FULL RESEARCH REPORT : AMBER

E85 - FUEL ETHANOL

1. IDENTIFICATION OF THE MATERIAL AND SUPPLIER

1.1 Product identifier

Product name	**E85 - FUEL ETHANOL**
Synonym(s)	DENATURED ETHANOL • E85 • FUEL ETHANOL • WIL0003201 - REFERENCE NUMBER

1.2 Uses and uses advised against

Use(s)	FUEL • SOLVENT

1.3 Details of the supplier of the product

Supplier name	**WILMAR BIOETHANOL (AUSTRALIA) PTY LTD**
Address	265 Whitehall St, Yarraville, VIC, Australia, 3013
Telephone	(03) 9283 4850; 1800 819 618
Fax	(03) 8660 2839; 1800 647 260
Email	bioethanol@wilmar.com.au
Website	http://www.wilmarbioethanol.com/

1.4 Emergency telephone number(s)

Emergency	1800 774 557 • 13 11 26 (available in Australia only)

2. HAZARDS IDENTIFICATION

2.1 Classification of the substance or mixture

CLASSIFIED AS HAZARDOUS ACCORDING TO SAFE WORK AUSTRALIA CRITERIA

GHS Classification(s)

Flammable Liquids: Category 2
Aspiration Hazard: Category 1
Skin Corrosion/Irritation: Category 2
Serious Eye Damage / Eye Irritation: Category 2A
Specific Target Organ Toxicity (Single Exposure): Category 3 (Narcotic Effects)
Aquatic Toxicity (Chronic): Category 3

2.2 GHS Label elements

Signal word: **DANGER**

Pictograms

Hazard statement(s)

H225	Highly flammable liquid and vapour.
H304	May be fatal if swallowed and enters airways.
H315	Causes skin irritation.
H319	Causes serious eye irritation.
H336	May cause drowsiness or dizziness.
H412	Harmful to aquatic life with long lasting effects.

Prevention statement(s)

P210	Keep away from heat/sparks/open flames/hot surfaces. No smoking.
P233	Keep container tightly closed.
P240	Ground/bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting equipment.
P243	Take precautionary measures against static discharge.
P261	Avoid breathing dust/fume/gas/mist/vapours/spray.
P264	Wash thoroughly after handling.
P271	Use only outdoors or in a well-ventilated area.
P273	Avoid release to the environment.
P280	Wear protective gloves/protective clothing/eye protection/face protection.

Response statement(s)

P301 + P310	IF SWALLOWED: Immediately call a POISON CENTER or doctor/physician.
P303 + P361 + P353	IF ON SKIN (or hair): Remove/Take off immediately all contaminated clothing. Rinse skin with water/shower.
P304 + P340	IF INHALED: Remove to fresh air and keep at rest in a position comfortable for breathing.
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 or doctor/physician if you feel unwell.
P321	Specific treatment is advised - see first aid instructions.
P331	Do NOT induce vomiting.
P332 + P337 + P313	If skin or eye irritation occurs: Get medical advice/ attention.
P362	Take off contaminated clothing and wash before re-use.
P370 + P378	In case of fire: Use appropriate media for extinction.
Storage statement(s)	
P403 + P233 + P235	Store in a well-ventilated place. Keep cool. Keep container tightly closed.
P405	Store locked up.
Disposal statement(s)	
P501	Dispose of contents/container in accordance with relevant regulations.

2.3 Other Hazards

None known.

3. COMPOSITION/ INFORMATION ON INGREDIENTS

3.1 Substances / Mixtures

Ingredient	CAS number	EC number	Content
ETHANOL	64-17-5	200-578-6	85%
GASOLINE	86290-81-5	289-220-8	15%
DYE(S)	Not Available	Not Available	<0.1%
CORROSION INHIBITOR(S)	Not Available	Not Available	<0.1%
NON HAZARDOUS INGREDIENTS	Not Available	Not Available	Remainder

4. FIRST AID MEASURES

4.1 Description of first aid measures

Eye	If in eyes, hold eyelids apart and flush continuously with running water. Continue flushing until advised to stop by a Poisons Information Centre, a doctor, or for at least 15 minutes.
Inhalation	If inhaled, remove from contaminated area. Apply artificial respiration if not breathing.
Skin	If skin or hair contact occurs, remove contaminated clothing and flush skin and hair with running water. Continue flushing with water until advised to stop by a Poisons Information Centre or a doctor.
Ingestion	For advice, contact a Poisons Information Centre on 13 11 26 (Australia Wide) or a doctor (at once). If swallowed, do not induce vomiting. Rinse mouth with water provided person is conscious.
First aid facilities	Eye wash facilities and safety shower should be available.

4.2 Most important symptoms and effects, both acute and delayed

Chronic exposure may result in cirrhosis of the liver. Over exposure may result in central nervous system (CNS) depression, with nausea, dizziness and unconsciousness at high levels.

4.3 Immediate medical attention and special treatment needed

Treat symptomatically.

5. FIREFIGHTING MEASURES

5.1 Extinguishing media

Water fog or foam. Prevent contamination of drains and waterways.

5.2 Special hazards arising from the substance or mixture

Highly flammable. May evolve toxic gases (carbon oxides, hydrocarbons) when heated to decomposition. Vapour may form explosive mixtures with air. Eliminate all ignition sources including cigarettes, open flames, spark producing switches/tools, heaters, naked lights, pilot lights, mobile phones, etc when handling. Earth containers when dispensing fluids. May evolve nitrogen oxides when heated to decomposition.

5.3 Advice for firefighters

Evacuate area and contact emergency services. Toxic gases may be evolved in a fire situation. Remain upwind and notify those downwind of hazard. Wear full protective equipment including Self Contained Breathing Apparatus (SCBA) when combating fire. Use waterfog to cool intact containers and nearby storage areas.

5.4 Hazchem code

•3YE

•	Alcohol Resistant Foam is the preferred firefighting medium. Else use;
3	Normal Foam (protein based foam that is not alcohol resistant).
Y	Risk of violent reaction or explosion. Wear full fire kit and breathing apparatus. Contain spill and run-off.
E	Evacuation of people in and around the immediate vicinity of the incident should be considered.

6. ACCIDENTAL RELEASE MEASURES

6.1 Personal precautions, protective equipment and emergency procedures

Wear Personal Protective Equipment (PPE) as detailed in section 8 of the SDS. Clear area of all unprotected personnel. Ventilate area where possible. Contact emergency services where appropriate.

6.2 Environmental precautions

Prevent product from entering drains and waterways.

6.3 Methods of cleaning up

Contain spillage, then cover / absorb spill with non-combustible absorbent material (vermiculite, sand, or similar), collect and place in suitable containers for disposal. Eliminate all sources of ignition.

6.4 Reference to other sections

See Sections 8 and 13 for exposure controls and disposal.

7. HANDLING AND STORAGE

7.1 Precautions for safe handling

Before use carefully read the product label. Use of safe work practices are recommended to avoid eye or skin contact and inhalation. Observe good personal hygiene, including washing hands before eating. Prohibit eating, drinking and smoking in contaminated areas.

7.2 Conditions for safe storage, including any incompatibilities

Store in a cool, dry, well ventilated area, removed from incompatible substances, heat or ignition sources and foodstuffs. Ensure containers are adequately labelled, protected from physical damage and sealed when not in use. Check regularly for leaks or spills. Large storage areas should have appropriate fire protection systems. Store removed from direct sunlight.

7.3 Specific end use(s)

None known.

8. EXPOSURE CONTROLS/ PERSONAL PROTECTION

8.1 Control parameters

Exposure standards

Ingredient	Reference	TWA		STEL	
		ppm	mg/m³	ppm	mg/m³
Ethanol	SWA [AUS]	1000	1880	--	--
Ethanol (Ethyl alcohol)	SWA [Proposed]	200	380	800	1500
Petrol (gasoline)	SWA [AUS]	--	900	--	--

Biological limits

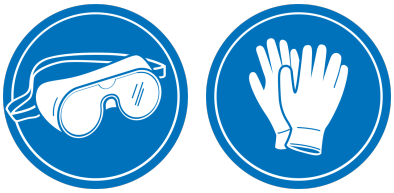
No biological limit values have been entered for this product.

8.2 Exposure controls

Engineering Controls: Avoid inhalation. Use in well ventilated areas. Where an inhalation risk exists, mechanical explosion proof extraction ventilation is recommended. Flammable/explosive vapours may accumulate in poorly ventilated areas. Vapours are heavier than air and may travel some distance to an ignition source and flash back. Maintain vapour levels below the recommended exposure standard.

PPE

- Eye/Face: Wear splash-proof goggles.
- Hand: Wear nitrile or neoprene gloves.
- Body: When using large quantities or where heavy contamination is likely, wear coveralls.
- Respiratory: Where an inhalation risk exists, wear a Type A (Organic vapour) respirator. At high vapour levels, wear Self Contained Breathing Apparatus (SCBA) or an Air-line respirator.



9. PHYSICAL AND CHEMICAL PROPERTIES

9.1 Information on basic physical and chemical properties

Appearance	CLEAR COLOURLESS LIQUID
Odour	CHARACTERISTIC FUEL ODOUR
Flammability	HIGHLY FLAMMABLE
Flash point	13°C (Ethanol) (cc)

Boiling point	78°C
Melting point	-117°C (Ethanol)
Evaporation rate	2.53 (n-Butyl acetate = 1)
pH	NOT AVAILABLE
Vapour density	1.59 (Air = 1) (Ethanol)
Relative density	0.73 to 0.81 (Approximately)
Solubility (water)	SOLUBLE
Vapour pressure	44 mm Hg @ 20°C (Ethanol)
Upper explosion limit	19 % (Ethanol)
Lower explosion limit	3.5 % (Ethanol)
Partition coefficient	NOT AVAILABLE
Autoignition temperature	392°C (Ethanol)
Decomposition temperature	NOT AVAILABLE
Viscosity	< 21 cSt @ 40°C
Explosive properties	NOT AVAILABLE
Oxidising properties	NOT AVAILABLE
Odour threshold	NOT AVAILABLE

9.2 Other information

None known.

10. STABILITY AND REACTIVITY

10.1 Reactivity

Carefully review all information in sections 10.2 to 10.6.

10.2 Chemical stability

Stable under recommended conditions of storage.

10.3 Possibility of hazardous reactions

Polymerization is not expected to occur.

10.4 Conditions to avoid

Avoid heat, sparks, open flames and other ignition sources.

10.5 Incompatible materials

Incompatible with oxidising agents (e.g. hypochlorites), heat and ignition sources.

10.6 Hazardous decomposition products

May evolve toxic gases (carbon oxides, hydrocarbons) when heated to decomposition. May evolve nitrogen oxides when heated to decomposition.

11. TOXICOLOGICAL INFORMATION

11.1 Information on toxicological effects

Acute toxicity **Information available for the product:**

Based on available data, the classification criteria are not met.

Information available for the ingredient(s):

Ingredient	Oral Toxicity (LD50)	Dermal Toxicity (LD50)	Inhalation Toxicity (LC50)
ETHANOL	3450 mg/kg (mouse)	--	20000 ppm/10 hours (rat)
GASOLINE	60 mL/kg (mouse)	--	--

Skin	Contact may result in drying and defatting of the skin, rash and dermatitis.
Eye	Causes serious eye irritation. Contact may result in irritation, lacrimation, pain and redness.
Sensitisation	Not classified as causing skin or respiratory sensitisation.
Mutagenicity	Not classified as a mutagen.
Carcinogenicity	Not classified as a carcinogen.
Reproductive	Not classified as a reproductive toxin.
STOT - single exposure	Over exposure may result in central nervous system (CNS) depression, with nausea, dizziness and unconsciousness at high levels.
STOT - repeated exposure	Not classified as causing organ damage from repeated exposure. However, repeated oral overexposure to ethanol may result in cirrhosis of the liver.
Aspiration	Aspiration or inhalation may cause chemical pneumonitis and pulmonary oedema.

12. ECOLOGICAL INFORMATION

12.1 Toxicity

Harmful to aquatic life with long lasting effects.

12.2 Persistence and degradability

Ethanol will oxidise quickly (less than a few days), with carbon dioxide and water as the final products. Ethanol present in soil or water will decompose in the presence of oxygen.

12.3 Bioaccumulative potential

Ethanol is not expected to bioconcentrate.

12.4 Mobility in soil

Ethanol is carried in the water and air. It is soluble in water and is volatile, so it can be carried quite long distances.

12.5 Results of PBT and vPvB assessment

No information provided.

12.6 Other adverse effects

No information provided.

13. DISPOSAL CONSIDERATIONS

13.1 Waste treatment methods

Waste disposal	For small amounts, absorb with sand, vermiculite or similar and dispose of to an approved landfill site. Contact the manufacturer/supplier for additional information if disposing of large quantities (if required). Prevent contamination of drains and waterways as aquatic life may be threatened and environmental damage may result.
Legislation	Dispose of in accordance with relevant local legislation.

14. TRANSPORT INFORMATION

CLASSIFIED AS A DANGEROUS GOOD BY THE CRITERIA OF THE ADG CODE

	Land Transport (ADG)	Sea Transport (IMDG/IMO)	Air Transport (IATA/ICAO)
14.1 UN number	3475	3475	3475
14.2 UN proper shipping name	ETHANOL AND GASOLINE MIXTURE or ETHANOL AND MOTOR SPIRIT MIXTURE or ETHANOL AND PETROL MIXTURE, with more than 10% ethanol		
14.3 Transport hazard classes	3	3	3
14.4 Packing group	II	II	II

14.5 Environmental hazards Not a Marine Pollutant.

14.6 Special precautions for user

Hazchem Code	•3YE
EMS	F-E, S-E

15. REGULATORY INFORMATION

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

Poison schedule	A poison schedule number has not been allocated to this product using the criteria in the Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP).
Classifications	Safework Australia criteria is based on the Globally Harmonised System (GHS) of Classification and Labelling of Chemicals. The classifications and phrases listed below are based on the Approved Criteria for Classifying Hazardous Substances [NOHSC: 1008(2004)]
Inventory listing(s)	**AUSTRALIA: AIIC (Australian Inventory of Industrial Chemicals)** All components are listed on AIIC, or are exempt.

15.2 Chemical safety assessment

No information provided.

16. OTHER INFORMATION

Additional information HEALTH EFFECTS FROM EXPOSURE:

Product name **E85 - FUEL ETHANOL**

It should be noted that the effects from exposure to this product will depend on several factors including: form of product; frequency and duration of use; quantity used; effectiveness of control measures; protective equipment used and method of application. Given that it is impractical to prepare a report which would encompass all possible scenarios, it is anticipated that users will assess the risks and apply control methods where appropriate.

PERSONAL PROTECTIVE EQUIPMENT GUIDELINES:
The recommendation for protective equipment contained within this report is provided as a guide only. Factors such as form of product, method of application, working environment, quantity used, product concentration and the availability of engineering controls should be considered before final selection of personal protective equipment is made.

Abbreviations

ACGIH	American Conference of Governmental Industrial Hygienists
CAS #	Chemical Abstract Service number - used to uniquely identify chemical compounds
CNS	Central Nervous System
EC No.	EC No - European Community Number
EMS	Emergency Schedules (Emergency Procedures for Ships Carrying Dangerous Goods)
GHS	Globally Harmonized System
GTEPG	Group Text Emergency Procedure Guide
IARC	International Agency for Research on Cancer
LC50	Lethal Concentration, 50% / Median Lethal Concentration
LD50	Lethal Dose, 50% / Median Lethal Dose
mg/m³	Milligrams per Cubic Metre
OEL	Occupational Exposure Limit
pH	relates to hydrogen ion concentration using a scale of 0 (high acidic) to 14 (highly alkaline).
ppm	Parts Per Million
STEL	Short-Term Exposure Limit
STOT-RE	Specific target organ toxicity (repeated exposure)
STOT-SE	Specific target organ toxicity (single exposure)
SUSMP	Standard for the Uniform Scheduling of Medicines and Poisons
SWA	Safe Work Australia
TLV	Threshold Limit Value
TWA	Time Weighted Average

Report Status

This ChemAlert report has been independently compiled by RMT's scientific department utilising the original Safety Data Sheet ('SDS') for the product provided to RMT by the manufacturer. The information is based on the latest chemical and toxicological research and is believed to represent the current state of knowledge as to the appropriate safety and handling precautions for the product at the time of issue. It is an independent collation by RMT of information obtained from the original SDS for this product. Its content has not been authorised or verified by the manufacturer / distributor of the chemical to which it relates.

This ChemAlert report does not constitute the manufacturer's original SDS and is not intended to be a replacement for same. It is provided to subscribers of ChemAlert as a reference tool only, is not all-inclusive and does not represent any guarantee as to the properties of the product. Further clarification regarding any aspect of the product should be obtained directly from the manufacturer.

While RMT has taken all due care to include accurate and up-to-date information in this ChemAlert report, it does not provide any warranty as to accuracy or completeness. As far as lawfully possible, RMT accepts no liability for any loss, injury or damage (including consequential loss) which may be suffered or incurred by any person as a consequence of their reliance on the information contained in this ChemAlert report.

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Product name	**E85 - FUEL ETHANOL**

Last Reviewed:	15-Dec-2020
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Based on SDS dated:	27-Nov-2020

End of Report

CHEMICAL FOOTPRINT

The following Chemical Footprint (CFP) assessment aims to provide further clarity surrounding the long-term health and environmental impact of hazardous chemicals.

Overall	45
CMR	15
Endocrine	0
Sensitising	0
Physical	75
Environment	50
Waste	71

INGREDIENT TOXICOLOGICAL DATA

ETHANOL (64-17-5) 85%

Physicochemical	Highly flammable. Flash Point ~ 13°C. Lower Explosive Limit (LEL): 3.3 % (NTP, 1992) Upper Explosive Limit (UEL): 19 % (NTP, 1992) Soluble in water in all proportions. ETHANOL reacts violently with acetyl chloride and acetyl bromide [Rose, (1961); Merck 11th ed., 1989]. Mixtures with concentrated sulfuric acid and strong hydrogen peroxide can cause explosions. Mixtures with concentrated hydrogen peroxide form powerful explosives. Reacts readily with hypochlorous acid and with chlorine to give ethyl hypochlorite, which decomposes in the cold and explodes on exposure to sunlight or heat. Base-catalysed reactions with isocyanates should be carried out in inert solvents. Such reactions in the absence of solvents often occur with explosive violence [Wischmeyer(1969)]. Highly oxidized potassium metal was dropped into a dish of ethyl alcohol, an immediate explosion shattered the dish. Potassium superoxide was considered the cause of the reaction [Health and Safety Inf. 251(1967)]. Ethanol or methanol can ignite on contact with a platinum-black catalyst. (Urben 1794). Source: Cameo Chemicals.
Acute Toxicity	Oral: The chemical has low acute toxicity by oral exposure in animal tests. The median lethal dose (LD50) in rats is >2000 mg/kg bw. Observed sub-lethal effects included central nervous system depression, e.g. inebriation, disturbances of gait, dose-related decreases in responses to painful stimuli, respiratory depression, and coma. Deaths were reported due to cardiorespiratory failure (OECD, 2005; HSDB; REACH). Dermal: Few studies are available on the dermal toxicity of the chemical. A poorly documented rabbit study reported death in one of four animals following a dose of 20000 mg/kg bw. Although limited data are available, the apparent low dermal toxicity from this study is regarded as consistent with low uptake of ethanol through intact skin. The median lethal dose (LD50) in rats is greater than 2000 mg/kg bw. Observed sub-lethal effects were not reported for the study (OECD, 2005; REACH). Inhalation: The chemical has low acute toxicity by inhalation exposure in animal tests. The lowest reported median lethal concentration (LC50) is 124.7 mg/L/four hours in rats. Observed sub-lethal effects included attempts to escape, reddish-watery eyes, nasal secretions, closing of eyelids, snout wiping, intermittent respiration, loss of pain reflex, abdominal position, and apathy (OECD, 2005; REACH). Observation in humans: Consumption of beverages containing the chemical has been associated with symptoms of intoxication (drowsiness, loss of concentration). However, there is no evidence of such symptoms occurring following dermal or inhalation exposures (OECD, 2005; HSDB). Source: AICIS (HUMAN HEALTH TIER II ASSESSMENT FOR Ethanol).
Skin corrosion / irritation	In a skin irritation study conducted in accordance with OECD Test Guideline (TG) 404, the chemical was applied to six New Zealand White rabbits for four hours using exposure chambers. The mean score for erythema was one at 24 hours and remained zero at all other time points (48, 72 hours); the mean score for oedema remained zero at all time points (24, 48, 72 hours). The chemical was concluded not to be irritating to the skin of rabbits. Another skin irritation study in rabbits, where the chemical was applied under occlusion for 24 hours, also showed only very slight skin irritation (OECD, 2005; REACH). Source: AICIS (HUMAN HEALTH TIER II ASSESSMENT FOR Ethanol).
Serious eye damage / irritation	In an eye irritation study conducted in accordance with US Federal guideline (Fed. Reg. Vol. 38, No. 187, 1973), the chemical (0.1 mL) was applied on the conjuctival sac of one eye of each of three New Zealand White rabbits. Irritation responses were observed at 24, 48 and 72 hours and eight days following application. Mean Draize scores following grading at 24, 48 and 72 hours for three rabbits were 1 for corneal opacity, 0.22 for iritis, 2.45 for conjunctivitis, and 1.89 for chemosis. Mean Draize scores following grading at day eight were 0.67 for corneal opacity, 1.67 for conjunctivitis, and 1.33 for chemosis. While iris lesions were fully reversible by day eight, other eye lesions were not fully reversible at this time. Given the observation period did not extend to 21 days, it is difficult to conclude any findings on the reversibility of the

and chemosis (³2) observed, that classification as an eye irritant is warranted (REACH).

In another eye irritation study (OECD TG 405), the chemical (0.1 mL) was applied to the eyes of three rabbits (strain not specified) and observed up to 14 days. Mean Draize scores at 24, 48 and 72 hours were 2.11 for conjunctivitis, 1.33 for chemosis, 0.44 for iritis, and 1.11 for corneal opacity. Although all symptoms subsided by day 14, conjunctivitis was still present at day seven. As positive responses for corneal opacity (mean score >1 for 2/3 animals) and conjunctival redness (mean score >2 for 2/3 animals) were noted in the study, the chemical is considered to be an eye irritant (category 2A) (OECD, 2005; REACH).

In an eye irritation study (OECD TG 405), the chemical (0.1 mL) was applied into the lower conjunctival sac of one eye of six New Zealand White rabbits and observed up to 72 hours. Reported average Draize scores at 24, 48 and 72 hours were 2.39 for redness of the conjunctivae, 1.2 for chemosis, 0.28 for iritis, and 1.2 for corneal opacity. As conjuctival redness persisted for 24 hours with a mean score of >2 and corneal opacity was noted with a mean score >1, the chemical is considered to be an eye irritant (category 2A) (OECD, 2005; REACH).

In an eye irritation study conducted in accordance with US Federal guideline (Fed. Reg. 28 (119), 5582, 1963), the chemical (0.1 mL) was applied on the lower lid of one eye of six New Zealand White rabbits. The eyes were examined at 24, 48, and 72 hours and at day seven following administration of the chemical. Mean Draize scores following grading at 24, 48 and 72 hours were 1.72 for conjunctivitis, 1.78 for chemosis, 0.83 for iritis, and 1.28 for corneal opacity. While iris lesions were fully reversible at day seven, other eye lesions were not. Mean Draize scores following grading at day seven were 0.83 for conjunctivitis, 0.83 for chemosis, and 1.17 for corneal opacity. As corneal opacity was noted with a mean score >1, the chemical is considered an eye irritant (category 2A). In addition, whilst mean scores for conjunctival redness and chemosis were <2, scores ³2 were noted in four out of six animals (OECD, 2005; REACH).

Source: AICIS (HUMAN HEALTH TIER II ASSESSMENT FOR Ethanol).

Sensitisation

The available data indicate that the chemical does not induce skin sensitisation in animals.

The chemical, at 75 % concentration, was used as a solvent in a Magnusson and Kligman guinea pig maximisation test of a polyalkalene glycol. Skin reactions were not observed at challenge with the polyalkalene glycol in 75 % ethanol in either the test or negative control animals (OECD, 2005). In a mouse ear swelling test, no increase in ear thickness was observed following a challenge application of the chemical at 95 % (OECD, 2005; REACH).

In a mouse local lymph node assay (LLNA) (OECD TG429) the chemical, or diethyl phthalate, were used as vehicles to examine the skin sensitisation potential of four test fragrance materials. The concentration of the chemical in this study varied from 0–100 %. The level of induced T-lymphocyte proliferation was low for the chemical compared with that for fragrance materials known to be mild to moderate skin sensitisers, and comparable with the other negative control vehicle (diethyl phthalate). On the basis of a lack of sensitising potential up to a concentration of 100 %, the test concluded that the chemical is an appropriate vehicle for use in a local lymph node assay (REACH).

Source: AICIS (HUMAN HEALTH TIER II ASSESSMENT FOR Ethanol).

Mutagenicity

Overall, the data indicate that the chemical has no mutagenic or genotoxic potential (OECD, 2005; REACH).

The results from numerous bacterial mutation assays of the chemical have generally been negative. A very weak positive effect of the chemical was found in an Escherichia coli DNA repair test but not in Ames tests with Salmonella typhimurium conducted by the same authors. In separate studies, there have been positive results reported in Ames tests, but only at concentrations of the chemical significantly greater than those specified in test guidelines. The chemical is therefore not considered mutagenic in bacteria.

The chemical has also been tested in several chromosome aberration assays. Many of these studies have limitations such as insufficient dose ranges and lack of metabolic activation. Accordingly, a weight of evidence approach is required to draw conclusions regarding clastogenic potential. No chromosome aberrations were found in testing with human lymphocyte cultures, lymphocyte cell lines, or Chinese hamster ovary (CHO) cells. Chromosome aberrations were detected in CHO cells but only in the presence of metabolic activation using plant microsomal extracts. Collectively, there is little evidence that the chemical is clastogenic in vitro. It has been considered that positive responses with high chemical concentrations may be an artefact, attributable to damage from high osmotic pressures.

The chemical has also been tested in cell mutation (mouse lymphoma) assays with negative results. A statistically significant increase in mutants was reported both in the presence and absence of metabolic activation in a mouse lymphoma assay designed to assess false positive results. However, the mutant frequencies remained low and the result was regarded as negative.

Several in vivo micronucleus assays have assessed the potential for the chemical to induce damage to chromosomes of erythroblasts. No effect was reported in rats when administered 5 % of the chemical (approximately 4 g/kg bw/day) in drinking water, or in mice at up to 40 % (approximately 31 g/kg bw/day). Chemical-related mortality was observed in the latter study. Marginally statistically significant increases in the incidence of micronucleated bone marrow erythrocytes were reported in rats fed for six weeks with a diet containing ethanol at 12–16 g/kg/day. Although there is very limited evidence that the chemical induces micronuclei in the bone marrow of rodents, the chemical has the potential to induce micronuclei in bone marrow erythrocytes at very high doses.

No chromosome aberrations were found in bone marrow or peripheral blood lymphocytes of rats receiving the chemical at up to 15.7 g/kg bw/day in drinking water. Similarly, no chromosome aberrations were found in the bone marrow of Chinese hamsters receiving the chemical in drinking water at up to 20 % for 12 weeks.
Results of dominant lethal assays with the chemical have been mixed. Interpreting the results has been confounded by inadequacies in methodologies, and using high ethanol doses often produced confounding

toxicological effects. The most robust dominant lethal testing was identified as a collaborative inter-laboratory study conducted to OECD test guidelines. In this study, male mice were exposed via intubation to doses of the chemical at and below the maximally tolerated dose. No significant effects were reported. Increased frequencies of chromosome aberrations have been reported in several studies of peripheral blood lymphocytes in alcoholics (IARC, 1998; IARC, 2010).

Source: AICIS (HUMAN HEALTH TIER II ASSESSMENT FOR Ethanol).

Carcinogenicity

Since the conclusion of a causal relationship between consumption of alcoholic beverages and carcinogenicity was published (IARC, 1988), a large number of additional epidemiological studies have reported on the association between alcohol consumption (containing ethanol and water as the two main components) and cancers at various sites (IARC, 2010; IARC, 2012). These indicate that regular alcoholic consumption is associated with an increased risk of malignant tumours of the oral cavity, pharynx (excluding the nasopharynx), larynx, oesophagus, liver, colorectum and female breast. An association between alcohol consumtion and a small increased risk of cancer of the pancreas has also been recently noted (IARC, 2012). Consumption in excess of 10–40 g ethanol a day appears necessary before there is an appreciable increase in relative risk for cancer of oral cavity, pharynx, larynx and oesophagus (OECD, 2005). Daily consumption of 50 g ethanol was associated with a 2–3 fold increase in the risk of upper digestive tract tumours compared with non-drinkers. Similarly, daily consumption of 50 g ethanol was associated with relative risks for colorectal cancer and breast cancer of 1.4 and 1.5 respectively, compared with non-drinkers (IARC, 2010; IARC, 2012).

However, the evidence did not suggest that carcinogenicity is linked to the mutagenic effects of ethanol, acetaldehyde or other beverage constituents (OECD, 2005). The aetiology of cancers of the oral cavity, pharynx (excluding the nasopharynx), larynx and oesophagus is thought to be linked to persistent irritation, hyperplasia and finally tumour formation (OECD, 2005). The aetiology of liver cancer following alcoholic beverage consumption is commonly linked to cirrhosis, normally seen only following chronic intakes of greater than 80g ethanol per day (OECD, 2005). The risk for liver tumours was more difficult to estimate due to the confounding effects of cirrhosis and other liver diseases that often occur before the cancer becomes manifest and lead to reductions in alcohol intake in patients (IARC, 2010; IARC, 2012). The chemical has also been used in ready-to-use mouthwashes in a concentration up to 27 %. The safety of these preparations with respect to carcinogenic effect (increased risk of oral cancer) has been a source of controversy over decades (Lachenmeier DW, 2008; Gardini et al, 2012).

There is sufficient epidemiological evidence showing that humans who are deficient in the oxidation of acetaldehyde to acetate have a substantially increased risk of developing alcohol-related cancers, in particular of the oesophagus and the upper aerodigestive tract.

Source: AICIS (HUMAN HEALTH TIER II ASSESSMENT FOR Ethanol).

Reproductive Toxicity

Effects of alcoholic beverages on reproduction in humans have been extensively reviewed. Alcohol consumption can interfere with both male and female reproductive function through effects on reproductive cells and adverse regulation of sex hormones. Ethanol is a recognised human teratogen. Multiple terms are used to describe a continuum of effects that result from prenatal exposure to ethanol. Foetal alcohol syndrome is the most common description of a collection of the most severe abnormalities linked with alcohol abuse. Abnormalities include pre- and/or postnatal growth retardation, characteristic craniofacial dysmorphology, mental retardation, cardiac septal defects, joint abnormalities and additional alterations in multiple organs and systems (IARC, 2010; IARC, 2012).

Source: AICIS (HUMAN HEALTH TIER II ASSESSMENT FOR Ethanol).

Repeated Dose Toxicity

Many repeated dose studies of chemical have been conducted in many species, predominantly with the aim of assessing adverse effects associated with the consumption of alcoholic beverages. Consequently, these are mostly conducted through oral exposure and with doses well in excess of those that might be encountered in occupational exposure or consumer products (OECD, 2005), or unintentional public exposures from environmental contamination.

Considering the lowest observed adverse effect level (LOAEL) available from a 90-day rat study (3600 mg/kg bw/day), and based on the treatment-related effects reported in various repeated dose toxicity studies, the chemical is not considered to cause serious damage to health from repeated oral exposure, except from exposure to high doses.

In a well-conducted repeated dose toxicity study, the chemical was administered (in a liquid diet) to Sprague Dawley (SD) rats at a 1, 2, 3, 4, 5, and 10 % concentration for 90 days. Water consumption in the 10 % group was reduced relative to controls. There were no adverse clinical signs or mortality during the study. Serum liver enzymes were unaffected by treatment and kidney findings were reported to be minimal. A LOAEL was established at 3 % (approximately 3600 mg/kg bw/day), based on dose-related hepatic yellowing, centrilobular steatosis, increased frequency and severity of Mallory bodies (hyaline), and acidophilic degeneration and necrosis. The no observed adverse effect level (NOAEL) was 2 % (approximately 2400 mg/kg bw/day) (OECD, 2005; REACH).

In another repeated dose toxicity study conducted in accordance with national test guidelines of USA (EPA OPPTS 870.3100), the chemical was administered in drinking water to Fischer 344 (F344) rats and B6C3F1 mice at a single dose of 5 % concentration for 90 days. Even though male rats showed minor changes in thymus weights, and some slight but inconsistent changes in haematology and clinical chemistry, these effects were not considered adverse. Based on water consumption data, this single dose study established a 5 % nominal NOAEL for male rats (approximately 3250 mg/kg bw/day). Although minor changes in clinical chemistry were also seen in female rats, some female rats (4/10) also exhibited liver nodules (diaphragmatic nodules) and small increases in liver weights. As no NOAEL could be established for female rats, a LOAEL of 4400 mg/kg bw/day was established. For male mice, a LOAEL at 9700 mg/kg bw/day was established, based on increased organ weights (liver, heart, kidney and lung) and decreased sperm counts in the cauda epididymis. Although female mice showed small changes in the length of dioestrus and pro-oestrus, the overall cycle length was unchanged. As biological significance of these changes was unclear, a NOAEL for

Product name	**E85 - FUEL ETHANOL**

Source: AICIS (HUMAN HEALTH TIER II ASSESSMENT FOR Ethanol).

Environment	If released to air, a vapor pressure of 59.3 mm Hg at 25 deg C indicates ethanol will exist solely as a vapor in the atmosphere. Vapor-phase Ethanol will be degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 36 hours. Ethanol does not contain chromophores that absorb at wavelengths >290 nm, and therefore is not expected to be susceptible to direct photolysis by sunlight. If released to soil, ethanol is expected to have very high mobility based upon a Koc of 2.75. Volatilization from moist soil surfaces is expected to be an important fate process based upon a Henry's Law constant of 5.0X10-6 atm-cu m/mole. Ethanol may volatilize from dry soil surfaces based upon its vapor pressure. Ethanol was biodegraded with half-lives on the order of a few days using microcosms constructed with a low organic sandy soil and groundwater, indicating that biodegradation is an important environmental fate process in soil and water. If released into water, ethanol is not expected to adsorb to suspended solids and sediment based upon the Koc. Volatilization from water surfaces is expected to be an important fate process based upon this compound's Henry's Law constant. Estimated volatilization half-lives for a model river and model lake are 5 and 39 days, respectively. An estimated BCF of 3 suggests the potential for bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyze under environmental conditions (pH 5 to 9).

Source: HSDB.

GASOLINE (86290-81-5) 15%

Physicochemical	GASOLINE may be incompatible with strong oxidising agents such as nitric acid, peroxides, and perchlorates. Charring may occur followed by ignition of unreacted hydrocarbon and other nearby combustibles. In other settings, mostly unreactive. Not affected by aqueous solutions of acids, alkalis, most oxidising agents, and most reducing agents. When heated sufficiently or when ignited in the presence of air, oxygen or strong oxidising agents, burns exothermically to produce carbon dioxide and water.

End of Additional Research Data