Chemical™ Concepts
Our expertise is your solution.

chemical-concepts.com
800.220.1966
410 Pike Road • Huntingdon Valley, PA 19006

AW307 Clear

Safety Data Sheet according to OSHA HazCom Standard (2012) requirements

Chemwatch Hazard Alert Code: **4**

Issue Date:**07/11/2022**
Print Date: **09/21/2022**
L.GHS.USA.EN

SECTION 1 Identification

Product Identifier

Product name	AW307 Clear
Synonyms	Not Available
Proper shipping name	Adhesives, containing a flammable liquid
Other means of identification	Not Available

Recommended use of the chemical and restrictions on use

Relevant identified uses	Adhesive

Manufacturer	**Chemical Concepts, Inc.**
Address	**410 Pike Road Huntingdon Valley, PA 19006**
Information Telephone Number	**800.220.1966**
Emergency Contact Number:	**800-535-5053 (INFOTRAC)**
Recommended Use	Adhesive

SECTION 2 Hazard(s) identification

Classification of the substance or mixture

Note: The hazard category numbers found in GHS classification in section 2 of this SDSs are NOT to be used to fill in the NFPA 704 diamond. Blue = Health Red = Fire Yellow = Reactivity White = Special (Oxidizer or water reactive substances)

Classification	Serious Eye Damage/Eye Irritation Category 2A, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Hazardous to the Aquatic Environment Long-Term Hazard Category 2, Specific Target Organ Toxicity - Repeated Exposure Category 2, Flammable Liquids Category 2, Skin Corrosion/Irritation Category 2, Reproductive Toxicity Category 2, Sensitisation (Skin) Category 1, Aspiration Hazard Category 1

Label elements

Hazard pictogram(s)	
Signal word	**Danger**

Hazard statement(s)

H319	Causes serious eye irritation.

H336	May cause drowsiness or dizziness.
H411	Toxic to aquatic life with long lasting effects.
H373	May cause damage to organs through prolonged or repeated exposure.
H225	Highly flammable liquid and vapour.
H315	Causes skin irritation.
H361	Suspected of damaging fertility or the unborn child.
H317	May cause an allergic skin reaction.
H304	May be fatal if swallowed and enters airways.

Hazard(s) not otherwise classified

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P233	Keep container tightly closed.
P260	Do not breathe mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P240	Ground/bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use only non-sparking tools.
P243	Take precautionary measures against static discharge.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P202	Do not handle until all safety precautions have been read and understood.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing must not be allowed out of the workplace.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.
P331	Do NOT induce vomiting.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam to extinguish.
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.
P314	Get medical advice/attention 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.
P391	Collect spillage.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water/shower.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

P403+P235	Store in a well-ventilated place. Keep cool.
P405	Store locked up.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

Precautionary statement(s) Disposal

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

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
79-20-9	30.0 – 60.0	methyl acetate

Continued...

CAS No	%[weight]	Name
110-54-3	5.0 – 10.0	n-hexane
64742-49-0.	3.0 – 7.0	naphtha petroleum, light, hydrotreated
110-82-7	0.5 – 1.5	cyclohexane
68515-02-6	5.0 – 10.0	rosin/ isophthalic acid/ pentaerythritol
1675-54-3	0.1 – 1.0	bisphenol A diglycidyl ether

The specific chemical identity and/or exact percentage (concentration) of composition has been withheld as a trade secret.

SECTION 4 First-aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Wash out immediately with fresh running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation. For thermal burns: ▸ Decontaminate area around burn. ▸ Consider the use of cold packs and topical antibiotics. For first-degree burns (affecting top layer of skin) ▸ Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides. ▸ Use compresses if running water is not available. ▸ Cover with sterile non-adhesive bandage or clean cloth. ▸ Do NOT apply butter or ointments; this may cause infection. ▸ Give over-the counter pain relievers if pain increases or swelling, redness, fever occur. For second-degree burns (affecting top two layers of skin) ▸ Cool the burn by immerse in cold running water for 10-15 minutes. ▸ Use compresses if running water is not available. ▸ Do NOT apply ice as this may lower body temperature and cause further damage. ▸ Do NOT break blisters or apply butter or ointments; this may cause infection. ▸ Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape. To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort): ▸ Lay the person flat. ▸ Elevate feet about 12 inches. ▸ Elevate burn area above heart level, if possible. ▸ Cover the person with coat or blanket. ▸ Seek medical assistance. For third-degree burns Seek immediate medical or emergency assistance. In the mean time: ▸ Protect burn area cover loosely with sterile, nonstick bandage or, for large areas, a sheet or other material that will not leave lint in wound. ▸ Separate burned toes and fingers with dry, sterile dressings. ▸ Do not soak burn in water or apply ointments or butter; this may cause infection. ▸ To prevent shock see above. ▸ For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway. ▸ Have a person with a facial burn sit up. ▸ Check pulse and breathing to monitor for shock until emergency help arrives.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay.
Ingestion	▸ Immediately give a glass of water. ▸ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor. ▸ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

Most important symptoms and effects, both acute and delayed

See Section 11

Indication of any immediate medical attention and special treatment needed

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

BASIC TREATMENT

- Establish a patent airway with suction where necessary.
- Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- Administer oxygen by non-rebreather mask at 10 to 15 l/min.

Continued...

- Monitor and treat, where necessary, for pulmonary oedema .
- Monitor and treat, where necessary, for shock.
- **DO NOT** use emetics. Where ingestion is suspected rinse mouth and give up to 200 ml water (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.
- Give activated charcoal.

ADVANCED TREATMENT

- Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- Positive-pressure ventilation using a bag-valve mask might be of use.
- Monitor and treat, where necessary, for arrhythmias.
- Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- Drug therapy should be considered for pulmonary oedema.
- Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- Treat seizures with diazepam.
- Proparacaine hydrochloride should be used to assist eye irrigation.

EMERGENCY DEPARTMENT

- Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.
- Positive end-expiratory pressure (PEEP)-assisted ventilation may be required for acute parenchymal injury or adult respiratory distress syndrome.
- Consult a toxicologist as necessary.

BRONSTEIN, A.C. and CURRANCE, P.L. EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

For acute and short term repeated exposures to methanol:
· Toxicity results from accumulation of formaldehyde/formic acid.
· Clinical signs are usually limited to CNS, eyes and GI tract Severe metabolic acidosis may produce dyspnea and profound systemic effects which may become intractable. All symptomatic patients should have arterial pH measured. Evaluate airway, breathing and circulation.
· Stabilise obtunded patients by giving naloxone, glucose and thiamine.
· Decontaminate with Ipecac or lavage for patients presenting 2 hours post-ingestion. Charcoal does not absorb well; the usefulness of cathartic is not established.
· Forced diuresis is not effective; haemodialysis is recommended where peak methanol levels exceed 50 mg/dL (this correlates with serum bicarbonate levels below 18 mEq/L).
· Ethanol, maintained at levels between 100 and 150 mg/dL, inhibits formation of toxic metabolites and may be indicated when peak methanol levels exceed 20 mg/dL. An intravenous solution of ethanol in D5W is optimal.
· Folate, as leucovorin, may increase the oxidative removal of formic acid. 4-methylpyrazole may be an effective adjunct in the treatment. 8.Phenytoin may be preferable to diazepam for controlling seizure.

[Ellenhorn Barceloux: Medical Toxicology]

Methanol poisoning can be treated with fomepizole, or if unavailable, ethanol. Both drugs act to reduce the action of alcohol dehydrogenase on methanol by means of competitive inhibition. Ethanol, the active ingredient in alcoholic beverages, acts as a competitive inhibitor by more effectively binding and saturating the alcohol dehydrogenase enzyme in the liver, thus blocking the binding of methanol. Methanol is excreted by the kidneys without being converted into the very toxic metabolites formaldehyde and formic acid. Alcohol dehydrogenase instead enzymatically converts ethanol to acetaldehyde, a much less toxic organic molecule. Additional treatment may include sodium bicarbonate for metabolic acidosis, and hemodialysis or hemodiafiltration to remove methanol and formate from the blood. Folinic acid or folic acid is also administered to enhance the metabolism of formate.

BIOLOGICAL EXPOSURE INDEX - BEI

Determinant	Index	Sampling Time	Comment
1. Methanol in urine	15 mg/l	End of shift	B, NS
2. Formic acid in urine	80 mg/gm creatinine	Before the shift at end of workweek	B, NS

B: Background levels occur in specimens collected from subjects **NOT** exposed.
NS: Non-specific determinant - observed following exposure to other materials.

SECTION 5 Fire-fighting measures

Extinguishing media

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

Special hazards arising from the substrate or mixture

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

Special protective equipment and precautions for fire-fighters

Fire Fighting	
Fire/Explosion Hazard	▸ Liquid and vapour are highly flammable. ▸ Severe fire hazard when exposed to heat, flame and/or oxidisers. ▸ Vapour may travel a considerable distance to source of ignition. ▸ Heating may cause expansion or decomposition leading to violent rupture of containers. ▸ On combustion, may emit toxic fumes of carbon monoxide (CO). Combustion products include: carbon dioxide (CO_2) other pyrolysis products typical of burning organic material. **Contains low boiling substance:** Closed containers may rupture due to pressure buildup under fire conditions.

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Continued...

Methods and material for containment and cleaning up

Minor Spills	▸ Remove all ignition sources. ▸ Clean up all spills immediately. ▸ Avoid breathing vapours and contact with skin and eyes. ▸ Control personal contact with the substance, by using protective equipment. ▸ Contain and absorb small quantities with vermiculite or other absorbent material. ▸ Wipe up. ▸ Collect residues in a flammable waste container.
Major Spills	Chemical Class: ester and ethers For release onto land: recommended sorbents listed in order of priority. SORBENT TYPE | RANK | APPLICATION | COLLECTION | LIMITATIONS LAND SPILL - SMALL cross-linked polymer - particulate | 1 | shovel | shovel | R, W, SS cross-linked polymer - pillow | 1 | throw | pitchfork | R, DGC, RT sorbent clay - particulate | 2 | shovel | shovel | R,I, P wood fiber - particulate | 3 | shovel | shovel | R, W, P, DGC wood fiber - pillow | 3 | throw | pitchfork | R, P, DGC, RT treated wood fiber - pillow | 3 | throw | pitchfork | DGC, RT LAND SPILL - MEDIUM cross-linked polymer - particulate | 1 | blower | skiploader | R,W, SS cross-linked polymer - pillow | 2 | throw | skiploader | R, DGC, RT sorbent clay - particulate | 3 | blower | skiploader | R, I, P polypropylene - particulate | 3 | blower | skiploader | W, SS, DGC expanded mineral - particulate | 4 | blower | skiploader | R, I, W, P, DGC wood fiber - particulate | 4 | blower | skiploader | R, W, P, DGC Legend DGC: Not effective where ground cover is dense R; Not reusable I: Not incinerable P: Effectiveness reduced when rainy RT:Not effective where terrain is rugged SS: Not for use within environmentally sensitive sites W: Effectiveness reduced when windy Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control; R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▸ Containers, even those that have been emptied, may contain explosive vapours. ▸ Do NOT cut, drill, grind, weld or perform similar operations on or near containers. **Contains low boiling substance:** Storage in sealed containers may result in pressure buildup causing violent rupture of containers not rated appropriately. ▸ Check for bulging containers. ▸ Vent periodically ▸ Always release caps or seals slowly to ensure slow dissipation of vapours ▸ 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, heat or ignition sources. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Vapour may ignite on pumping or pouring due to static electricity. ▸ **DO NOT** use plastic buckets. ▸ Earth and secure metal containers when dispensing or pouring product. ▸ Use spark-free tools when handling. ▸ Avoid contact with incompatible materials. ▸ Keep containers securely sealed. ▸ 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. ▸ **DO NOT** allow clothing wet with material to stay in contact with skin

Continued...

Other information	▸ Store in original containers in approved flame-proof area. ▸ No smoking, naked lights, heat or ignition sources. ▸ **DO NOT** store in pits, depressions, basements or areas where vapours may be trapped. ▸ Keep containers securely sealed. ▸ Store away from incompatible materials in a cool, dry well ventilated area. ▸ 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	▸ Packing as supplied by manufacturer. ▸ Plastic containers may only be used if approved for flammable liquid. ▸ Check that containers are clearly labelled and free from leaks. ▸ For low viscosity materials (i) : Drums and jerry cans must be of the non-removable head type. (ii) : Where a can is to be used as an inner package, the can must have a screwed enclosure. ▸ For materials with a viscosity of at least 2680 cSt. (23 deg. C) ▸ For manufactured product having a viscosity of at least 250 cSt. (23 deg. C) ▸ Manufactured product that requires stirring before use and having a viscosity of at least 20 cSt (25 deg. C): (i) Removable head packaging; (ii) Cans with friction closures and (iii) low pressure tubes and cartridges may be used. ▸ Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages ▸ In addition, where inner packagings are glass and contain liquids of packing group I there must be sufficient inert absorbent to absorb any spillage, unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
Storage incompatibility	Methyl acetate: ▸ reacts violently with oxidisers ▸ decomposes on contact with acid or bases forming methanol ▸ is incompatible with nitrates ▸ attacks some plastics ▸ may generate electrostatic charges ▸ Esters react with acids to liberate heat along with alcohols and acids. ▸ Strong oxidising acids may cause a vigorous reaction with esters that is sufficiently exothermic to ignite the reaction products. ▸ Heat is also generated by the interaction of esters with caustic solutions. ▸ Flammable hydrogen is generated by mixing esters with alkali metals and hydrides. ▸ Esters may be incompatible with aliphatic amines and nitrates.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US OSHA Permissible Exposure Limits (PELs) Table Z-1	methyl acetate	Methyl acetate	200 ppm / 610 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	methyl acetate	Methyl acetate	200 ppm / 610 mg/m3	760 mg/m3 / 250 ppm	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	n-hexane	n-Hexane	500 ppm / 1800 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	n-hexane	n-Hexane	50 ppm / 180 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	cyclohexane	Cyclohexane	300 ppm / 1050 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	cyclohexane	Cyclohexane	300 ppm / 1050 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	bisphenol A diglycidyl ether	Particulates Not Otherwise Regulated (PNOR)- Respirable fraction	5 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	bisphenol A diglycidyl ether	Particulates Not Otherwise Regulated (PNOR)- Total dust	15 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	bisphenol A diglycidyl ether	Inert or Nuisance Dust: Total Dust	15 mg/m3 / 50 mppcf	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	bisphenol A diglycidyl ether	Inert or Nuisance Dust: Respirable fraction	5 mg/m3 / 15 mppcf	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	bisphenol A diglycidyl ether	Particulates not otherwise regulated	Not Available	Not Available	Not Available	See Appendix D

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
methyl acetate	250 ppm	1,700 ppm	10000* ppm
n-hexane	260 ppm	Not Available	Not Available
naphtha petroleum, light, hydrotreated	1,000 mg/m3	11,000 mg/m3	66,000 mg/m3
cyclohexane	300 ppm	1700* ppm	10000** ppm
bisphenol A diglycidyl ether	39 mg/m3	430 mg/m3	2,600 mg/m3
bisphenol A diglycidyl ether	90 mg/m3	990 mg/m3	5,900 mg/m3

Continued...

Ingredient	Original IDLH	Revised IDLH
methyl acetate	3,100 ppm	Not Available
n-hexane	1,100 ppm	Not Available
naphtha petroleum, light, hydrotreated	Not Available	Not Available
cyclohexane	1,300 ppm	Not Available
rosin/ isophthalic acid/ pentaerythritol	Not Available	Not Available
bisphenol A diglycidyl ether	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
naphtha petroleum, light, hydrotreated	E	≤ 0.1 ppm
rosin/ isophthalic acid/ pentaerythritol	E	≤ 0.01 mg/m³
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

Continued...

for methyl acetate:
Odour Threshold Value: 182 ppm (detection), 297 ppm (recognition)
Methyl acetate is metabolised to methanol in manner proportional to the exposure level and the TLV-TWA is analagous to that proposed for methanol. The TLV-TWA is thought to be protective against narcosis, eye and skin irritation and pulmonary irritation.
Odour Safety Factor(OSF)
OSF=43 (METHYL ACETATE)

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

Exposed individuals are **NOT** reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odour Safety Factor (OSF) is determined to fall into either Class C, D or E.

The Odour Safety Factor (OSF) is defined as:

OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm

Classification into classes follows:

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

For cyclohexane:
Odour Threshold Value: 784 ppm (detection)
NOTE: Detector tubes for cyclohexane, measuring in excess of 100 ppm are commercially available.
The recommended TLV-TWA represents the borderline of irritation but takes into account the practical difficulties of achieving lower values in the workplace. Whether serious or long-lasting consequences result from exposure at 300 ppm or whether humans become narcosed or fatigued remains to be established. The present value is thought to be a satisfactory bench-mark until further studies are made.
Odour Safety Factor(OSF)
OSF=4 (CYCLOHEXANE)

for heptane (all isomers)
The TLV-TWA is protective against narcotic and irritant effects which are greater than those of pentane or n-hexane but less than those of octane. The TLV-TWA applies to all isomers. Inhalation by humans of 1000 ppm for 6 minutes produced slight dizziness. Higher concentrations for shorter periods produce marked vertigo, incoordination and hilarity. Signs of central nervous system depression occur in the absence of mucous membrane irritation. Brief exposures to high levels (5000 ppm for 4 minutes) produce nausea, loss of appetite and a 'gasoline-like' taste in the mouth that persists for many hours after exposure ceases
for: hexane, isomers (excluding n-hexane)
The TLV-TWA is thought to be protective against nausea, headache, upper respiratory tract irritation and CNS depression. The STEL is added to prevent objective depression of the CNS. The lower value ascribed
to n-hexane is due to the neurotoxicity of its metabolites, principally 5-hydroxy-2-hexanone and 2,5-hexanedione. It is considered unlikely that other hexanes follow the same metabolic route. It should be noted however that the n-hexane TLV-TWA also applies to commercial hexane having a concentration of greater than 5% n-hexane.

For n-hexane:
Odour Threshold Value: 65 ppm
NOTE: Detector tubes for n-hexane, measuring in excess of 100 ppm, are available commercially.
Occupational polyneuropathy may result from exposures as low as 500 ppm (as hexane), whilst nearly continuous exposures of 250 ppm have caused neurotoxic effects in animals. Many literature reports have failed to distinguish hexane from n-hexane and on the assumption that the commercial hexane contains 30% n-hexane, a worst case recommendation for TLV is assumed to reduce the risk of peripheral neuropathies (due to the metabolites 2,5-heptanedione and 3,6-octanedione) and other adverse neuropathic effects.
Concurrent exposure to chemicals (including MEK) and drugs which induce hepatic liver oxidative metabolism can reduce the time for neuropathy to appear.
Odour Safety Factor(OSF)
OSF=0.15 (n-HEXANE)

Exposure controls

Appropriate engineering controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard 'physically' away from the worker and ventilation that strategically 'adds' and 'removes' air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
Employers may need to use multiple types of controls to prevent employee overexposure.
For flammable liquids and flammable gases, local exhaust ventilation or a process enclosure ventilation system may be required. Ventilation equipment should be explosion-resistant.
Air contaminants generated in the workplace possess varying 'escape' velocities which, in turn, determine the 'capture velocities' of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)

Continued...

	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion) | 1-2.5 m/s (200-500 f/min.)

Within each range the appropriate value depends on:

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

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.
· Adequate ventilation is typically taken to be that which limits the average concentration to no more than 25% of the LEL within the building, room or enclosure containing the dangerous substance.
· Ventilation for plant and machinery is normally considered adequate if it limits the average concentration of any dangerous substance that might potentially be present to no more than 25% of the LEL. However, an increase up to a maximum 50% LEL can be acceptable where additional safeguards are provided to prevent the formation of a hazardous explosive atmosphere. For example, gas detectors linked to emergency shutdown of the process might be used together with maintaining or increasing the exhaust ventilation on solvent evaporating ovens and gas turbine enclosures.
· Temporary exhaust ventilation systems may be provided for non-routine higher-risk activities, such as cleaning, repair or maintenance in tanks or other confined spaces or in an emergency after a release. The work procedures for such activities should be carefully considered.. The atmosphere should be continuously monitored to ensure that ventilation is adequate and the area remains safe. Where workers will enter the space, the ventilation should ensure that the concentration of the dangerous substance does not exceed 10% of the LEL (irrespective of the provision of suitable breathing apparatus)

Personal protection

Eye and face protection

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

Skin protection

See Hand protection below

Hands/feet protection

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

NOTE:

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

For esters:

- Do NOT use natural rubber, butyl rubber, EPDM or polystyrene-containing materials.

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

Continued...

	data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Body protection	See Other protection below
Other protection	▸ Overalls. ▸ PVC Apron. ▸ PVC protective suit may be required if exposure severe. ▸ Eyewash unit. ▸ Ensure there is ready access to a safety shower. ▸ Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity. ▸ For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets). ▸ Non sparking safety or conductive footwear should be considered. Conductive footwear describes a boot or shoe with a sole made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground the foot an shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
'Forsberg Clothing Performance Index'.
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
AW307

Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C
HYPALON	C
NATURAL RUBBER	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVA	C
PVC	C
SARANEX-23 2-PLY	C
TEFLON	C
VITON	C
VITON/CHLOROBUTYL	C

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as 'feel' or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

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

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

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

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

- Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Rosin is a solid resin obtained from pines and some other plants, mostly conifers, produced by heating fresh liquid resin to vapourise the volatile liquid (terpene) components.Solidified resin from which the volatile terpene components have been removed by distillation is known as rosin. Typical rosin is a transparent or translucent mass, with a vitreous fracture and a faintly yellow or brown colour, non-odorous or having only a slight turpentine odor and taste. Rosin is insoluble in water, mostly soluble in alcohol, essential oils, ether and hot fatty oils, and softens and melts under the influence of heat, and burns with a bright but smoky flame. Solid resins are delivered as prills, flakes, pellets, or in drums (cast solid).They are brittle materials prone to create dust during handling. These combustible dusts present a fire or explosion hazard when dispersed in air or other gaseous oxidiser. This may lead to violent explosions if ignited Rosin consists of a complex mixture of different substances including organic acids named the resin acids. These are closely related to the terpenes, and derive from them through partial oxidation. Resin acids can be dissolved in alkalis to form resin soaps, from which the purified resin acids are regenerated by treatment with acids. Use may require material be molten. Molten or heated material may be compounded, moulded or extruded. Family of products which vary in their physical properties as a result of variations in production. Data presented here is for typical family member.

Continued...

	Amber Amber

Physical state	Liquid	**Relative density (Water = 1)**	0.9
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature (°C)**	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 Available
Flash point (°C)	-28.9	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	HIGHLY FLAMMABLE.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	14.23	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	2.70	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Immiscible	**pH as a solution (Not Available%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. The main effects of simple aliphatic esters are narcosis and irritation and anaesthesia at higher concentrations. These effects become greater as the molecular weights and boiling points increase. Central nervous system depression , headache, drowsiness, dizziness, coma and neurobehavioral changes may also be symptomatic of overexposure. Respiratory tract involvement may produce mucous membrane irritation, dyspnea, and tachypnea, pharyngitis, bronchitis, pneumonitis and, in massive exposures, pulmonary oedema (which may be delayed). Gastrointestinal effects include nausea, vomiting, diarrhoea and abdominal cramps. Liver and kidney damage may result from massive exposures. Shortness of breath and a irregular heartbeat may also occur as a result of exposure to methyl acetate fume. Inhalation of methyl acetate causes severe headache and considerable somnolence in humans The material has **NOT** been classified by EC Directives or other classification systems as 'harmful by inhalation'. This is because of the lack of corroborating animal or human evidence. In the absence of such evidence, care should be taken nevertheless to ensure exposure is kept to a minimum and that suitable control measures be used, in an occupational setting to control vapours, fumes and aerosols. Material is highly volatile and may quickly form a concentrated atmosphere in confined or unventilated areas. The vapour may displace and replace air in breathing zone, acting as a simple asphyxiant. This may happen with little warning of overexposure. Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.

Continued...

Ingestion	Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result. Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis). Methanol may produce a burning or painful sensation in the mouth, throat, chest and stomach. This may be accompanied by nausea, vomiting, headache, dizziness, shortness of breath, weakness, fatigue, leg cramps, restlessness, confusion, drunken behaviour, visual disturbance, drowsiness, coma and death. Onset of symptoms may be delayed for several hours. Effects are due partly to acidosis and partly to cerebral oedema. Visual impairment produces blurring, double vision (diplopia), changes in colour perception, restriction of visual fields and blindness. 60-200 ml of methanol is a fatal dose for most adults with as little as 10 ml producing blindness. In massive overdose, liver, kidney, heart and muscle injury have been described. Methanol exhibits potential hazardous properties for human health (neurological effects, CNS depression, ocular effects, reproductive and developmental effects, and other organ toxicity). The effects of methanol on the CNS and retina in humans only occur at doses at which formate accumulates due to a rate-limiting conversion to carbon dioxide. In primates, formate accumulation was observed at methanol doses greater than 500 mg/kg bw. Methanol intoxication can cause severe visual dysfunction and death. Indeed, small amounts of ingested methanol are sufficient to produce acute destruction of parts of the central nervous system leading to permanent neurological dysfunction and irreversible blindness. Ingestion of large doses of methyl acetate may result in severe cramping, intoxication and depression of the central nervous system. The material has **NOT** been classified by EC Directives or other classification systems as 'harmful by ingestion'. This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern. Considered an unlikely route of entry in commercial/industrial environments. The liquid may produce gastrointestinal discomfort and may be harmful if swallowed. Ingestion may result in nausea, pain and vomiting. Vomit entering the lungs by aspiration may cause potentially lethal chemical pneumonitis Accidental ingestion of the material may be damaging to the health of the individual.
Skin Contact	The material may accentuate any pre-existing dermatitis condition Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions. Methyl acetate has proven to cause only weak skin irritation in humans and in rabbits (no oedema, erythema with maximum grade 1 reversible within 48 hours). Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected. The material may produce moderate skin irritation; limited evidence or practical experience suggests, that the material either: ▸ produces moderate inflammation of the skin in a substantial number of individuals following direct contact and/or ▸ produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.
Eye	Over-exposure to methyl acetate vapour may result in a condition known as amylopia (dimming of vision) as a result of atrophy of the optic nerve. Methyl acetate may resemble methanol in this respect. Eye irritation is strong but reversible within 7 days in a Draize eye test with rabbits (with mean scores for observations after 24, 48 and 72 hours of 1/1/1 for iridial irritation and of 2.7/2.3/3 for conjunctival oedema). 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. Repeated or prolonged eye contact may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. 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. 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. Substances than can cuase 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 Wherever it is reasonably practicable, exposure to substances that can cuase 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. 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. Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests. Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Chronic effects of exposure to methyl acetate may be similar to those from methanol exposure because methyl acetate can be hydrolysed to yield methanol and acetic acid. Optic nerve damage is the predominant hazard. Chronic inhalation or skin exposure to n-hexane may cause peripheral neuropathy, which is damage to nerve ends in extremities, e.g. fingers, with loss of sensation and characteristic thickening. Nerve damage has been documented with chronic exposures of greater than 500 ppm. Improvement in condition does not immediately follow removal from exposure and symptoms may progress for two or three months. Recovery may take a year or more depending on severity of exposure, and may not always be complete. Exposure to n-hexane with methyl ethyl ketone (MEK) will accelerate the appearance of damage, but MEK alone will not cause the nerve damage. Other isomers of hexane do not cause nerve damage. [*Source: Shell Co.*]

Continued...

Long-term exposure to methanol vapour, at concentrations exceeding 3000 ppm, may produce cumulative effects characterised by gastrointestinal disturbances (nausea, vomiting), headache, ringing in the ears, insomnia, trembling, unsteady gait, vertigo, conjunctivitis and clouded or double vision. Liver and/or kidney injury may also result. Some individuals show severe eye damage following prolonged exposure to 800 ppm of the vapour.

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

AW307

TOXICITY	IRRITATION
Not Available	Not Available

methyl acetate

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[2]	Eye (rabbit):100 mg/24h-moderate
Oral (Rabbit) LD50; 3700 mg/kg[2]	Skin (rabbit): 20 mg/24h - mild
	Skin (rabbit): 500 mg/24h - mild

n-hexane

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >2000 mg/kg[1]	Eye(rabbit): 10 mg - mild
Inhalation(Rat) LC50; 48000 ppm4h[2]	
Oral (Rat) LD50; 28710 mg/kg[2]	

naphtha petroleum, light, hydrotreated

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >1900 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
Inhalation(Rat) LC50; >4.42 mg/L4h[1]	Skin: adverse effect observed (irritating)[1]
Oral (Rat) LD50; >2000 mg/kg[1]	

cyclohexane

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
Inhalation(Rat) LC50; >5540 ppm4h[1]	Skin(rabbit): 1548 mg/48hr - mild
Oral (Rat) LD50; 12705 mg/kg[2]	Skin: adverse effect observed (irritating)[1]
	Skin: no adverse effect observed (not irritating)[1]

rosin/ isophthalic acid/ pentaerythritol

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >2000 mg/kg[2]	Not Available
Oral (Rat) LD50; >5000 mg/kg[2]	

bisphenol A diglycidyl ether

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	Eye (rabbit): 2 mg/24h - SEVERE
Oral (Rat) LD50; >2000 mg/kg[1]	Eye: adverse effect observed (irritating)[1]
	Skin (rabbit): 500 mg - mild
	Skin: adverse effect observed (irritating)[1]

Legend: *1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*

AW307

Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.
No evidence of a sensitization response was observed in the Gum roins key study, a guideline Local Lymph Node Assay conducted in mice, or in ten supporting studies conducted in guinea pigs according to the GPMT or Buehler methods. Gum Rosin is not classified for dermal sensitization according to the UN Globally Harmonized System of Classification and Labelling of Chemicals (GHS). Gum Rosin is currently classified for Skin Sensitization according to Annex I to Directive 67/548/EEC as R43: May cause sensitization by skin contact. Gum Rosin is also classified according to EU Classification, Labelling and Packaging of Substances and Mixtures (CLP) Regulation (EC) No. 1272/2008. As part of the harmonized translation between Directive 67/548/EEC and EU CLP Regulation (EC) No. 1272/2008, Table 3.1 of EU CLP Regulation (EC) No. 1272/2008 classifies Gum Rosin as “Skin Sensitizer Category 1” and assigns the hazard statement H317: May cause an allergic skin reaction. Table 3.2 of EU CLP Regulation (EC) No. 1272/2008 contains a list of harmonized classifications and labelling of hazardous substances from Annex I to Directive 67/548/EEC. Gum Rosin is assigned the risk phrase R43: May cause sensitization by skin contact in Table 3.2.
Subsequent evaluation determined that the single positive study for Gum Rosin was actually conducted with an oxidized form of the test material.

Continued...

	Several esters of Rosin have been tested using similar protocols with similar results. When the Rosin esters were heated beyond the specified protocol, the oxidized material caused a positive sensitization response. When those same esters were retested using a different protocol which did not cause oxidation, all sensitization responses were negative. While the oxidized form of Gum Rosin should be considered a skin sensitizer, the recommendation is made to declassify non-oxidized Gum Rosin (CAS # 8050-09-7). Generally,linear and branched-chain alkyl esters are hydrolysed to their component alcohols and carboxylic acids in the intestinal tract, blood and most tissues throughout the body. Following hydrolysis the component alcohols and carboxylic acids are metabolized Oral acute toxicity studies have been reported for 51 of the 67 esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids. The very low oral acute toxicity of this group of esters is demonstrated by oral LD50 values greater than 1850 mg/kg bw Genotoxicity studies have been performed in vitro using the following esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids: methyl acetate, butyl acetate, butyl stearate and the structurally related isoamyl formate and demonstrates that these substances are not genotoxic. The JEFCA Committee concluded that the substances in this group would not present safety concerns at the current levels of intake the esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids are generally used as flavouring substances up to average maximum levels of 200 mg/kg. Higher levels of use (up to 3000 mg/kg) are permitted in food categories such as chewing gum and hard candy. In Europe the upper use levels for these flavouring substances are generally 1 to 30 mg/kg foods and in special food categories like candy and alcoholic beverages up to 300 mg/kg foods **Internationl Program on Chemical Safety: the Joint FAO/WHO Expert Committee on Food Additives (JECFA)** **Esters of Aliphatic acyclic primary alcohols with aliphatic linear saturated carboxylic acids.; 1998**
METHYL ACETATE	The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. 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 the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
N-HEXANE	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
NAPHTHA PETROLEUM, LIGHT, HYDROTREATED	For Low Boiling Point Naphthas (LBPNs): **Acute toxicity:** LBPNs generally have low acute toxicity by the oral (median lethal dose [LD50] in rats > 2000 mg/kg-bw), inhalation (LD50 in rats > 5000 mg/m3) and dermal (LD50 in rabbits > 2000 mg/kg-bw) routes of exposure Most LBPNs are mild to moderate eye and skin irritants in rabbits, with the exception of heavy catalytic cracked and heavy catalytic reformed naphthas, which have higher primary skin irritation indices. **Sensitisation:** LBPNs do not appear to be skin sensitizers, but a poor response in the positive control was also noted in these studies **Repeat dose toxicity:** The lowest-observed-adverse-effect concentration (LOAEC) and lowest-observed-adverse-effect level (LOAEL) values identified following short-term (2-89 days) and subchronic (greater than 90 days) exposure to the LBPN substances. These values were determined for a variety of endpoints after considering the toxicity data for all LBPNs in the group. Most of the studies were carried out by the inhalation route of exposure. Renal effects, including increased kidney weight, renal lesions (renal tubule dilation, necrosis) and hyaline droplet formation, observed in male rats exposed orally or by inhalation to most LBPNs, were considered species- and sex-specific These effects were determined to be due to a mechanism of action not relevant to humans -specifically, the interaction between hydrocarbon metabolites and alpha-2-microglobulin, an enzyme not produced in substantial amounts in female rats, mice and other species, including humans. The resulting nephrotoxicity and subsequent carcinogenesis in male rats were therefore not considered in deriving LOAEC/LOAEL values. Only a limited number of studies of short-term and subchronic duration were identified for site-restricted LBPNs. The lowest LOAEC identified in these studies, via the inhalation route, is 5475 mg/m3, based on a concentration-related increase in liver weight in both male and female rats following a 13-week exposure to light catalytic cracked naphtha. Shorter exposures of rats to this test substance resulted in nasal irritation at 9041 mg/m3 No systemic toxicity was reported following dermal exposure to light catalytic cracked naphtha, but skin irritation and accompanying histopathological changes were increased, in a dose-dependent manner, at doses as low as 30 mg/kg-bw per day when applied 5 days per week for 90 days in rats No non-cancer chronic toxicity studies (= 1 year) were identified for site-restricted LBPNs and very few non-cancer chronic toxicity studies were identified for other LBPNs. An LOAEC of 200 mg/m3 was noted in a chronic inhalation study that exposed mice and rats to unleaded gasoline (containing 2% benzene). This inhalation LOAEC was based on ocular discharge and ocular irritation in rats. At the higher concentration of 6170 mg/m3, increased kidney weight was observed in male and female rats (increased kidney weight was also observed in males only at 870 mg/m3). Furthermore, decreased body weight in male and female mice was also observed at 6170 mg/m3 A LOAEL of 714 mg/kg-bw was identified for dermal exposure based on local skin effects (inflammatory and degenerative skin changes) in mice following application of naphtha for 105 weeks. No systemic toxicity was reported. **Genotoxicity:** Although few genotoxicity studies were identified for the site-restricted LBPNs, the genotoxicity of several other LBPN substances has been evaluated using a variety of in vivo and in vitro assays. While in vivo genotoxicity assays were negative overall, the in vitro tests exhibited mixed results. For in vivo genotoxicity tests, LBPNs exhibited negative results for chromosomal aberrations and micronuclei induction, but exhibited positive results in one sister chromatid exchange assay although this result was not considered definitive for clastogenic activity as no genetic material was unbalanced or lost. Mixtures that were tested, which included a number of light naphthas, displayed mixed results (i.e., both positive and negative for the same assay) for chromosomal aberrations and negative results for the dominant lethal mutation assay. Unleaded gasoline (containing 2% benzene) was tested for its ability to induce unscheduled deoxyribonucleic acid (DNA) synthesis (UDS) and replicative DNA synthesis (RDS) in rodent hepatocytes and kidney cells. UDS and RDS were induced in mouse hepatocytes via oral exposure and RDS was induced in rat kidney cells via oral and inhalation exposure. Unleaded gasoline (benzene content not stated) exhibited negative results for chromosomal aberrations and the dominant lethal mutation assay and mixed results for atypical cell foci in rodent renal and hepatic cells. For in vitro genotoxicity studies, LBPNs were negative for six out of seven Ames tests, and were also negative for UDS and for forward mutations LBPNs exhibited mixed or equivocal results for the mouse lymphoma and sister chromatid exchange assays, as well as for cell transformation and positive results for one bacterial DNA repair assay. Mixtures that were tested, which included a number of light naphthas, displayed negative results for the Ames and mouse lymphoma assays Gasoline exhibited negative results for the Ames test battery, the sister chromatid exchange assay and for one mutagenicity assay . Mixed results were observed for UDS and the mouse lymphoma assay. While the majority of in vivo genotoxicity results for LBPN substances are negative, the potential for genotoxicity of LBPNs as a group cannot be discounted based on the mixed in vitro genotoxicity results. **Carcinogenicity:** Although a number of epidemiological studies have reported increases in the incidence of a variety of cancers, the majority of these studies are considered to contain incomplete or inadequate information. Limited data, however, are available for skin cancer and leukemia incidence, as well as mortality among petroleum refinery workers. It was concluded that there is limited evidence supporting the view that working in petroleum refineries entails a carcinogenic risk (Group 2A carcinogen). IARC (1989a) also classified gasoline as a Group 2B carcinogen; it considered the evidence for carcinogenicity in humans from gasoline to be inadequate and noted that published epidemiological studies had several limitations, including a lack of exposure data and the fact that it was not possible to separate the effects of combustion products from those of gasoline itself. Similar conclusions were drawn from other reviews of epidemiological studies for gasoline (US EPA 1987a, 1987b). Thus, the evidence gathered from these epidemiological studies is considered to be inadequate to conclude on the effect s of human exposure to LBPN substances. No inhalation studies assessing the carcinogenicity of the site-restricted LBPNs were identified. Only unleaded gasoline has been examined for its carcinogenic potential, in several inhalation studies. In one study, rats and mice were exposed to 0, 200, 870 or 6170 mg/m3 of a 2% benzene

Continued...

formulation of the test substance, via inhalation, for approximately 2 years. A statistically significant increase in hepatocellular adenomas and carcinomas, as well as a non-statistical increase in renal tumours, were observed at the highest dose in female mice. A dose-dependent increase in the incidence of primary renal neoplasms was also detected in male rats, but this was not considered to be relevant to humans, as discussed previously.Carcinogenicity was also assessed for unleaded gasoline, via inhalation, as part of initiation/promotion studies. In these studies, unleaded gasoline did not appear to initiate tumour formation, but did show renal cell and hepatic tumour promotion ability, when rats and mice were exposed, via inhalation, for durations ranging from 13 weeks to approximately 1 year using an initiation/promotion protocol However, further examination of data relevant to the composition of unleaded gasoline demonstrated that this is a highly-regulated substance; it is expected to contain a lower percentage of benzene and has a discrete component profile when compared to other substances in the LBPN group.
Both the European Commission and the International Agency for Research on Cancer (IARC) have classified LBPN substances as carcinogenic. All of these substances were classified by the European Commission (2008) as Category 2 (R45: may cause cancer) (benzene content = 0.1% by weight). IARC has classified gasoline, an LBPN, as a Group 2B carcinogen (possibly carcinogenic to humans) and “occupational exposures in petroleum refining” as Group 2A carcinogens (probably carcinogenic to humans).
Several studies were conducted on experimental animals to investigate the dermal carcinogenicity of LBPNs. The majority of these studies were conducted through exposure of mice to doses ranging from 694-1351 mg/kg-bw, for durations ranging from 1 year to the animals lifetime or until a tumour persisted for 2 weeks. Given the route of exposure, the studies specifically examined the formation of skin tumours. Results for carcinogenicity via dermal exposure are mixed. Both malignant and benign skin tumours were induced with heavy catalytic cracked naphtha, light catalytic cracked naphtha, light
straight-run naphtha and naphtha Significant increases in squamous cell carcinomas were also observed when mice were dermally treated with Stoddard solvent, but the latter was administered as a mixture (90% test substance), and the details of the study were not available. In contrast, insignificant increases in tumour formation or no tumours were observed when light alkylate naphtha, heavy catalytic reformed naphtha, sweetened naphtha, light catalytically cracked naphtha
or unleaded gasoline was dermally applied to mice. Negative results for skin tumours were also observed in male mice dermally exposed to sweetened naphtha using an initiation/promotion protocol.
Reproductive/ Developmental toxicity:
No reproductive or developmental toxicity was observed for the majority of LBPN substances evaluated. Most of these studies were carried out by inhalation exposure in rodents.
NOAEC values for reproductive toxicity following inhalation exposure ranged from 1701 mg/m3 (CAS RN 8052-41-3) to 27 687 mg/m3 (CAS RN 64741-63-5) for the LBPNs group evaluated, and from 7690 mg/m3 to 27 059 mg/m3 for the site-restricted light catalytic cracked and full-range catalytic reformed naphthas. However, a decreased number of pups per litter and higher frequency of post-implantation loss were observed following inhalation exposure of female rats to hydrotreated heavy naphtha (CAS RN 64742-48-9) at a concentration of 4679 mg/m3, 6 hours per day, from gestational days 7-20. For dermal exposures, NOAEL values of 714 mg/kg-bw (CAS RN 8030-30-6) and 1000 mg/kg-bw per day (CAS RN 68513-02-0) were noted . For oral exposures, no adverse effects on reproductive parameters were reported when rats were given
site-restricted light catalytic cracked naphtha at 2000 mg/kg on gestational day 13 .
For most LBPNs, no treatment-related developmental effects were observed by the different routes of exposure However, developmental toxicity was observed for a few naphthas. Decreased foetal body weight and an increased incidence of ossification variations were observed when rat dams were exposed to light aromatized solvent naphtha, by gavage, at 1250 mg/kg-bw per day. In addition, pregnant rats exposed by inhalation to hydrotreated heavy naphtha at 4679 mg/m3 delivered pups with higher birth weights. Cognitive and memory impairments were also observed in the offspring.
Low Boiling Point Naphthas [Site-Restricted]
Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length,with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent that iso- or cyclo-paraffins.
The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption,is known as the 'hydrocarbon continuum hypothesis', and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.
The High Benzene Naphthas (HBNs; Lower Olefins and Aromatics -LOA - CAT H) Category was developed for the HPV Program by grouping ethylene manufacturing streams (products) that exhibit commonalities from both manufacturing process and compositional perspectives. intermediates. The category includes hydrocarbon product streams associated with the ethylene industry that contain significant levels of benzene, generally with a benzene content greater than 10% and averaging about 55%. This grouping of CAS numbers represents hydrocarbon streams with a carbon number distribution that is predominantly C5- C11, through components boiling at 350 C or higher..
The high benzene naphthas category contains hydrocarbons (aliphatic, aromatic and olefinic) with carbon numbers predominantly in the C5-C10 range and boiling from approximately 30 deg C to 300 deg C. Members of this category contain >0.1% benzene and contain varying amounts of toluene, xylenes and n-hexane. Some category members contain naphthalenes, isoprene and 1,3-butadiene and this has been quantified where possible
All the streams in this category are complex UVCBs containing = 50% paraffins, = 60% isoparaffins, = 90% olefins, = 90% naphthenics, =100% aromatics, and above 0.1% benzene. All streams within this category are expected to have the following classifications H304, H315 and H336, H340, H350 (given their composition) and a flammability classification (either H224 or H226, depending on the flash point and / or the boiling point)
Benzene, as the predominant component in most streams, is expected to be the key driver with respect to health effects endpoints within the SIDS battery of tests. However, as the concentration of benzene is decreased and the concentrations of other components are increased, the observed effects of benzene are expected to diminish and the effects of other components are expected to increase.
The existing epidemiology and toxicology database for the components other than benzene and for mixtures containing the components is extensive. All components present in the streams at concentrations greater than 5% have been tested in at least one toxicity study. Those components having only limited data lack structural alerts for mammalian toxicity and data exist for their structural analogs. The C5 and C6 alkanes and alkenes present in the streams are not expected to significantly contribute to the toxicity profile as these substances are present in the streams at low concentrations and, with the exception of hexane, generally have a low level of toxicity. The toxic effects of hexane (present at < 15%) are unlikely to be observed due to the presence of the other components.
Genotoxicity: When tested as pure substances, some of the components other than benzene have caused genetic damage and adverse target organ effects in repeated-dose animal studies. When tested as pure substances, some of the components other than benzene have caused genetic damage and adverse target organ effects in repeated-dose animal studies. However, since the biologically active components of the High Benzene Naphthas streams are metabolized through a common P450 metabolic pathway, it is anticipated that multiple components will compete for the same active enzyme sites. Component toxicities, which are dependent on the formation of biologically active metabolites, may be reduced as less metabolite(s) will be produced through competition for these sites. Direct support for reduction or elimination of toxicities of individual components is provided by results of an existing mouse bone marrow micronucleus test with one of the High Benzene Naphthas streams, Hydrotreated C6-8 Fraction. This stream, containing approximately 55% benzene, was negative in a mouse bone marrow micronucleus test when administered by oral gavage at 5000 mg/kg to male and female CD-1 mice. Several studies have shown that benzene administered orally to CD-1 mice induces high frequencies of micronuclei in bone marrow erythrocytes at doses as low as 110 mg/kg . The presence in the Hydrotreated C6-8 Fraction of other components (approximately 25% toluene, 10% xylene, 7% pentane, 7% ethylbenzene, 3% cyclohexane, and 2% hexane) apparently inhibited the expected clastogenicity of benzene. Other similar interactions between components of the category have also been reported.
Repeat dose toxicity: Repeated oral or inhalation exposures to many of the components of the streams in the category have been shown to cause adverse health effects in a variety of organs. However, existing data also show that antagonistic and synergistic interactions occur between some components comprising the streams.

Continued...

	Developmental toxicity: Developmental toxicity data exist for most components present in this category at concentrations greater than 5% . In these studies, no convincing evidence was seen for teratogenicity in the absence of maternal toxicity. Foetotoxicity has been reported for some components, but mostly in the presence of maternal toxicity. A Pyrolysis Gasoline Fraction stream similar to the Pyrolysis Gasoline streams in the HBNs Category has been tested in an oral developmental toxicity study in rabbits. No developmental effects were seen. **Reproductive toxicity:** Some data for benzene indicates adverse gonadal effects (e.g., atrophy/degeneration, decrease in spermatozoa, moderate increases in abnormal sperm forms), data on reproductive outcomes are either inconclusive or conflicting. However, most studies indicate no effects on reproductive indices, even at high doses. Reproductive organ effects were seen after inhalation exposure to isoprene and hexane. **Gene Mutation**: Of the identified category components present at concentrations greater than 5%, only 1,3-butadiene and benzene have consistently caused gene mutations in genetic toxicity tests . 1,3- Butadiene was positive in several *in vivo* and *in vitro* tests. Benzene was negative in several standard tests but was positive in an *in vivo* HPRT gene mutation test in mouse spleenocytes. Based on the data for components, the streams in the category are predicted to be negative in the HPV gene mutation test (Ames Test). Negative Ames Tests conducted with two streams (one from this category and one similar to category streams) support this prediction **Chromosome Aberration:**: Benzene has caused chromosome aberrations in *in vitro* and *in vivo* tests. The other most prevalent component in streams in this category, toluene, is negative in both *in vitro* and *in vivo* tests. Of the remaining identified category components present at concentrations greater than 5%, only vinyl acetate, 1,3-butadiene, isoprene, hexane, and naphthalene have been reported to cause chromosome aberrations. For petroleum: This product contains benzene, which can cause acute myeloid leukaemia, and n-hexane, which can be metabolized to compounds which are toxic to the nervous system. This product contains toluene, and animal studies suggest high concentrations of toluene lead to hearing loss. This product contains ethyl benzene and naphthalene, from which animal testing shows evidence of tumour formation. Cancer-causing potential: Animal testing shows inhaling petroleum causes tumours of the liver and kidney; these are however not considered to be relevant in humans. Mutation-causing potential: Most studies involving gasoline have returned negative results regarding the potential to cause mutations, including all recent studies in living human subjects (such as in petrol service station attendants). Reproductive toxicity: Animal studies show that high concentrations of toluene (>0.1%) can cause developmental effects such as lower birth weight and developmental toxicity to the nervous system of the foetus. Other studies show no adverse effects on the foetus. Human effects: Prolonged or repeated contact may cause defatting of the skin which can lead to skin inflammation and may make the skin more susceptible to irritation and penetration by other materials. Animal testing shows that exposure to gasoline over a lifetime can cause kidney cancer, but the relevance in humans is questionable. DHC Solvent Chemie (for EC No.: 926-605-8)
CYCLOHEXANE	Bacteria mutagen
ROSIN/ ISOPHTHALIC ACID/ PENTAERYTHRITOL	IIrritation Corrosion - Eye, Data is for similar product. Result: Positive Species: New Zealand white rabbit Organ: Eye Test Duration: 4 hr Observation Period: 72 hr Notes: OECD 405 Irritation Corrosion - Skin, No skin irritation.; Data is for similar product. Result: Negative Species: New Zealand white rabbit Organ: Skin Test Duration: 4 hr Observation Period: 72 hr Notes: OECD 404 NOAEL Wistar rat 300 mg/kg/day, 8 weeks Developmental; Data is for similar product. NOEL Wistar rat 1000 mg/kg/day, 8 weeks Reproductive; Data is for similar product. * Estimates for product may be based on additional component data not shown. Skin sensitization 50 % w/w Local Lymph Node Assay - Lowest Concentration Producing Reaction, SI=5; May cause sensitization by skin contact. Result: Positive Species: Mouse Notes: OECD 429 Germ cell mutagenicity: No data available to indicate product or any components present at greater than 0.1% are mutagenic or genotoxic. Mutagenicity Germ Cell Mutagenicity: Ames, Data is for similar product. Result: Negative Species: Salmonella typhimurium Notes: OECD 471 Germ Cell Mutagenicity: Chromosome Abberation, Data is for similar product. Result: Negative Species: Human Notes: OECD 473 In Vitro Mammalian Cell Gene Mutation Test, No data available to indicate product or any components present at greater than 0.1% are mutagenic or genotoxic.; Data is for similar product. Result: Negative Species: Mouse Notes: OECD 476 Reproductive toxicity This product is not expected to cause reproductive or developmental effects. * Sylvalite RE 110L SDS
BISPHENOL A DIGLYCIDYL ETHER	In mice, dermal application of bisphenol A diglycidyl ether (BADGE) (1, 10, or 100 mg/kg) for 13 weeks produced mild to moderate chronic active dermatitis. At the high dose, spongiosis and epidermal micro abscess formation were observed. In rats, dermal application of BADGE (10, 100, or 1000 mg/kg) for 13 weeks resulted in a decrease in body weight at the high dose. The no-observable effect level (NOEL) for dermal exposure was 100 mg/kg for both sexes. In a separate study, application of BADGE (same doses) five times per week for ~13 weeks not only caused a decrease in body weight but also produced chronic dermatitis at all dose levels in males and at >100 mg/kg in females (as well as in a satellite group of females given 1000 mg/kg). **Reproductive and Developmental Toxicity**: BADGE (50, 540, or 750 mg/kg) administered to rats via gavage for 14 weeks (P1) or 12 weeks (P2) produced decreased body weight in all males at the mid dose and in both males and females at the high dose, but had no reproductive effects. The NOEL for reproductive effects was 750 mg/kg. **Carcinogenicity:** IARC concluded that 'there is limited evidence for the carcinogenicity of bisphenol A diglycidyl ether in experimental animals.' Its overall evaluation was 'Bisphenol A diglycidyl ether is not classifiable as to its carcinogenicity to humans (Group 3). In a lifetime tumourigenicity study in which 90-day-old C3H mice received three dermal applications per week of BADGE (undiluted dose) for 23 months, only one out of 32 animals developed a papilloma after 16 months. A retest, in which skin paintings were done for 27 months, however, produced no tumours (Weil et al., 1963). In another lifetime skin-painting study, BADGE (dose n.p.) was also reported to be noncarcinogenic to the skin of C3H mice; it was, however, weakly carcinogenic to the skin of C57BL/6 mice (Holland et al., 1979; cited by Canter et al., 1986). In a two-year bioassay, female Fisher 344 rats dermally exposed to BADGE (1, 100, or 1000 mg/kg) showed no evidence of dermal carcinogenicity but did have low incidences of tumours in the oral cavity (U.S. EPA, 1997). **Genotoxicity**: In S. typhimurium strains TA100 and TA1535, BADGE (10-10,000 ug/plate) was mutagenic with and without S9; negative results were obtained in TA98 and TA1537 (Canter et al., 1986; Pullin, 1977). In a spot test, BADGE (0.05 or 10.00 mg) failed to show mutagenicity in strains TA98 and TA100 (Wade et al., 1979). Negative results were also obtained in the body fluid test using urine of female BDF and ICR mice (1000 mg/kg BADGE), the mouse host-mediated assay (1000 mg/kg), micronucleus test (1000 mg/kg), and dominant lethal assay (~3000 mg/kg). **Immunotoxicity:** Intracutaneous injection of diluted BADGE (0.1 mL) three times per week on alternate days (total of 8 injections) followed by a three-week incubation period and a challenge dose produced sensitisation in 19 of 20 guinea pigs - **Consumer exposure** to BADGE is almost exclusively from migration of BADGE from can coatings into food. Using a worst-case scenario that assumes BADGE migrates at the same level into all types of food, the estimated per capita daily intake for a 60-kg individual is approximately 0.16 ug/kg body weight/day. A review of one- and two-generation reproduction studies and developmental investigations found no evidence of reproductive or endocrine toxicity, the upper ranges of dosing being determined by maternal toxicity. The lack of endocrine toxicity in the reproductive and developmental toxicological tests is supported by negative results from both in vivo and in vitro assays designed specifically to detect oestrogenic and androgenic properties of BADGE. An examination of data from sub-chronic and chronic toxicological studies support a NOAEL of 50 mg/ kg/body weight day from the 90-day study, and a NOAEL of 15 mg/kg body weigh/day (male rats) from the 2-year carcinogenicity study. Both NOAELS are considered appropriate for risk assessment. Comparing the estimated daily human intake of 0.16 ug/kg body weight/day with the NOAELS of 50 and 15 mg/kg body weight/day shows human exposure to BADGE from can coatings is between 250,000 and 100,000-fold lower than the NOAELs from the most sensitive toxicology tests. These large margins of safety together with lack of reproductive, developmental, endocrine and carcinogenic effects supports the continued use of BADGE for use in articles intended to come into contact with foodstuffs. Bisphenol A exhibits hormone-like properties that raise concern about its suitability in consumer products and food containers. Bisphenol A is thought to be an endocrine disruptor which can mimic oestrogen and may lead to negative health effects. More specifically, bisphenol A closely mimics the structure and function of the hormone oestradiol with the ability to bind to and activate the same oestrogen receptor as the natural hormone. The presence of the p-hydroxy group on the benzene rings is though to be responsible for the oestradiol mimicry. . Early developmental stages appear to be the period of greatest sensitivity to its effects and some studies have linked prenatal exposure to later physical and neurological difficulties. Regulatory bodies have determined safety levels for humans, but those safety levels are being questioned

Continued...

	or are under review. A 2009 study on Chinese workers in bisphenol A factories found that workers were four times more likely to report erectile dysfunction, reduced sexual desire and overall dissatisfaction with their sex life than workers with no heightened bisphenol A exposure. Bisphenol A workers were also seven times more likely to have ejaculation difficulties. They were also more likely to report reduced sexual function within one year of beginning employment at the factory, and the higher the exposure, the more likely they were to have sexual difficulties. Bisphenol A in weak concentrations is sufficient to produce a negative reaction on the human testicle. The researchers found that a concentration equal to 2 ug/ litre of bisphenol A in the culture medium, a concentration equal to the average concentration generally found in the blood, urine and amniotic fluid of the population, was sufficient to produce the effects. The researchers believe that exposure of pregnant women to bisphenol A may be one of the causes of congenital masculinisation defects of the hypospadia and cryptorchidism types the frequency of which has doubled overall since the 70's. They also suggested that 'it is also possible that bisphenol A contributes to a reduction in the production of sperm and the increase in the incidence of testicular cancer in adults that have been observed in recent decades' One review has concluded that obesity may be increased as a function of bisphenol A exposure, which '...merits concern among scientists and public health officials' One study demonstrated that adverse neurological effects occur in non-human primates regularly exposed to bisphenol A at levels equal to the United States Environmental Protection Agency's (EPA) maximum safe dose of 50 ug/kg/day This research found a connection between bisphenol A and interference with brain cell connections vital to memory, learning, and mood. A further review concluded that bisphenol-A has been shown to bind to thyroid hormone receptor and perhaps have selective effects on its functions. Carcinogenicity studies have shown increases in leukaemia and testicular interstitial cell tumours in male rats. However, 'these studies have not been considered as convincing evidence of a potential cancer risk because of the doubtful statistical significance of the small differences in incidences from controls'. Another in vitro study has concluded that bisphenol A is able to induce neoplastic transformation in human breast epithelial cells.[whilst a further study concluded that maternal oral exposure to low concentrations of bisphenol A, during lactation, increases mammary carcinogenesis in a rodent model. In vitro studies have suggested that bisphenol A can promote the growth of neuroblastoma cells and potently promotes invasion and metastasis of neuroblastoma cells. Newborn rats exposed to a low-dose of bisphenol A (10 ug/kg) showed increased prostate cancer susceptibility when adults. At least one study has suggested that bisphenol A suppresses DNA methylation which is involved in epigenetic changes. Bisphenol A is the isopropyl adduct of 4,4'-dihydroxydiphenyl oxide (DHDPO). A series of DHDPO analogues have been investigated as potential oestrogen receptor/anti-tumour drug carriers in the development of a class of therapeutic drugs called 'cytostatic hormones'. Oestrogenic activity is induced with 1 to 100 mg/kg body weight in animal models. Bisphenol A sealants are frequently used in dentistry for treatment of dental pits and fissures. Samples of saliva collected from dental patients during a 1-hour period following application contain the monomer. A bisphenol-A sealant has been shown to be oestrogenic in vitro; such sealants may represent an additional source of xenoestrogens in humans and may be the cause of additional concerns in children. Concerns have been raised about the possible developmental effects on the foetus/embryo or neonate resulting from the leaching of bisphenol A from epoxy linings in metal cans which come in contact with food-stuffs. Many drugs, including naproxen, salicylic acid, carbamazepine and mefenamic acid can, in vitro, significantly inhibit bisphenol A glucuronidation (detoxification). BPA belongs to the list of compounds having this property as the rodent models have shown that BPA exposure is linked with increased body weigh (obesogens)t. Several mechanisms can help explain the effect of BPA on body weight increase. A possible mechanism leading to triglyceride accumulation is the decreased production of the hormone adiponectin from all human adipose tissue tested when exposed to very low levels (below nanomolar range) of BPA in cell or explant culture settings . The expression of leptin as well as several enzymes and transcription factors is also affected by BPA exposure in vivo as well as in vitro. Together, the altered expression and activity of these important mediators of fat metabolism could explain the increase in weight following BPA exposure in rodent models. These results also suggest that, together with other obesogens, low, environmentally relevant levels of BPA may contribute to the human obesity phenomenon. All glycidyl ethers show genotoxic potential due their alkylating properties. Those glycidyl ethers that have been investigated in long term studies exhibit more or less marked carcinogenic potential. Alkylating agents may damage the stem cell which acts as the precursor to components of the blood. Loss of the stem cell may result in pancytopenia (a reduction in the number of red and white blood cells and platelets) with a latency period corresponding to the lifetime of the individual blood cells. Granulocytopenia (a reduction in granular leukocytes) develops within days and thrombocytopenia (a disorder involving platelets), within 1-2 weeks, whilst loss of erythrocytes (red blood cells) need months to become clinically manifest. Aplastic anaemia develops due to complete destruction of the stem cells. Reported adverse effects in laboratory animals include sensitization, and skin and eye irritation, as well as mutagenic and tumorigenic activity.. Testicular abnormalities (including testicular atrophy with decreased spermatogenic activity) following exposure to glycidyl ethers have been reported. Haemopoietic abnormalities following exposure to glycidyl ethers, including alteration of the leukocyte count, atrophy of lymphoid tissue, and bone marrow cytotoxicity have also been reported. These abnormalities were usually observed along with pneumonia and/or toxemia, and therefore may be secondary effects. However, especially in light of the generalized reduction in leukocytes and the atrophy of lymphoid tissues, the observed haemopoietic abnormalities may have been predisposing factors to pneumonia. While none of the individual research reports are conclusive with respect to the ability of glycidyl ethers to produce permanent changes to the testes or haemopoietic system in laboratory animals, the pattern of displayed effects is reason for concern Glycidyl ethers have been shown to cause allergic contact dermatitis in humans. Glycidyl ethers generally cause skin sensitization in experimental animals. Necrosis of the mucous membranes of the nasal cavities was induced in mice exposed to allyl glycidyl ether. A study of workers with mixed exposures was inconclusive with regard to the effects of specific glycidyl ethers. Phenyl glycidyl ether, but not n-butyl glycidyl ether, induced morphological transformation in mammalian cells in vitro. n-Butyl glycidyl ether induced micronuclei in mice in vivo following intraperitoneal but not oral administration. Phenyl glycidyl ether did not induce micronuclei or chromosomal aberrations in vivo or chromosomal aberrations in animal cells in vitro. Alkyl C12 or C14 glycidyl ether did not induce DNA damage in cultured human cells or mutation in cultured animal cells. Allyl glycidyl ether induced mutation in Drosophila. The glycidyl ethers were generally mutagenic to bacteria. Oxiranes (including glycidyl ethers and alkyl oxides, and epoxides) exhibit many common characteristics with respect to animal toxicology. One such oxirane is ethyloxirane; data presented here may be taken as representative. for 1,2-butylene oxide (ethyloxirane): Ethyloxirane increased the incidence of tumours of the respiratory system in male and female rats exposed via inhalation. Significant increases in nasal papillary adenomas and combined alveolar/bronchiolar adenomas and carcinomas were observed in male rats exposed to 1200 mg/m3 ethyloxirane via inhalation for 103 weeks. There was also a significant positive trend in the incidence of combined alveolar/bronchiolar adenomas and carcinomas. Nasal papillary adenomas were also observed in 2/50 high-dose female rats with none occurring in control or low-dose animals. In mice exposed chronically via inhalation, one male mouse developed a squamous cell papilloma in the nasal cavity (300 mg/m3) but other tumours were not observed. Tumours were not observed in mice exposed chronically via dermal exposure. When trichloroethylene containing 0.8% ethyloxirane was administered orally to mice for up to 35 weeks, followed by 0.4% from weeks 40 to 69, squamous-cell carcinomas of the forestomach occurred in 3/49 males (p=0.029, age-adjusted) and 1/48 females at week 106. Trichloroethylene administered alone did not induce these tumours and they were not observed in control animals . Two structurally related substances, oxirane (ethylene oxide) and methyloxirane (propylene oxide), which are also direct-acting alkylating agents, have been classified as carcinogenic 55badger The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.
AW307 & ROSIN/ ISOPHTHALIC ACID/ PENTAERYTHRITOL & BISPHENOL A DIGLYCIDYL ETHER	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.

Continued...

AW307 & METHYL ACETATE	for methyl acetate **Acute toxicity:** Methyl acetate is a water soluble substance with high volatility. The substance has narcotic properties if inhaled at concentrations of 34 mg/l (mice) and 56 mg/l (cats) with a short duration of the narcotic action after cessation of exposure. Methyl acetate is absorbed via the lungs in animals and humans, absorption via the oral route is demonstrated. After absorption the substance undergoes hydrolysis to methanol and acetic acid. From the available *in vitro* data it may be anticipated that the half-life of methyl acetate in blood ranges between 2 and 4 hours. Immediately after stopping a 6-hour inhalation exposure to rats (2,000 ppm (6,040 mg/m3)) blood concentrations below the limit of quantification (less than 4.6 mg/l) were determined indicating rapid hydrolysis and high clearance of the substance. It appears from these data that the systemic availability of methyl acetate is low. The main metabolite is methanol which itself is metabolised to formic acid. Formate is introduced into C1-metabolism after activation by reacting with tetrahydrofolate. Humans as well as monkeys are more sensitive to methanol poisoning compared with rats because of a lower tetrahydrofolate content in liver. Therefore interspecies differences in the metabolism were considered mainly of concern at dose levels leading to acute toxicity. Thus rat is a useful model to indicate subacute/subchronic toxic effects below sublethal dosages. Assessment of the available animal toxicology data indicates that methyl acetate is of low acute toxicity (rats LD50 oral: 6,482 mg/kg bw, dermal: >2,000 mg/kg bw, LC50 inhalative >49 mg/l/4h). After oral application and after inhalation of substance vapours, animals showed narcotic symptoms, spasms, dyspnea and vomiting; inhalation of vapours in addition caused irritation of eyes and upper respiratory tract. The narcotic concentration for mice starts at 34 mg/l and for cats with 56 mg/l inhaled. In humans, accidental inhalation of vapours of methyl acetate caused severe headache and considerable somnolence. Methyl acetate has proven to cause only weak skin irritation in humans and in rabbits (no oedema, erythema with maximum grade 1 reversible within 48 hours). Eye irritation however, was strong but reversible within 7 days in a Draize eye test with rabbits. Exposure to methyl acetate vapours causes irritation to eyes and respiratory tract of humans. Taking into account the long experience with human exposure to the substance, methyl acetate is not supposed to exhibit skin sensitising properties although no relevant human or animal date are available. **Sensitisation:** Relevant human data are not available. In a maximisation test with 25 volunteers no sensitisation was observed after exposure to 10% methyl acetate in petrolatum (Kligman, 1976). Taking into account the long experience with human exposure to the substance, and the absence of any reports on contact allergy in exposed persons , methyl acetate is not expected to exhibit skin sensitising properties, especially since the substance is hydrolysed in contact with water by non-specific tissue esterases to methanol and acetic acid. For these substances a skin sensitisation potential is either absent (methanol, or restricted to a few cases (acetic acid). **Repeat dose toxicity:** Overall, reliable experimental animal data on the local and systemic effects after repeated administration of methyl acetate are restricted to the inhalation exposure. After nose-only inhalation during a 28-day treatment period, methyl acetate induced degeneration/necrosis of the rat olfactory mucosa at a concentration of 2,000 ppm on 6 hours/day, 5 days/week (6,040 mg/m3). There was some concern on minimal effects of systemic toxicity at this concentration diureses, minimal liver cell dysfunction, adrenal weight increase, and reduced serum cholesterol concentrations). There are no adequate data from human experience on repeated or prolonged exposure. Based on general experience that acute and long-time or repeated exposure to methyl acetate defats skin and cause dryness and cracking of the skin. **No-observed-adverse-effect-level (NOAEL)** *Inhalation route* The NOAEC for local effects on the respiratory tract derived from an accurate 28-day inhalation study in rats was 350 ppm (1,057 mg/m3). The NOAEC for systemic effects also derived from a 28-day inhalation study was 350 ppm (1,057 mg/m3). **Mutagenicity** Methyl acetate is negative in a bacterial mutation test and a rat bone marrow micronucleus test. Furthermore, the hydrolysis products methanol and acetic acid do not reveal evidence for a mutagenic potential. There is no concern with respect to mutagenicity. Methyl acetate should not be classified as a mutagen. **Reproductive toxicity:** There are no data on reproductive toxicity of methyl acetate. However, due to the rapid hydrolysis of this compound it is justified to base hazard assessment with respect to reproduction on the toxicological properties of the immediate metabolites. Concerning the metabolites of methyl acetate, acetic acid appears to be of less significance, since there are no indications of a foetotoxic or teratogenic potential, whereas for methanol some embryo-/foetotoxic and teratogenic effects were demonstrated in rodents, however at relatively high concentrations, respectively maternal toxic concentrations only. A NOEC/fertility for methanol of 1,000 ppm (1,300 mg methanol/m3) was derived from a 2-generation inhalation study in rats. With the assumption that methyl acetate is immediately degraded to methanol at a molar ratio of 1, this value can be converted to NOAEC/fertility of about 3,000 mg methyl acetate/m3. A NOAEC/developmental toxicity for methanol of 1,000 ppm (1,300 mg methanol/m3) was derived from two studies in mice and rats from intermittent as well as from continuous inhalatory exposure, which can be converted to a NOAEC/developmental toxicity of about 3,000 mg methyl acetate/m3.

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

AW307

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

methyl acetate

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	72h	Algae or other aquatic plants	>=120mg/l	1
EC50	72h	Algae or other aquatic plants	>120mg/l	1
EC50	48h	Crustacea	1026.7mg/l	1
LC50	96h	Fish	250mg/l	1

Continued...

n-hexane

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	240h	Algae or other aquatic plants	25.023-137.802mg/L	4

naphtha petroleum, light, hydrotreated

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	504h	Crustacea	0.17mg/l	2
EC50	48h	Crustacea	0.64mg/l	2
LC50	96h	Fish	4.26mg/l	2
EC50	96h	Algae or other aquatic plants	64mg/l	2

cyclohexane

Endpoint	Test Duration (hr)	Species	Value	Source
BCF	1344h	Fish	31-102	7
EC50	72h	Algae or other aquatic plants	3.428mg/l	2
EC50	48h	Crustacea	0.9mg/l	2
EC50(ECx)	48h	Crustacea	0.9mg/l	2
LC50	96h	Fish	4.53mg/l	2
EC50	96h	Algae or other aquatic plants	2.17mg/l	2

rosin/ isophthalic acid/ pentaerythritol

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	48h	Crustacea	>100mg/l	Not Available
EC50	72h	Algae or other aquatic plants	1000mg/l	Not Available
EC50	48h	Crustacea	>100mg/l	Not Available
LC50	96h	Fish	1000mg/l	Not Available

bisphenol A diglycidyl ether

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	9.4mg/l	2
EC50	48h	Crustacea	1.1mg/l	2
NOEC(ECx)	504h	Crustacea	0.3mg/l	2
LC50	96h	Fish	1.2mg/l	2

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*

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 methyl actetate:
Environmental fate:
Biodegradation
The substance can be classified as “readily biodegradable” on the basis of an available study according to OECD-guideline 301 D. This closed bottle test indicates 74% biodegradation after 14 days, 75% after 19 days and 70% after 28 days. There is no information on possible intermediates before ultimate degradation of methyl acetate. Probably methanol and acetic acid could be intermediates of the biodegradation. The degradation of the possible intermediates is included in the results of the biodegradation test.
Photodegradation
Direct photolysis of methyl acetate in the atmosphere is not to be expected. However, in the atmosphere gaseous methyl acetate reacts with hydroxyl radicals which have been formed photochemically. On the basis of an atmospheric concentration of the OH-radicals amounting to 5.10exp5 OH/cm3 and the rate constant (kdeg(air)) of 0.3182.10exp-12cm3.molecule-1.s-1, a half-life of 50.4 days is calculated for the photochemical degradation in the atmosphere. A half-life of 94 days was determined on the basis of laboratory investigations into photochemical degradation.
Hydrolysis
The hydrolysis of methyl acetate was examined in an older investigation from 1935. In this, a hydrolysis half-life of approximately 53 days at a temperature of 23.2 to 25.4 deg C was determined for methyl acetate (148.6 g/l). No information was provided on the pH value of the solution.
Hydrolysis half-lives of between approximately 63 days (pH = 8) and approximately 627 days (pH = 7) were calculated for the substance using QSAR calculations. Hydrolysis should therefore not represent a significant elimination process for methyl acetate in the environment.
Distribution
On account of the vapour pressure of 217 hPa, methyl acetate is expected to evaporate quickly from surfaces.
A Henrys Constant of 6.43 Pa m3/mol at 20 deg C is calculated from the data on the vapour pressure and water solubility of methyl acetate given in Section 1. Consequently, the substance is moderately volatile from an aqueous solution..
No bioaccumulation potential is to be expected due to the measured log Kow value for methyl acetate of 0.18. On the basis of this value the Koc is calculated as 12.99 l/kg and the partition coefficients can be calculated according to the organic carbon content in the individual environmental compartments.
Accumulation
No investigations on bioaccumulation are available. The measured log Kow of 0.18 does not provide any indication of a relevant bioaccumulation potential.
The calculated Koc value of 12.99 l/kg also does not indicate that a significant geoaccumulation potential is to be expected for methyl acetate. The substance may be washed out from soil to groundwater by rainwater depending on the elimination in soil by degradation and distribution.
Atmosphere
Due to the atmospheric half-life (t1/2 = 74 to 94 days), abiotic effects on the atmosphere, such as global warming and ozone depletion, are not to be expected in connection with methyl acetate
For n-heptane:
log Kow : 4.66
Koc : 2400-8100
Half-life (hr) air : 52.8
Half-life (hr) H2O surface water : 2.9-312

Continued...

Henry's atm m3 /mol: 2.06
BOD 5 if unstated: 1.92
COD : 0.06
BCF : 340-2000
log BCF : 2.53-3.31
Environmental fate:
Photolysis or hydrolysis of n-heptane are not expected to be important environmental fate processes. Biodegradation of n-heptane may occur in soil and water, however volatilisation and adsorption are expected to be more important fate processes. A high Koc (2400-8200) indicates n-heptane will be slightly mobile to immobile in soil. In aquatic systems n-heptane may partition from the water column to organic matter in sediments and suspended solids. The bioconcentration of n-heptane may be important in aquatic environments. the Henry's Law constant suggests rapid volatilisation from environmental waters and surface soils. The volatilisation half-lives from a model river and a model pond (the latter considers the effect of adsorption) have been estimated to be 2.9 hr and 13 days, respectively.
n-Heptane is expected to exist entirely in the vapour phase in ambient air. Reactions with photochemically produced hydroxyl radicals in the atmosphere have been shown to be important (estimated half-life of 2.4 days calculated from its rate constant of 7.15x10-12 cu cm/molecule-sec at 25 deg C). Data also suggests that night-time reactions with nitrate radicals may contribute to the atmospheric transformation of n-heptane, especially in urban environments. n-Heptane does not contain chromophores that absorb at wavelengths >290 nm and therefore is not expected to be susceptible to direct photolysis by sunlight
An estimated BCF of 2,000 using log Kow suggests the potential for bioconcentration in aquatic organisms is very high. Based on 100% degradation after 4 days in water inoculated with gasoline contaminated soil and 100% degradation after 25 days in water inoculated with activated sewage sludge, biodegradation is expected to be an important fate process for n-heptane in water.
Ecotoxicity:
Fish LC50 (48 h): goldfish (Carrasius auratus) 4 mg/l; golden orfe (Idus melanotus) 2940 mg/l; western mosquitofish (Gambusia affinis) 4924 mg/l
Daphnia LC50 (24 h): >10 mg/l
Daphnia EC50 (96 h): 82 mg/l (immobilisation)
Opposum shrimp (Mysidopsis bahia) LC50 (96 h): 0.1 mg/l
Snail EC50 (96 h): 472 mg/l
For n-hexane:
log Kow: 3.17-3.94
BOD 5 if unstated: 2.21
COD: 0.04
ThOD: 3.52
Environmental fate:
Transport and Partitioning: The physical properties of *n*-hexane that affect its transport and partitioning in the environment are: water solubility of 9.5 mg/L; log[Kow] (octanol/water partition coefficient), estimated as 3.29; Henry s law constant, 1.69 atm-m3 mol; vapor pressure, 150 mm Hg at 25 C; and log[Koc] in the range of 2.90 to 3.61. As with many alkanes, experimental methods for the estimation of the Koc parameter are lacking, so that estimates must be made based on theoretical considerations.
The dominant transport process from water is volatilization. Based on mathematical models the half-life for *n*-hexane in bodies of water with any degree of turbulent mixing (e.g., rivers) would be less than 3 hours. For standing bodies of water (e.g. small ponds), a half-life no longer than one week (6.8 days) is estimated Based on the log octanol/water partition coefficient (i.e. log[Koc]) and the estimated log sorption coefficient (i.e. log[Koc]) *n*-hexane is not expected to become concentrated in biota. A calculated bioconcentration factor (BCF) of 453 for a fathead minnow further suggests a low potential for *n*-hexane to bioconcentrate or bioaccumulate in trophic food chains.
In soil, the dominant transport mechanism for *n*-hexane present near the surface probably is volatilisation (based on its Henry s law constant, water solubility, vapor pressure, and Koc). While its estimated Koc values suggest a moderate ability to sorb to soil particles, *n*-hexane has a density (0.6603 g/mL at 20 C) well below that of water and a very low water solubility of 9.5 mg/L. *n*-Hexane would, therefore, be viewed as a light nonaqueous phase liquid (LNAPL), which would suggest a low potential for leaching into the lower soil depths since the *n*-hexane would tend to float on the top of the saturated zone of the water table. *n*-Hexane would generally stay near the soil surface and, if not appreciably sorbed into the soil matrix, would be expected eventually to volatilise to the atmosphere. Exceptions would involve locations with shallow groundwater tables where there were large spills of hexane products. In such cases, the *n*-hexane could spread out to contaminant a large volume of soil materials.
Air: *n*-Hexane does not absorb ultraviolet (UV) light at 290 nm and is thus not expected to undergo direct photolysis reactions. The dominant tropospheric removal mechanism for *n*-hexane is generally regarded to be decomposition by hydroxyl radicals. Calculations assuming typical hydroxyl radical concentrations suggest a half-life of approximately 2.9 days. While *n*-hexane can react with nitrogen oxides to produce ozone precursors under controlled laboratory conditions, the smog-producing potential of *n*-hexane is very low compared to that of other alkanes or chlorinated VOCs. Hydroxyl ion reactions in the upper troposphere, therefore, are probably the primary mechanisms for *n*-hexane degradation in the atmosphere. As with most alkanes, *n*-hexane is resistant to hydrolysis
Water: Although few data are available dealing explicitly with the biodegradation of *n*-hexane in water, neither hydrolysis nor biodegradation in surface waters appears to be rapid compared with volatilization. In surface waters, as in the atmosphere, alkanes such as *n*-hexane would be resistant to hydrolysis. Biodegradation is probably the most significant degradation mechanism in groundwater. The ability of *Pseudomonas mendocina* bacteria to metabolise *n*-hexane in laboratory microcosms simulating groundwater conditions has been documented. Mixed bacterial cultures as well as pure cultures are documented as capable of metabolizing *n*-hexane under aerobic conditions. In general, linear alkanes (such as *n*-hexane) are viewed as the most readily biodegradable fractions in petroleum , particularly when oxygen is present in solution. Once introduced into groundwater, *n*-hexane may be fairly persistent since its degradation by chemical hydrolysis is slow and opportunities for biodegradation may be limited under anoxic conditions or where nutrients such as nitrogen or phosphorus are in limited supply.
Sediment and Soil: The most important biodegradation processes involve the conversion of the *n*-hexane to primary alcohols, aldehydes and, ultimately, into fatty acids. Similar processes are encountered with other light hydrocarbons such as heptane. In general, unless the *n*-hexane is buried at some depth within a soil or sediment, volatilisation is generally assumed to occur at a much more rapid rate than chemical or biochemical degradation processes. Once introduced into deeper sediments, *n*-hexane may be fairly persistent.
Ecotoxicity:
Fish LC50 (96 h): Oncorhyncus mykiss 4.14 mg/l; Pimephales promelus 2.5 mg/l (flow through); Lepomis macrochirus 4.12 mg/l
Daphnia EC50 (48 h): 3.87 mg/l
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
methyl acetate	LOW	LOW
n-hexane	LOW	LOW
cyclohexane	HIGH (Half-life = 360 days)	LOW (Half-life = 3.63 days)
bisphenol A diglycidyl ether	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
methyl acetate	LOW (LogKOW = 0.18)
n-hexane	MEDIUM (LogKOW = 3.9)
cyclohexane	LOW (BCF = 242)
bisphenol A diglycidyl ether	MEDIUM (LogKOW = 3.8446)

Mobility in soil

Ingredient	Mobility
methyl acetate	MEDIUM (KOC = 3.324)

Continued...

Ingredient	Mobility
n-hexane	LOW (KOC = 149)
cyclohexane	LOW (KOC = 165.5)
bisphenol A diglycidyl ether	LOW (KOC = 1767)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible. ▸ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▸ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material). ▸ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	

Land transport (DOT)

UN number	1133
UN proper shipping name	Adhesives, containing a flammable liquid
Transport hazard class(es)	Class: 3 Subrisk: Not Applicable
Packing group	II
Environmental hazard	Environmentally hazardous
Special precautions for user	Hazard Label: 3 Special provisions: 149, B52, IB2, T4, TP1, TP8

Air transport (ICAO-IATA / DGR)

UN number	1133
UN proper shipping name	Adhesives containing flammable liquid
Transport hazard class(es)	ICAO/IATA Class: 3 ICAO / IATA Subrisk: Not Applicable ERG Code: 3L
Packing group	II

Continued...

Environmental hazard	Environmentally hazardous
Special precautions for user	Special provisions: A3 Cargo Only Packing Instructions: 364 Cargo Only Maximum Qty / Pack: 60 L Passenger and Cargo Packing Instructions: 353 Passenger and Cargo Maximum Qty / Pack: 5 L Passenger and Cargo Limited Quantity Packing Instructions: Y341 Passenger and Cargo Limited Maximum Qty / Pack: 1 L

Sea transport (IMDG-Code / GGVSee)

UN number	1133
UN proper shipping name	ADHESIVES containing flammable liquid
Transport hazard class(es)	IMDG Class: 3 IMDG Subrisk: Not Applicable
Packing group	II
Environmental hazard	Marine Pollutant
Special precautions for user	EMS Number: F-E, S-D Special provisions: Not Applicable 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
methyl acetate	Not Available
n-hexane	Not Available
naphtha petroleum, light, hydrotreated	Not Available
cyclohexane	Not Available
rosin/ isophthalic acid/ pentaerythritol	Not Available
bisphenol A diglycidyl ether	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
methyl acetate	Not Available
n-hexane	Not Available
naphtha petroleum, light, hydrotreated	Not Available
cyclohexane	Not Available
rosin/ isophthalic acid/ pentaerythritol	Not Available
bisphenol A diglycidyl ether	Not Available

SECTION 15 Regulatory information

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

methyl acetate is found on the following regulatory lists

US - Massachusetts - Right To Know Listed Chemicals
US DOE Temporary Emergency Exposure Limits (TEELs)
US NIOSH Recommended Exposure Limits (RELs)
US OSHA Permissible Exposure Limits (PELs) Table Z-1
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances
US TSCA Section 4/12 (b) - Sunset Dates/Status

n-hexane is found on the following regulatory lists

Continued...

Chemical Footprint Project - Chemicals of High Concern List	US DOE Temporary Emergency Exposure Limits (TEELs)
US - California Hazardous Air Pollutants Identified as Toxic Air Contaminants	US EPA Integrated Risk Information System (IRIS)
US - California Proposition 65 - Maximum Allowable Dose Levels (MADLs) for Chemicals Causing Reproductive Toxicity	US EPCRA Section 313 Chemical List
US - California Proposition 65 - Reproductive Toxicity	US NIOSH Recommended Exposure Limits (RELs)
US - California Safe Drinking Water and Toxic Enforcement Act of 1986 - Proposition 65 List	US OSHA Permissible Exposure Limits (PELs) Table Z-1
US - Massachusetts - Right To Know Listed Chemicals	US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US ATSDR Minimal Risk Levels for Hazardous Substances (MRLs)	US TSCA Chemical Substance Inventory - Interim List of Active Substances
US Clean Air Act - Hazardous Air Pollutants	

naphtha petroleum, light, hydrotreated is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List	US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs	US TSCA Chemical Substance Inventory - Interim List of Active Substances
US DOE Temporary Emergency Exposure Limits (TEELs)	

cyclohexane is found on the following regulatory lists

US - Massachusetts - Right To Know Listed Chemicals	US NIOSH Recommended Exposure Limits (RELs)
US CWA (Clean Water Act) - List of Hazardous Substances	US OSHA Permissible Exposure Limits (PELs) Table Z-1
US DOE Temporary Emergency Exposure Limits (TEELs)	US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US EPA Integrated Risk Information System (IRIS)	US TSCA Chemical Substance Inventory - Interim List of Active Substances
US EPCRA Section 313 Chemical List	US TSCA Section 4/12 (b) - Sunset Dates/Status

rosin/ isophthalic acid/ pentaerythritol is found on the following regulatory lists

US List of Active Substances Exempt from the TSCA Inventory Notifications (Active-Inactive) Rule	US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

bisphenol A diglycidyl ether is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List	US NIOSH Recommended Exposure Limits (RELs)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs	US OSHA Permissible Exposure Limits (PELs) Table Z-1
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)	US OSHA Permissible Exposure Limits (PELs) Table Z-3
US - Alaska Air Quality Control - Concentrations Triggering an Air Quality Episode for Air Pollutants Other Than PM-2.5	US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US - California - Biomonitoring - Priority Chemicals	US TSCA Chemical Substance Inventory - Interim List of Active Substances
US DOE Temporary Emergency Exposure Limits (TEELs)	US TSCA Section 4/12 (b) - Sunset Dates/Status

Federal Regulations

Superfund Amendments and Reauthorization Act of 1986 (SARA)

Section 311/312 hazard categories

Flammable (Gases, Aerosols, Liquids, or Solids)	Yes
Gas under pressure	No
Explosive	No
Self-heating	No
Pyrophoric (Liquid or Solid)	No
Pyrophoric Gas	No
Corrosive to metal	No
Oxidizer (Liquid, Solid or Gas)	No
Organic Peroxide	No
Self-reactive	No
In contact with water emits flammable gas	No
Combustible Dust	No
Carcinogenicity	No
Acute toxicity (any route of exposure)	No
Reproductive toxicity	Yes
Skin Corrosion or Irritation	Yes
Respiratory or Skin Sensitization	Yes
Serious eye damage or eye irritation	Yes
Specific target organ toxicity (single or repeated exposure)	Yes
Aspiration Hazard	Yes
Germ cell mutagenicity	No
Simple Asphyxiant	No
Hazards Not Otherwise Classified	No

US. EPA CERCLA Hazardous Substances and Reportable Quantities (40 CFR 302.4)

Name	Reportable Quantity in Pounds (lb)	Reportable Quantity in kg
n-hexane	5000	2270

Continued...

Name	Reportable Quantity in Pounds (lb)	Reportable Quantity in kg
cyclohexane	1000	454

State Regulations

US. California Proposition 65

WARNING: This product can expose you to chemicals including **n-hexane**, which is known to the State of California to cause birth defects or other reproductive harm. For more information, go to www.P65Warnings.ca.gov.

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (methyl acetate; n-hexane; naphtha petroleum, light, hydrotreated; cyclohexane; rosin/ isophthalic acid/ pentaerythritol; bisphenol A diglycidyl ether)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (naphtha petroleum, light, hydrotreated)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (rosin/ isophthalic acid/ pentaerythritol; bisphenol A diglycidyl ether)
Vietnam - NCI	No (rosin/ isophthalic acid/ pentaerythritol)
Russia - FBEPH	No (rosin/ isophthalic acid/ pentaerythritol)
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.*

SECTION 16 Other information

Revision Date	07/11/2022
Initial Date	10/27/2021

CONTACT POINT

PLEASE NOTE THAT TITANIUM DIOXIDE IS NOT PRESENT IN CLEAR OR NEUTRAL BASES

SDS Version Summary

Version	Date of Update	Sections Updated
2.4	07/11/2022	Ingredients

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
AIIC: Australian Inventory of Industrial Chemicals
DSL: Domestic Substances List
NDSL: Non-Domestic Substances List
IECSC: Inventory of Existing Chemical Substance in China
EINECS: European INventory of Existing Commercial chemical Substances
ELINCS: European List of Notified Chemical Substances
NLP: No-Longer Polymers
ENCS: Existing and New Chemical Substances Inventory
KECI: Korea Existing Chemicals Inventory
NZIoC: New Zealand Inventory of Chemicals

Continued...

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

Powered by AuthorITe, from Chemwatch.

end of SDS