoms may not be evident until several hours after exposure.</p> <p>At about 15% concentration of blood methaemoglobin there is observable cyanosis of the lips, nose and earlobes. Symptoms may be absent although euphoria, flushed face and headache are commonly experienced. At 25-40%, cyanosis is marked but little disability occurs other than that produced on physical exertion. At 40-60%, symptoms include weakness, dizziness, lightheadedness, increasingly severe headache, ataxia, rapid shallow respiration, drowsiness, nausea, vomiting, confusion, lethargy and stupor. Above 60% symptoms include dyspnea, respiratory depression, tachycardia or bradycardia, and convulsions. Levels exceeding 70% may be fatal.</p>
Skin Contact	<p>The material may produce mild skin irritation; limited evidence or practical experience suggests, that the material either:</p> <ul style="list-style-type: none"> ▶ produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or ▶ produces significant, but mild, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. <p>Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (non allergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>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.</p>
Eye	<p>Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals.</p> <p>Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p>
Chronic	<p>Prolonged or repeated contact may irritate the skin causing dermatitis and photosensitisation. Persons with allergic history or pre-existing dermatitis should use extra care.</p> <p>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.</p> <p>Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.</p> <p>Substances than can cause occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers</p> <p>Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.</p> <p>Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>Most arylamines are powerful haemopoietic poisons producing methaemoglobinaemia in humans. Addition of alkyl groups may modify the toxic responses but nevertheless these remains similar to the parent compound. High chronic doses cause splenic congestion and in turn sarcoma formation. Single ring aromatic amines are relatively weak carcinogens requiring large doses to produce any effect in animal experiments. The polycyclic aromatic amines exhibit a wide range of carcinogenic activity which appear, in part, to be dependent on the position on which benzene rings are substituted and the nature of the substituent.</p> <p>Most monocyclic arylamines produce haemosiderosis (deposition of iron-containing proteins in tissues and organs). It is not clear whether the genotoxic and acute toxic effects are influenced by the release of iron during methaemoglobin formation or erythrocyte turnover and by the "oxidative stress" associated with these processes. In any case, toxic tissue changes and fibrosis precede tumour development in the spleen, liver and kidneys.</p> <p>Metabolism of arylamines generally proceeds through N-oxidation, hydroxylation of aromatic ring carbons, and formation of conjugates such as glucuronides, sulfates, and acetates. Ring alkyl substituents may also be oxidised to alcohols and further metabolised to acids. N-oxidation is an important step that can lead to the formation of metabolites that will react with cellular macromolecules. The N-phenylhydroxylamines and nitrosobenzenes produced by N-oxidation are capable of binding to the haeme ion in haemoglobin and causing oxidation. This reaction can produce the methaemoglobinaemia that is the most typical toxicity associated with aromatic amines.</p> <p>Metabolites of aromatic amines, especially those which have undergone N-hydroxylation appear to be the active principle in the development of bladder cancers. Induced methaemoglobinaemia may be an indicator of the formation of an N-hydroxylated metabolite. Formation of N-hydroxylated metabolites and conjugates thereof is considered to be an important step in the mechanism of activation for various carcinogenic aromatic amines.</p> <p>Due to methaemoglobin forming activity aromatic amino or nitro compounds may exert developmental toxicity at least as a secondary consequence of maternal toxicity..</p>

N-oxidation leading to the formation of N-hydroxylamines (see above) can further produce reactive nitrosoarenes ultimately leading to the formation of reactive arylnitrenium ions. These in turn may react with skin proteins to produce sensitising effects (as haptens)

Aromatic amines are closely related to catechols and hydroquinones with p-phenylenediamine (PPD) as a prominent example. PPD is a frequently occurring and potent skin sensitizer commonly used in hair dyes. PPD is known to readily autoxidize to a variety of degradation products and p-quinonediimines and semiquinoneimine radicals (known as Würster radicals) are examples of intermediates of potential importance in contact allergy to PPD and related compounds.

AC Menace 960 Herbicide	TOXICITY	IRRITATION
	Dermal (Rat) LD50: 2200 mg/kg ^[2]	Not Available
	Inhalation (Rat) LC50: >4060 mg/m ³ /4h ^[2]	
metolachlor	Oral (Rat) LD50: 2267 mg/kg ^[2]	
	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	Eye (rabbit): 100 mg - mild
solvent naphtha petroleum, heavy aromatic	Inhalation(Rat) LC50; >1.75 mg/l4h ^[2]	Skin (rabbit): 334 mg open - mild
	Oral (Mouse) LD50; 1150 mg/kg ^[2]	
	TOXICITY	IRRITATION
nonylphenol, ethoxylated, propoxylated	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	Eye (rabbit): Irritating
	Inhalation(Rat) LC50; >0.003 mg/L4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50; 512 mg/kg ^[1]	Skin: adverse effect observed (irritating) ^[1]
tallow alkylamine, ethoxylated	TOXICITY	IRRITATION
	Oral (Rat) LD50; 1990 mg/kg ^[2]	Not Available
Legend:	TOXICITY	IRRITATION
	dermal (rat) LD50: >10000 mg/kg ^[2]	Eye(rabbit)(Draize): moderate
	Oral (Rat) LD50; 500 mg/kg ^[2]	
<p>Legend: 1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. * Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances</p>		

METOLACHLOR

Toxicity Class WHO III; EPA III * NOEL (90 d) for rats 300 mg/kg diet (50 mg/kg daily); for dogs 300 mg/kg diet (12.5 mg/kg daily)
 * ADI: 0.08 mg/kg/day NOEL: 7.5 mg/kg/day

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.

In mammals, fatty acid elongation greater than C18 also occurs, primarily on the endoplasmic reticulum, and utilizes CoA derivatives, as is found in plants. In mammals, long-chain fatty acids are important for membrane phospholipids and for neural growth and myelination.

The acetanilide and thiocarbamate herbicides are relatively non-toxic to mammals but some effects have been noted. Molinate, a thiocarbamate, has caused testicular lesions in rats with a single dose, after sulfoxidation within the organism. The lesion was characterized by failed spermiogenesis and phagocytosis of spermatids. In a 2-year rat study, metolachlor, an acetanilide, caused the wasting of testicles at doses of 150 mg/kg/day. Acetochlor has also been shown to cause testicular toxicity in male dogs given 10 and 50 mg/kg/day with a decrease in testes weight, atrophy and degeneration of seminiferous tubules and hypospermia. There were also effects on the kidney and severe neurological effects at 50 mg/kg/day consisting of abnormal head movements, stiffness and rigidity of hind limbs, ataxia tremor and other symptoms. These effects were accompanied by histopathological findings in the vermis cerebellum. The toxic effect of the sulfoxide metabolite of molinate was attributed to inhibition of esterase activity, which decreased plasma and testicular testosterone concentrations. However, this metabolite seems to be selectively produced in rodents and is not found in other mammals, including humans. No connection has ever been made between the toxic effects of acetanilides and thiocarbamates on mammals and inhibition of VLCFAs. However, very-long-chain polyunsaturated fatty acids (>24) are normally found in excitatory tissues, and myelin-deficient mouse mutants have very low fatty acid elongation activity. In addition, very-long-chain fatty acids are highly important in rat sperm maturation. During their transit from the caput to the cauda segments of the epididymis, rat spermatozoa lipid content and composition change significantly. The proportions of oleate and linoleate fatty acids decrease and there is an increase in the longer-chain fatty acids (C20 – C24) as well as the uncommon long-chain polyenoic fatty acids of the n-9 series. It might be highly informative to determine whether these two classes of herbicides inhibit very-long-chain fatty acid biosynthesis in mammals as well as in plants, and to see whether there is any connection between the mammalian toxicity of these chemicals and very-long-chain fatty acid synthesis..

	<p>The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <p>Various (chloro)acetanilide (syn: chloracetamide) pesticides have been shown to result in several different types of tumor responses in laboratory animals (e.g., nasal, thyroid, liver, and stomach tumors).</p> <p>There is sufficient evidence to support the grouping of certain chloroacetanilides that cause nasal turbinate tumors by a common mechanism of toxicity; these produce a highly, tissue reactive metabolite, i.e., quinoneimine. Only acetochlor, alachlor, and butachlor are grouped based on a common mechanism of toxicity for nasal turbinate tumors. Although metolachlor does distribute to the nasal turbinates, and might produce a quinoneimine, it is not apparent from currently available data that it shares the same target site in the nasal tissue as acetochlor, alachlor and butachlor. Although propachlor does produce a precursor of a quinoneimine, the available data do not support its tumorigenicity to the nasal turbinates.</p> <p>Alachlor, acetochlor, metalochlor, and dimethenamid are potent skin sensitisers whilst alachlor and acetochlor exhibit mutagenic potential and significant oncogenic potential in both rats and mice.</p> <p>Chloroacetamides block the formation of very-long-chain fatty acids (VLCFA) by inhibiting fatty acid elongase. Vertebrates, including humans, have the biochemical mechanisms needed to synthesis long chain fatty acids including the enzyme long chain fatty acid elongase. Humans, however lack the desaturase enzymes that produce the health-promoting very long-chain polyunsaturated fatty acids synthesised by plants and fish. It is plausible that chloroacetamides could perturb fatty acid synthesis but there is no direct evidence of this in animal studies.</p> <p>[* The Pesticides Manual, Incorporating The Agrochemicals Handbook, 10th Edition, Editor Clive Tomlin, 1994, British Crop Protection Council]</p>
SOLVENT NAPHTHA PETROLEUM, HEAVY AROMATIC	<p>Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length, with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent than iso- or cyclo-paraffins.</p> <p>The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption, is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.</p> <p>for petroleum:</p> <p>Altered mental state, drowsiness, peripheral motor neuropathy, irreversible brain damage (so-called Petrol Sniffer's Encephalopathy), delirium, seizures, and sudden death have been reported from repeated overexposure to some hydrocarbon solvents, naphthas, and gasoline</p> <p>This product may contain benzene which is known to cause acute myeloid leukaemia and n-hexane which has been shown to metabolize to compounds which are neuropathic.</p> <p>This product contains toluene. There are indications from animal studies that prolonged exposure to high concentrations of toluene may lead to hearing loss.</p> <p>This product contains ethyl benzene and naphthalene from which there is evidence of tumours in rodents</p> <p>Carcinogenicity: Inhalation exposure to mice causes liver tumours, which are not considered relevant to humans. Inhalation exposure to rats causes kidney tumours which are not considered relevant to humans.</p> <p>Mutagenicity: There is a large database of mutagenicity studies on gasoline and gasoline blending streams, which use a wide variety of endpoints and give predominantly negative results. All in vivo studies in animals and recent studies in exposed humans (e.g. petrol service station attendants) have shown negative results in mutagenicity assays.</p> <p>Reproductive Toxicity: Repeated exposure of pregnant rats to high concentrations of toluene (around or exceeding 1000 ppm) can cause developmental effects, such as lower birth weight and developmental neurotoxicity, on the foetus. However, in a two-generation reproductive study in rats exposed to gasoline vapour condensate, no adverse effects on the foetus were observed.</p> <p>Human Effects: Prolonged/ repeated contact may cause defatting of the skin which can lead to dermatitis and may make the skin more susceptible to irritation and penetration by other materials.</p> <p>Lifetime exposure of rodents to gasoline produces carcinogenicity although the relevance to humans has been questioned. Gasoline induces kidney cancer in male rats as a consequence of accumulation of the alpha2-microglobulin protein in hyaline droplets in the male (but not female) rat kidney. Such abnormal accumulation represents lysosomal overload and leads to chronic renal tubular cell degeneration, accumulation of cell debris, mineralisation of renal medullary tubules and necrosis. A sustained regenerative proliferation occurs in epithelial cells with subsequent neoplastic transformation with continued exposure. The alpha2-microglobulin is produced under the influence of hormonal controls in male rats but not in females and, more importantly, not in humans.</p>
NONYLPHENOL, ETHOXYLATED, PROPOXYLATED	<p>For nonylphenol and its compounds:</p> <p>Alkylphenols like nonylphenol and bisphenol A have estrogenic effects in the body. They are known as xenoestrogens. Estrogenic substances and other endocrine disruptors are compounds that have hormone-like effects in both wildlife and humans. Xenoestrogens usually function by binding to estrogen receptors and acting competitively against natural estrogens. Nonylphenol has been found to act as an agonist of GPER (G protein-coupled estrogen receptor). Nonylphenol has been shown to mimic the natural hormone 17beta-estradiol, and it competes with the endogenous hormone for binding with the estrogen receptors ERalpha and ERbeta.</p> <p>Effects in pregnant women.</p> <p>Subcutaneous injections of nonylphenol in late pregnancy causes the expression of certain placental and uterine proteins,</p>

namely CaBP-9k, which suggest it can be transferred through the placenta to the fetus. It has also been shown to have a higher potency on the first trimester placenta than the endogenous estrogen 17 β -estradiol. In addition, early prenatal exposure to low doses of nonylphenol cause an increase in apoptosis (programmed cell death) in placental cells. These "low doses" ranged from 10-13-10-9 M, which is lower than what is generally found in the environment.

Nonylphenol has also been shown to affect cytokine signaling molecule secretions in the human placenta. In vitro cell cultures of human placenta during the first trimester were treated with nonylphenol, which increase the secretion of cytokines including interferon gamma, interleukin 4, and interleukin 10, and reduced the secretion of tumor necrosis factor alpha. This unbalanced cytokine profile at this part of pregnancy has been documented to result in implantation failure, pregnancy loss, and other complications.

Effects on metabolism

Nonylphenol has been shown to act as an obesity enhancing chemical or obesogen, though it has paradoxically been shown to have anti-obesity properties. Growing embryos and newborns are particularly vulnerable when exposed to nonylphenol because low-doses can disrupt sensitive processes that occur during these important developmental periods. Prenatal and perinatal exposure to nonylphenol has been linked with developmental abnormalities in adipose tissue and therefore in metabolic hormone synthesis and release. Specifically, by acting as an estrogen mimic, nonylphenol has generally been shown to interfere with hypothalamic appetite control. The hypothalamus responds to the hormone leptin, which signals the feeling of fullness after eating, and nonylphenol has been shown to both increase and decrease eating behavior by interfering with leptin signaling in the midbrain. Nonylphenol has been shown mimic the action of leptin on neuropeptide Y and anorectic POMC neurons, which has an anti-obesity effect by decreasing eating behavior. This was seen when estrogen or estrogen mimics were injected into the ventromedial hypothalamus. On the other hand, nonylphenol has been shown to increase food intake and have obesity enhancing properties by lowering the expression of these anorexigenic neurons in the brain. Additionally, nonylphenol affects the expression of ghrelin: an enzyme produced by the stomach that stimulates appetite. Ghrelin expression is positively regulated by estrogen signaling in the stomach, and it is also important in guiding the differentiation of stem cells into adipocytes (fat cells). Thus, acting as an estrogen mimic, prenatal and perinatal exposure to nonylphenol has been shown to increase appetite and encourage the body to store fat later in life. Finally, long-term exposure to nonylphenol has been shown to affect insulin signaling in the liver of adult male rats.

Cancer

Nonylphenol exposure has also been associated with breast cancer. It has been shown to promote the proliferation of breast cancer cells, due to its agonistic activity on ER α (estrogen receptor alpha) in estrogen-dependent and estrogen-independent breast cancer cells. Some argue that nonylphenol's suggested estrogenic effect coupled with its widespread human exposure could potentially influence hormone-dependent breast cancer disease.

No significant acute toxicological data identified in literature search.

Human beings have regular contact with alcohol ethoxylates through a variety of industrial and consumer products such as soaps, detergents, and other cleaning products. Exposure to these chemicals can occur through ingestion, inhalation, or contact with the skin or eyes. Studies of acute toxicity show that volumes well above a reasonable intake level would have to occur to produce any toxic response. Moreover, no fatal case of poisoning with alcohol ethoxylates has ever been reported. Multiple studies investigating the acute toxicity of alcohol ethoxylates have shown that the use of these compounds is of low concern in terms of oral and dermal toxicity.

Clinical animal studies indicate these chemicals may produce gastrointestinal irritation such as ulcerations of the stomach, pilo-erection, diarrhea, and lethargy. Similarly, slight to severe irritation of the skin or eye was generated when undiluted alcohol ethoxylates were applied to the skin and eyes of rabbits and rats. The chemical shows no indication of being a genotoxin, carcinogen, or mutagen (HERA 2007). No information was available on levels at which these effects might occur, though toxicity is thought to be substantially lower than that of nonylphenol ethoxylates.

Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.

Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-penta-oxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture.

On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing.

Alcohol ethoxylates are according to CESIO (2000) classified as Irritant or Harmful depending on the number of EO-units:

EO < 5 gives Irritant (Xi) with R38 (Irritating to skin) and R41 (Risk of serious damage to eyes)

EO > 5-15 gives Harmful (Xn) with R22 (Harmful if swallowed) - R38/41

EO > 15-20 gives Harmful (Xn) with R22-41

>20 EO is not classified (CESIO 2000)

Oxo-AE, C13 EO10 and C13 EO15, are Irritating (Xi) with R36/38 (Irritating to eyes and skin).

AE are not included in Annex 1 of the list of dangerous substances of the Council Directive 67/548/EEC

In general, alcohol ethoxylates (AE) are readily absorbed through the skin of guinea pigs and rats and through the gastrointestinal mucosa of rats. AE are quickly eliminated from the body through the urine, faeces, and expired air (CO₂). Orally dosed AE was absorbed rapidly and extensively in rats, and more than 75% of the dose was absorbed. When applied to the skin of humans, the doses were absorbed slowly and incompletely (50% absorbed in 72 hours). Half of the absorbed surfactant was excreted promptly in the urine and smaller amounts of AE appeared in the faeces and expired air (CO₂). The metabolism of C12 AE yields PEG, carboxylic acids, and CO₂ as metabolites. The LD₅₀ values after oral administration to rats range from about 1-15 g/kg body weight indicating a low to moderate acute toxicity.

The ability of nonionic surfactants to cause a swelling of the stratum corneum of guinea pig skin has been studied. The swelling

AC Menace 960 Herbicide

mechanism of the skin involves a combination of ionic binding of the hydrophilic group as well as hydrophobic interactions of the alkyl chain with the substrate. One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been established that there is a connection between the potential of surfactants to denature protein *in vitro* and their effect on the skin. Nonionic surfactants do not carry any net charge and, therefore, they can only form hydrophobic bonds with proteins. For this reason, proteins are not deactivated by nonionic surfactants, and proteins with poor solubility are not solubilized by nonionic surfactants. A substantial amount of toxicological data and information *in vivo* and *in vitro* demonstrates that there is no evidence for alcohol ethoxylates (AEs) being genotoxic, mutagenic or carcinogenic. No adverse reproductive or developmental effects were observed. The majority of available toxicity studies revealed NOAELs in excess of 100 mg/kg bw/d but the lowest NOAEL for an individual AE was established to be 50 mg/kg bw/day. This value was subsequently considered as a conservative, representative value in the risk assessment of AE. The effects were restricted to changes in organ weights with no histopathological organ changes with the exception of liver hypertrophy (indicative of an adaptive response to metabolism rather than a toxic effect). It is noteworthy that there was practically no difference in the NOAEL in oral studies of 90-day or 2 years of duration in rats. A comparison of the aggregate consumer exposure and the systemic NOAEL (taking into account an oral absorption value of 75%) results in a Margin of Exposure of 5,800. Taking into account the conservatism in the exposure assessment and the assigned systemic NOAEL, this margin of exposure is considered more than adequate to account for the inherent uncertainty and variability of the hazard database and inter and intra-species extrapolations.

AEs are not contact sensitizers. Neat AE are irritating to eyes and skin. The irritation potential of aqueous solutions of AEs depends on concentrations. Local dermal effects due to direct or indirect skin contact in certain use scenarios where the products are diluted are not of concern as AEs are not expected to be irritating to the skin at in-use concentrations. Potential irritation of the respiratory tract is not a concern given the very low levels of airborne AE generated as a consequence of spray cleaner aerosols or laundry powder detergent dust.

In summary, the human health risk assessment has demonstrated that the use of AE in household laundry and cleaning detergents is safe and does not cause concern with regard to consumer use.

For high boiling ethylene glycol ethers (typically triethylene- and tetraethylene glycol ethers):

Skin absorption: Available skin absorption data for triethylene glycol ether (TGBE), triethylene glycol methyl ether (TGME), and triethylene glycol ethylene ether (TGEE) suggest that the rate of absorption in skin of these three glycol ethers is 22 to 34 micrograms/cm²/hr, with the methyl ether having the highest permeation constant and the butyl ether having the lowest. The rates of absorption of TGBE, TGEE and TGME are at least 100-fold less than EGME, EGEE, and EGBE, their ethylene glycol monoalkyl ether counterparts, which have absorption rates that range from 214 to 2890 micrograms/cm²/hr. Therefore, an increase in either the chain length of the alkyl substituent or the number of ethylene glycol moieties appears to lead to a decreased rate of percutaneous absorption. However, since the ratio of the change in values of the ethylene glycol to the diethylene glycol series is larger than that

of the diethylene glycol to triethylene glycol series, the effect of the length of the chain and number of ethylene glycol moieties on absorption diminishes with an increased number of ethylene glycol moieties. Therefore, although tetraethylene glycol methyl ether (TetraME) and tetraethylene glycol butyl ether (TetraBE) are expected to be less permeable to skin than TGME and TGBE, the differences in permeation between these molecules may only be slight.

Metabolism: The main metabolic pathway for metabolism of ethylene glycol monoalkyl ethers (EGME, EGEE, and EGBE) is oxidation via alcohol and aldehyde dehydrogenases (ALD/ADH) that leads to the formation of an alkoxy acids. Alkoxy acids are the only toxicologically significant metabolites of glycol ethers that have been detected *in vivo*. The principal metabolite of TGME is believed to be 2-[2-(2-methoxyethoxy)ethoxy] acetic acid. Although ethylene glycol, a known kidney toxicant, has been identified as an impurity or a minor metabolite of glycol ethers in animal studies it does not appear to contribute to the toxicity of glycol ethers.

The metabolites of category members are not likely to be metabolized to any large extent to toxic molecules such as ethylene glycol or the mono alkoxy acids because metabolic breakdown of the ether linkages also has to occur

Acute toxicity: Category members generally display low acute toxicity by the oral, inhalation and dermal routes of exposure. Signs of toxicity in animals receiving lethal oral doses of TGBE included loss of righting reflex and flaccid muscle tone, coma, and heavy breathing. Animals administered lethal oral doses of TGEE exhibited lethargy, ataxia, blood in the urogenital area and piloerection before death.

Irritation: The data indicate that the glycol ethers may cause mild to moderate skin irritation. TGEE and TGBE are highly irritating to the eyes. Other category members show low eye irritation.

Repeat dose toxicity: Results of these studies suggest that repeated exposure to moderate to high doses of the glycol ethers in this category is required to produce systemic toxicity

In a 21-day dermal study, TGME, TGEE, and TGBE were administered to rabbits at 1,000 mg/kg/day. Erythema and oedema were observed. In addition, testicular degeneration (scored as trace in severity) was observed in one rabbit given TGEE and one rabbit given TGME. Testicular effects included spermatid giant cells, focal tubular hypospermatogenesis, and increased cytoplasmic vacuolisation. Due to a high incidence of similar spontaneous changes in normal New Zealand White rabbits, the testicular effects were considered not to be related to treatment. Thus, the NOAELs for TGME, TGEE and TGBE were established at 1000 mg/kg/day. Findings from this report were considered unremarkable.

A 2-week dermal study was conducted in rats administered TGME at doses of 1,000, 2,500, and 4,000 mg/kg/day. In this study, significantly-increased red blood cells at 4,000 mg/kg/day and significantly-increased urea concentrations in the urine at 2,500 mg/kg/day were observed. A few of the rats given 2,500 or 4,000 mg/kg/day had watery caecal contents and/or haemolysed blood in the stomach. These gross pathologic observations were not associated with any histologic abnormalities in these tissues or alterations in haematologic and clinical chemistry parameters. A few males and females treated with either 1,000 or 2,500 mg/kg/day had a few small scabs or crusts at the test site. These alterations were slight in degree and did not adversely affect the rats.

In a 13-week drinking water study, TGME was administered to rats at doses of 400, 1,200, and 4,000 mg/kg/day. Statistically-significant changes in relative liver weight were observed at 1,200 mg/kg/day and higher. Histopathological effects included hepatocellular cytoplasmic vacuolisation (minimal to mild in most animals) and hypertrophy (minimal to mild) in males at all doses and hepatocellular hypertrophy (minimal to mild) in high dose females. These effects were statistically significant at 4,000

mg/kg/day. Cholangiofibrosis was observed in 7/15 high-dose males; this effect was observed in a small number of bile ducts and was of mild severity. Significant, small decreases in total test session motor activity were observed in the high-dose animals, but no other neurological effects were observed. The changes in motor activity were secondary to systemic toxicity

Mutagenicity: Mutagenicity studies have been conducted for several category members. All in vitro and in vivo studies were negative at concentrations up to 5,000 micrograms/plate and 5,000 mg/kg, respectively, indicating that the category members are not genotoxic at the concentrations used in these studies. The uniformly negative outcomes of various mutagenicity studies performed on category members lessen the concern for carcinogenicity.

Reproductive toxicity: Although mating studies with either the category members or surrogates have not been performed, several of the repeated dose toxicity tests with the surrogates have included examination of reproductive organs. A lower molecular weight glycol ether, ethylene glycol methyl ether (EGME), has been shown to be a testicular toxicant. In addition, results of repeated dose toxicity tests with TGME clearly show testicular toxicity at an oral dose of 4,000 mg/kg/day four times greater than the limit dose of 1,000 mg/kg/day recommended for repeat dose studies. It should be noted that TGME is 350 times less potent for testicular effects than EGME. TGME is not associated with testicular toxicity, TetraME is not likely to be metabolised by any large extent to 2-MAA (the toxic metabolite of EGME), and a mixture containing predominantly methylated glycol ethers in the C5-C11 range does not produce testicular toxicity (even when administered intravenously at 1,000 mg/kg/day).

Developmental toxicity: The bulk of the evidence shows that effects on the foetus are not noted in treatments with 1,000 mg/kg/day during gestation. At 1,250 to 1,650 mg/kg/day TGME (in the rat) and 1,500 mg/kg/day (in the rabbit), the developmental effects observed included skeletal variants and decreased body weight gain.

for nonylphenol:

Nonylphenol was studied for oral toxicity in rats in a 28-day repeat dose toxicity test at doses of 0, 4, 15, 60 and 250 mg/kg/day. Changes suggesting renal dysfunction were mainly noted in both sexes given 250 mg/kg. Liver weights were increased in males given 60 mg/kg and in both sexes given 250 mg/kg group. Histopathologically, hypertrophy of the centrilobular hepatocytes was noted in both sexes given 250 mg/kg. Kidney weights were increased in males given 250 mg/kg and macroscopically, disseminated white spots, enlargement and pelvic dilatation were noted in females given 250 mg/kg. Histopathologically, the following lesions were noted in the 250 mg/kg group: basophilic change of the proximal tubules in both sexes, single cell necrosis of the proximal tubules, inflammatory cell infiltration in the interstitium and casts in females, basophilic change and dilatation of the collecting tubules in both sexes, simple hyperplasia of the pelvic mucosa and pelvic dilatation in females. In the urinary bladder, simple hyperplasia was noted in both sexes given 250 mg/kg. In the caecum, macroscopic dilatation was noted in both sexes given 250 mg/kg. Almost all changes except those in the kidney disappeared after a 14-day recovery period. The NOELs for males and females are considered to be 15 mg/kg/day and 60 mg/kg/day, respectively, under the conditions of the present study.

Nonylphenol was not mutagenic to *Salmonella typhimurium*, TA100, TA1535, TA98, TA1537 and *Escherichia coli* WP2 uvrA, with or without an exogenous metabolic activation system.

Nonylphenol induced neither structural chromosomal aberrations nor polyploidy in CHL/IU cells, in the absence or presence of an exogenous metabolic activation system.

TALLOW ALKYLAMINE, ETHOXYLATED

Alkyl amine polyalkoxylates are not acutely toxic by the oral and dermal routes of exposure, or via inhalation under normal use conditions. Concentrated materials are generally corrosive, eye and skin irritants and may be dermal sensitizers. There is no evidence that alkyl amine polyalkoxylates are neurotoxic, mutagenic, or clastogenic.

Surfactants are surface-active materials that can damage the structural integrity of cellular membranes at high dose levels. Thus, surfactants are often corrosive and irritating in concentrated solutions, as indicated by the acute toxicity studies for these inert materials. It is possible that some of the observed toxicity seen in the repeated studies, such as diarrhea or decreased body weight gain, can be attributed to the corrosive and irritating nature of these surfactants.

Generally, lower molecular weight AAPs (lower carbon chain units and less alkoxylation) may potentially be more bioavailable because they may be more easily absorbed and distributed than higher molecular weight compounds. Thus overall, the longer chain carbon amine higher polyalkoxylates should be less bioavailable.

There are no dermal absorption data on the AAPs. However, data on functionally and structurally similar surfactants suggest that dermal absorption of the AAPs is likely to be low.

Following subchronic exposure to rats, some gastrointestinal irritation was observed, but no specific target organ toxicity or neurotoxicity was seen. In subchronic studies in rats and/or dogs, the most sensitive effects noted were increased mortality, clinical signs (salivation, wheezing, emesis, and/or soft faeces), cataracts, cellular changes in the stomach, and liver effects characterized by enzyme induction, and pigment accumulation in Kupffer cells and bile canaliculi. There was no increased susceptibility to the offspring of rats following in utero exposure in two prenatal developmental toxicity studies. However, there is evidence of increased susceptibility in a reproductive screening study in rats.

In rat developmental studies, no adverse fetal effects were seen, even at maternally toxic doses. No effects were observed on estrous cyclicity, spermatogenic endpoints, or testosterone and thyroid levels in a two-generation rat reproduction study.

However, reproductive and offspring toxicity were noted for AAPs based on litter loss, increase mean number of unaccounted-for implantation sites and decreased mean number of pups born, live litter size and postnatal survival from birth to LD 4.

Very little metabolism information is available for the alkyl amine polyalkoxylates. However, it is possible to predict mammalian metabolism based on studies for the alkyl alcohol alkoxyates, which are another class of surfactants. It has been proposed that the primary metabolic pathway involves the excretion of the polyalkoxylate moiety and conversion of the alkyl amine group to a fatty acid that is then converted via oxidative degradation to carbon dioxide and water. In general, the gastrointestinal absorption of AAPs with relatively short alkoxyate chain lengths is expected to be rapid and extensive, while less absorption is likely for the more extensively polyalkoxylated AAPs with larger molecular weights.

No structural alerts for potential carcinogenicity of both a representative alkyl amine polyalkoxylate, as well as a possible metabolite/degradate of alkyl amine polyalkoxylate that had been extensively dealkylated, with the amine group intact have been identified. Alkyl amine polyalkoxylates are not expected to be carcinogenic. Therefore a cancer dietary exposure assessment is not necessary to assess cancer risk.

The US EPA has not found alkyl amine polyalkoxylates to share a common mechanism of toxicity with any other substances, and alkyl amine polyalkoxylates do not appear to produce a toxic metabolite produced by other substances. For the purposes of this tolerance action, therefore, EPA has assumed that alkyl amine polyalkoxylates do not have a common mechanism of toxicity with

other substances

Alkyl Amine Polyalkoxylates (JITF CST 4 Inert Ingredients). Human Health Risk Assessment to Support Proposed Exemption from the Requirement of a Tolerance When Used as Inert Ingredients in Pesticide Formulations. June 2009
<http://beta.regulations.gov/document/EPA-HQ-OPP-2008-0738-0005>

For Fatty Nitrogen-Derived ether amines and Fatty Nitrogen-derived amines (FND ether amines and FND amines):

FND ether amines and FND amines are very similar in structure and function. . The minimal difference among the alkyl substituents and the large database for the FND categories indicates that the structural differences in these large alkyl chains do not result in differences in toxicity or mutagenicity.

The differences in chain length, degree of saturation of the carbon chains, source of the natural oils, or addition of an amino group in the chain would not be expected to have an impact on the toxicity profile. This conclusion is supported by a number of studies in the FND family of chemicals (amines, cationics, and amides as separate categories) that show no differences in the length or degree of saturation of the alkyl substituents and is also supported by the limited toxicity of these long-chain substituted chemicals

The available acute oral LD50 study for the propanamine derivative with the extensive data for the other supporting chemicals provides adequate evidence that the FND ether amines are only moderately to slightly toxic via this route and exposure period. Acute dermal studies for the supporting chemicals indicate these chemicals can be classified as minimally toxic. Acute inhalation studies did not result in deaths under normal exposure conditions for two chemicals. Repeated dose toxicity studies had similar NOAELs (12.5 to 50 mg/kg/day for rats and 3 or 13 mg/kg/day for dogs). Importantly because the highest exposure potential for some of the FND ether amines is via skin contact, a number of repeat dose dermal studies indicate the chemicals are highly irritating.

No clear organ-specific toxicity occurred in any of the repeat dose studies with the supporting chemicals in the FND ether amines category. In addition, available data indicate that the FND ether amines are unlikely to be mutagenic and that they are not reproductive or developmental toxins

In evaluating potential toxicity of the FND Amines chemicals, it is also useful to review the available data for the related FND Cationic and FND Amides Category chemicals. Acute oral toxicity studies (approximately 80 studies for 40 chemicals in the three categories) provide LD50 values from approximately 400 to 10,000 mg/kg with no apparent organ specific toxicity. Similarly, repeated dose toxicity studies (approximately 35 studies for 15 chemicals) provide NOAELs between 10 and 100 mg/kg/day for rats and slightly lower for dogs. More than 60 genetic toxicity studies (*in vitro* bacterial and mammalian cells as well as *in vivo* studies) indicated no mutagenic activity among more than 30 chemicals tested. For reproductive evaluations, 14 studies evaluated reproductive endpoints and/or reproductive organs for 11 chemicals, and 15 studies evaluated developmental toxicity for 13 chemicals indicating no reproductive or developmental effects for the FND group as a whole.

The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

Tallow derivatives used in the manufacture of cosmetic products such as fatty acids, glycerol, fatty acid esters and soap are regarded as safe if they are obtained by the following minimal processes which must be strictly certified:

- transesterification or hydrolysis at 200°C, under pressure for 20 minutes (glycerol and fatty acids and esters)
- saponification with NaOH 12M (glycerol and soap)

* batch process: at 95°C for 3 hours

* continuous process: at 140°C, 2 bars for 8 minutes or equivalent.

Moreover, other tallow derivatives (e.g. fatty alcohols, fatty amines, fatty amides) produced from the above mentioned and submitted to further processes are regarded as safe.

Opinion of The Scientific Committee on Cosmetic Products and Non-Food Products intended for Consumers concerning Tallow Derivatives revised and adapted opinion of 24.6.97 adopted by the plenary session of the SCCNFP of 23 September 1998

None of the constituents of tallow were toxic through oral and dermal exposure, they were not ocular or dermal irritants, and they were neither dermal sensitizers nor photosensitizers. The same was true for other oils which contain varying concentrations of the constituents of tallow.

Based on the CIR safety evaluations of the individual constituents of tallow and of cosmetic ingredients containing the constituents of tallow, and on the approval of tallow for use in foods and other consumer products, it is concluded that tallow, tallow glyceride, tallow glycerides, hydrogenated tallow glyceride, and hydrogenated tallow glycerides are safe as cosmetic ingredients in the present practices of use

Cosmetic Ingredient Review Expert Panel

While it is difficult to generalise about the full range of potential health effects posed by exposure to the many different amine compounds, characterised by those used in the manufacture of polyurethane and polyisocyanurate foams, it is agreed that overexposure to the majority of these materials may cause adverse health effects.

- ▶ Many amine-based compounds can induce histamine liberation, which, in turn, can trigger allergic and other physiological effects, including bronchoconstriction or bronchial asthma and rhinitis.
- ▶ Systemic symptoms include headache, nausea, faintness, anxiety, a decrease in blood pressure, tachycardia (rapid heartbeat), itching, erythema (reddening of the skin), urticaria (hives), and facial edema (swelling). Systemic effects (those affecting the body) that are related to the pharmacological action of amines are usually transient.

Typically, there are four routes of possible or potential exposure: inhalation, skin contact, eye contact, and ingestion.

Inhalation:

Inhalation of vapors may, depending upon the physical and chemical properties of the specific product and the degree and length of exposure, result in moderate to severe irritation of the tissues of the nose and throat and can irritate the lungs.

Products with higher vapour pressures have a greater potential for higher airborne concentrations. This increases the probability of worker exposure.

Higher concentrations of certain amines can produce severe respiratory irritation, characterised by nasal discharge, coughing, difficulty in breathing, and chest pains.

Chronic exposure via inhalation may cause headache, nausea, vomiting, drowsiness, sore throat, bronchopneumonia, and possible lung damage. Also, repeated and/or prolonged exposure to some amines may result in liver disorders, jaundice, and liver enlargement. Some amines have been shown to cause kidney, blood, and central nervous system disorders in laboratory animal studies.

AC Menace 960 Herbicide

While most polyurethane amine catalysts are not sensitizers, some certain individuals may also become sensitized to amines and may experience respiratory distress, including asthma-like attacks, whenever they are subsequently exposed to even very small amounts of vapor. Once sensitized, these individuals must avoid any further exposure to amines. Although chronic or repeated inhalation of vapor concentrations below hazardous or recommended exposure limits should not ordinarily affect healthy individuals, chronic overexposure may lead to permanent pulmonary injury, including a reduction in lung function, breathlessness, chronic bronchitis, and immunologic lung disease.

Inhalation hazards are increased when exposure to amine catalysts occurs in situations that produce aerosols, mists, or heated vapors. Such situations include leaks in fitting or transfer lines. Medical conditions generally aggravated by inhalation exposure include asthma, bronchitis, and emphysema.

Skin Contact:

Skin contact with amine catalysts poses a number of concerns. Direct skin contact can cause moderate to severe irritation and injury i.e., from simple redness and swelling to painful blistering, ulceration, and chemical burns. Repeated or prolonged exposure may also result in severe cumulative dermatitis.

Skin contact with some amines may result in allergic sensitization. Sensitized persons should avoid all contact with amine catalysts. Systemic effects resulting from the absorption of the amines through skin exposure may include headaches, nausea, faintness, anxiety, decrease in blood pressure, reddening of the skin, hives, and facial swelling. These symptoms may be related to the pharmacological action of the amines, and they are usually transient.

Eye Contact:

Amine catalysts are alkaline in nature and their vapours are irritating to the eyes, even at low concentrations.

Direct contact with the liquid amine may cause severe irritation and tissue injury, and the "burning" may lead to blindness.

(Contact with solid products may result in mechanical irritation, pain, and corneal injury.)

Exposed persons may experience excessive tearing, burning, conjunctivitis, and corneal swelling.

The corneal swelling may manifest itself in visual disturbances such as blurred or "foggy" vision with a blue tint ("blue haze") and sometimes a halo phenomenon around lights. These symptoms are transient and usually disappear when exposure ceases.

Some individuals may experience this effect even when exposed to concentrations below doses that ordinarily cause respiratory irritation.

Ingestion:

The oral toxicity of amine catalysts varies from moderately to very toxic.

Some amines can cause severe irritation, ulceration, or burns of the mouth, throat, esophagus, and gastrointestinal tract.

Material aspirated (due to vomiting) can damage the bronchial tubes and the lungs.

Affected persons also may experience pain in the chest or abdomen, nausea, bleeding of the throat and the gastrointestinal tract, diarrhea, dizziness, drowsiness, thirst, circulatory collapse, coma, and even death.

Polyurethane Amine Catalysts: Guidelines for Safe Handling and Disposal; Technical Bulletin June 2000**Alliance for Polyurethanes Industry**

Most undiluted cationic surfactants satisfy the criteria for classification as Harmful (Xn) with R22 and as Irritant (Xi) for skin and eyes with R38 and R41.

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergic 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.

Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.

Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture.

On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autooxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing.

Allergic Contact Dermatitis—Formation, Structural Requirements, and Reactivity of Skin Sensitizers.

Ann-Therese Karlberg et al; Chem. Res. Toxicol. 2008, 21, 53-69

Polyethylene glycols (PEGs) have a wide variety of PEG-derived mixtures due to their readily linkable terminal primary hydroxyl groups in combination with many possible compounds and complexes such as ethers, fatty acids, castor oils, amines, propylene glycols, among other derivatives. PEGs and their derivatives are broadly utilized in cosmetic products as surfactants, emulsifiers, cleansing agents, humectants, and skin conditioners.

PEGs and PEG derivatives were generally regulated as safe for use in cosmetics, with the conditions that impurities and by-products, such as ethylene oxides and 1,4-dioxane, which are known carcinogenic materials, should be removed before they are mixed in cosmetic formulations.

Most PEGs are commonly available commercially as mixtures of different oligomer sizes in broadly- or narrowly-defined molecular weight (MW) ranges. For instance, PEG-10,000 typically designates a mixture of PEG molecules (n = 195 to 265) having an average MW of 10,000. PEG is also known as polyethylene oxide (PEO) or polyoxyethylene (POE), with the three

**NONYLPHENOL,
ETHOXYLATED,
PROPOXYLATED &
TALLOW ALKYLAMINE,
ETHOXYLATED**

AC Menace 960 Herbicide

names being chemical synonyms. However, PEGs mainly refer to oligomers and polymers with molecular masses below 20,000 g/mol, while PEOs are polymers with molecular masses above 20,000 g/mol, and POEs are polymers of any molecular mass. Relatively small molecular weight PEGs are produced by the chemical reaction between ethylene oxide and water or ethylene glycol (or other ethylene glycol oligomers), as catalyzed by acidic or basic catalysts. To produce PEO or high-molecular weight PEGs, synthesis is performed by suspension polymerization. It is necessary to hold the growing polymer chain in solution during the course of the poly-condensation process. The reaction is catalyzed by magnesium-, aluminum-, or calcium-organoelement compounds. To prevent coagulation of polymer chains in the solution, chelating additives such as dimethylglyoxime are used

Safety Evaluation of Polyethylene Glycol (PEG) Compounds for Cosmetic Use: Toxicol Res 2015; 31:105-136 The Korean Society of Toxicology

<http://doi.org/10.5487/TR.2015.31.2.105>

Acute Toxicity	✓	Carcinogenicity	✗
Skin Irritation/Corrosion	✓	Reproductivity	✓
Serious Eye Damage/Irritation	✓	STOT - Single Exposure	✗
Respiratory or Skin sensitisation	✓	STOT - Repeated Exposure	✗
Mutagenicity	✗	Aspiration Hazard	✗

Legend: ✗ – Data either not available or does not fill the criteria for classification
 ✓ – Data available to make classification

SECTION 12 Ecological information

Toxicity

AC Menace 960 Herbicide	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
metolachlor	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	120h	Algae or other aquatic plants	0.001mg/l	1
	LC50	96h	Fish	3.696-5.152mg/L	4
	EC50	72h	Algae or other aquatic plants	63.952mg/L	4
	EC50	48h	Crustacea	4.25mg/l	4
	EC50	96h	Algae or other aquatic plants	0.078mg/L	4
solvent naphtha petroleum, heavy aromatic	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	48h	Crustacea	0.95mg/l	1
	LC50	96h	Fish	0.58mg/l	2
	EC50	72h	Algae or other aquatic plants	<1mg/l	1
	EC50	48h	Crustacea	0.95mg/l	1
	EC50	96h	Algae or other aquatic plants	1mg/l	2
nonylphenol, ethoxylated, propoxylated	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
tallow alkylamine, ethoxylated	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	48h	Crustacea	3.7-7.2mg/l	4
	LC50	96h	Fish	>0.44mg/l	4
	EC50	48h	Crustacea	3.7-7.2mg/l	4
Legend:	Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data				

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

Do NOT 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.

for (chloro)acetanilide (syn: chloroacetamide) pesticides:

Alachlor, metolachlor and acetochlor are degradable in top soil in aquatic environments and to some extent also in groundwater. One study described a 17-57%

Continued...

decrease in alachlor concentrations after 138 days in four out of 81 aquifer sediment samples incubated aerobically. Degradation products were more stable than the mother compound under groundwater conditions. Sulfonic and oxanilic acid metabolites contribute to the total amount of the acetanilide pesticides, detected from 3 to 45 times more frequently than the parent compounds and in concentrations substantially higher.

Soil adsorption seems to be governed by organic matter content (e.g. humic acids) of the soil

Acetanilide pesticides, e.g. alachlor, are subject to glutathione conjugation in higher organisms like plants and insects. The process results in the formation of ethanesulfonic and oxalinic acids. Sulfonic and oxanilic acid metabolites contribute to the total amount of the acetanilide.

Aromatic amines (arylamines), particularly primary aromatic amines, covalently and irreversibly bind to humic substances present in most natural waters.

All metabolites with moieties of: anilines, benzidines and toluidines are of environmental concern. Anilines and benzidines are both acutely toxic and toxic depending on the specific aquatic species (except algae). Toluidines represent a similar concern. It has been speculated that aqueous solutions of aromatic amines can be oxidised by organic radicals, but there are no actual data on reaction rates. Based on a study of reaction rate data for these compounds an estimate of the half-life of aromatic amines in water is approximately 100 days, assuming a peroxy radical concentration of 10-10 mole/L in sunlit, oxygenated water

DO NOT discharge into sewer or waterways.

Persistence and degradability

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

Bioaccumulative potential

Ingredient	Bioaccumulation
metolachlor	LOW (BCF = 69)
solvent naphtha petroleum, heavy aromatic	LOW (BCF = 159)

Mobility in soil

Ingredient	Mobility
metolachlor	LOW (KOC = 291.6)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	
	<ul style="list-style-type: none"> Recycle wherever possible or consult manufacturer for recycling options. Consult State Land Waste Authority for disposal. Bury or incinerate residue at an approved site. Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information

Labels Required

Marine Pollutant	
HAZCHEM	•3Z

Land transport (ADG)

UN number	3082				
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains metolachlor)				
Transport hazard class(es)	<table> <tr> <td>Class</td><td>9</td></tr> <tr> <td>Subrisk</td><td>Not Applicable</td></tr> </table>	Class	9	Subrisk	Not Applicable
Class	9				
Subrisk	Not Applicable				
Packing group	III				

Environmental hazard	Environmentally hazardous	
Special precautions for user	Special provisions	274 331 335 375 AU01
	Limited quantity	5 L

Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082

are not subject to this Code when transported by road or rail in;

(a) packagings;

(b) IBCs; or

(c) any other receptacle not exceeding 500 kg(L).

- Australian Special Provisions (SP AU01) - ADG Code 7th Ed.

Air transport (ICAO-IATA / DGR)

UN number	3082	
UN proper shipping name	Environmentally hazardous substance, liquid, n.o.s. * (contains metolachlor)	
Transport hazard class(es)	ICAO/IATA Class	9
	ICAO / IATA Subrisk	Not Applicable
	ERG Code	9L
Packing group	III	
Environmental hazard	Environmentally hazardous	
Special precautions for user	Special provisions	A97 A158 A197 A215
	Cargo Only Packing Instructions	964
	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 metolachlor)	
Transport hazard class(es)	IMDG Class	9
	IMDG Subrisk	Not Applicable
Packing group	III	
Environmental hazard	Marine Pollutant	
Special precautions for user	EMS Number	F-A, S-F
	Special provisions	274 335 969
	Limited Quantities	5 L

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

Not Applicable

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

Product name	Group
metolachlor	Not Available
solvent naphtha petroleum, heavy aromatic	Not Available
nonylphenol, ethoxylated, propoxylated	Not Available
tallow alkylamine, ethoxylated	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
metolachlor	Not Available
solvent naphtha petroleum, heavy aromatic	Not Available
nonylphenol, ethoxylated, propoxylated	Not Available
tallow alkylamine, ethoxylated	Not Available

SECTION 15 Regulatory information

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

metolachlor is found on the following regulatory lists

Australia Chemicals with non-industrial uses removed from the Australian Inventory of Chemical Substances (old Inventory)

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 7

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

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

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

nonylphenol, ethoxylated, propoxylated is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

tallow alkylamine, ethoxylated is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

Australian Inventory of Industrial Chemicals (AIIC)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	No (metolachlor)
Canada - NDSL	No (metolachlor; solvent naphtha petroleum, heavy aromatic; nonylphenol, ethoxylated, propoxylated; tallow alkylamine, ethoxylated)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (nonylphenol, ethoxylated, propoxylated)
Japan - ENCS	No (metolachlor; nonylphenol, ethoxylated, propoxylated)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (metolachlor)
USA - TSCA	No (metolachlor)
Taiwan - TCSI	Yes
Mexico - INSEQ	No (nonylphenol, ethoxylated, propoxylated)
Vietnam - NCI	Yes
Russia - FBEPH	No (nonylphenol, ethoxylated, propoxylated)
Legend:	<p>Yes = All CAS declared ingredients are on the inventory</p> <p>No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.</p>

SECTION 16 Other information

Revision Date	20/08/2021
Initial Date	10/03/2017

SDS Version Summary

Version	Date of Update	Sections Updated
4.1	03/09/2020	Classification change due to full database hazard calculation/update.
5.1	20/08/2021	Classification change due to full database hazard calculation/update.

Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.

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

Definitions and abbreviations

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

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