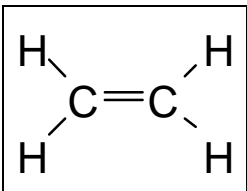


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**ETHYLENE**  
**CAS N°: 74-85-1**

## SIDS INITIAL ASSESSMENT PROFILE

CAS No.	74-85-1
CHEMICAL NAME	Ethylene
STRUCTURAL FORMULA	
<u>CONCLUSIONS</u>	
<p><u>Environment:</u> Ethylene is, due to its physical and chemical properties released mainly into the atmospheric compartment. About three quarters of atmospheric ethylene originates from natural sources, while one quarter is from anthropogenic sources. The main anthropogenic release is from burning of hydrocarbons and biomass.</p> <p>It has been well documented through relevant toxicity studies that the minute amounts measured in water implies little environmental hazard to the organisms in this compartment.</p> <p>In the terrestrial compartment, the vegetation has proven highly susceptible to this gas, probably through a mechanism related to the hormone function of ethylene in plants. Ethylene levels in urban areas have reached levels which inhibit growth of certain plant species. This is of concern both for decorative plants and agriculture in exposed areas. This is mainly due to incomplete burning of coal and petrol products in urban areas. The industrial emission is a minor contribution to total emissions and effects on vegetation are only expected close to production and processing plants.</p>	
<p><u>Human Health:</u></p> <p>Relevant studies have indicated a low toxicity of ethylene and no risk to human health has been identified neither from occupational exposure nor exposure of general public, either exposed directly or indirectly via the environment. However, metabolic studies in animals and man have revealed that ethylene is metabolised to ethylene oxide which is known to have carcinogenic and mutagenic effects.</p>	
<u>RECOMMENDATIONS</u>	
<p>No further testing of ethylene toxicity is recommended.</p> <p>It is recommended to do a closer monitoring of the environmental ethylene levels in urban and polluted regions with respect to its potential intoxication of vegetation, but this goes beyond the OECD HVP Chemical Programme.</p>	

## FULL SIDS SUMMARY - ETHYLENE

CAS NO: 74-85-1		SPECIES	PROTOCOL	RESULTS
<b>PHYSICAL-CHEMICAL</b>				
2.1	Melting Point			- 169.15 °C
2.2	Boiling Point			- 103.71 °C
2.3	Density			0.57 g/m <sup>3</sup> (at boiling point)
2.4	Vapour Pressure			4.27 MPa at 0°C
2.5	Partition Coefficient (Log Pow)			1.13 (calculated)
2.6 A.	Water Solubility			131 mg/l at 20 °C
B.	pH			-
	pKa			-
2.12	Oxidation: Reduction Potential			-
<b>ENVIRONMENTAL FATE AND PATHWAY</b>				
3.1.1	Photodegradation			Lifetime = 0.37 to 4 days best estimate = 1.45 days
3.1.2	Stability in Water			-
3.2	Monitoring Data			<u>In air:</u> Rural areas < 1 - 5 µg/m <sup>3</sup> Urban areas < 50 µg/m <sup>3</sup> Heavy traffic < 1000 µg/m <sup>3</sup> Occupational exposure < 5 mg/m <sup>3</sup> <u>In water:</u> Baseline 6.0 µg/l Polluted areas 44 µg/l
3.3	Transport and Distribution		Calculated (Fugacity Level 1 type)	In Air 99.99915 % In Water - % In Sediment - % In Soil - % In Biota - %
3.5	Biodegradation			<u>Biodegradation in water:</u> Aerobic: T <sub>1/2</sub> = 1 - 28 days Anaerobic: T <sub>1/2</sub> = 72 - 112 days
<b>ECOTOXICOLOGY</b>				
4.1	Acute/Prolonged Toxicity to Fish	Orange spotted sunfish various fish Fathead minnow		LC <sub>50</sub> (1 hr) = 22 mg/l QSAR values: LC <sub>50</sub> (4 days) = 50-119.5 mg/l NOEC (28 days) = 13mg/l

CAS NO: 74-85-1		SPECIES	PROTOCOL	RESULTS
4.2	Acute Toxicity to Aquatic Invertebrates	<i>Daphnia magna</i>		QSAR value: EC <sub>50</sub> (48 hr) = 53 - 152.9 mg/l
4.3	Toxicity to Aquatic Plants e.g. Algae	<i>Selinastrum capricornutum</i>		<u>Growth inhibition:</u> EC <sub>50</sub> ( 72 hr) = 40 mg/l <u>Growth rate inhibition:</u> EC <sub>50</sub> ( 72 hr) = 72 mg/l NOEC ( 72 hr) = 13.9 mg/l
4.5.2	Chronic Toxicity to Aquatic Invertebrates	<i>Daphnia magna</i>		NOECs ( 16 d) = 37.4 mg/l
4.6.1	Toxicity to Soil Dwelling Organisms			-
4.6.2	Toxicity to Terrestrial Plants	Peas, potatos, oats, cotton Cattleya orchid		EC <sub>50</sub> (24 hr) = 8 - 700 µg/m <sup>3</sup> EC <sub>50</sub> (24 hr) = 2.3 µg/m <sup>3</sup>
(4.6.3)	Toxicity to Other Non-Mammalian Terrestrial Species (Including Birds)			-
<b>TOXICOLOGY</b>				
5.1.1	Acute Oral Toxicity			
5.1.2	Acute Inhalation Toxicity	Mice		LD <sub>50</sub> = 950,000 ppm
5.1.3	Acute Dermal Toxicity			-
5.4	Repeated Dose Toxicity	Rat, SD		NOEC > 10,000 ppm
5.5	Genetic Toxicity In Vitro			
A.	Bacterial Test (Gene mutation)	<i>E.Coli</i>		neg. (With metabolic activation) neg. (Without metabolic activation)
B.	Non-Bacterial In Vitro Test (Chromosomal aberrations)	CHO cells	OECD TG 473	neg (With metabolic activation) neg. (Without metabolic activation)
5.6	Genetic Toxicity <i>In Vivo</i>	Mouse	Micronuc. test	neg
		Rat	Micronuc. test	neg
5.7	Carcinogenicity	Rat		2 years inhalation study negative
5.8	Toxicity to Reproduction (Inhalation administration)	Rat	OECD TG 421 Inhalation	NOEL = 5,000 ppm (General toxicity) NOEL = 5,000 ppm (Repro. Tox.)
5.9	Developmental Toxicity/ Teratogenicity			-
5.11	Experience with Human Exposure			Work place exposure ≤ 4 mg/m <sup>3</sup> Peak levels ≤ 50 mg/m <sup>3</sup>

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# SIDS INITIAL ASSESSMENT REPORT

## 1. IDENTITY

<b>Name (IUPAC):</b>	Ethylene
<b>CAS number:</b>	74-85-1
<b>Molecular formula:</b>	CH <sub>2</sub> CH <sub>2</sub>
<b>Molecular Weight:</b>	28.05
<b>Other names:</b>	Ethene, acetene, bicarburetted hydrogen, olefiant gas, elayl.

Even the high volume industrial product is of high purity (about 99.9%). The main impurities are methane and ethane. There are no impurities or additives known to represent any risk of toxicity.

Ethylene is gaseous with a boiling point of -104 °C at atmospheric pressure. Ethylene is stored in the liquid state under high pressure or at low temperatures.

Ethylene has a solubility in water of 131 mg/l at 20 °C. The log Pow value of 1.13 (calculated) indicates only a slightly higher solubility in octanol than in water and no potential for bioaccumulation is indicated.

Classification and labelling of ethylene focus on the flammability and the explosive properties. Within EU ethylene is classified as extremely flammable and should be labelled according to Fx, R12 and correspondingly according to S2-9-16-33.

## 2. GENERAL INFORMATION ON EXPOSURE

Ethylene is the petrochemical produced in largest quantities worldwide. For 1996 the total production volume is estimated to be 83,000,000 tonnes. EU has a production capacity of 18,200,000 tonnes. More than 95% of the annual commercial production of ethylene is currently based on steam cracking of petroleum hydrocarbons.

About 80 % of the ethylene consumed in US, Western Europe and Japan is used for production of ethylene oxide, ethylene dichloride, linear low density and high density polyethylene. Significant amounts are also used to make ethylbenzene, alcohols, olefins, acetaldehyde and vinylacetate. Minor quantities are used as anaesthetic gas, for fruit ripening and for welding and cutting metals.

Industrially produced ethylene is, due to the physical state of the product, kept in closed systems during both production, storage and in further processing. Often production plants utilizing ethylene as raw material are situated close to where ethylene is produced, however large quantities are also traded internationally. Ethylene is transported by sea in gas tankers in liquid form. On land transport mainly by pipelines. Even if the gas is kept in closed systems, transport implies a risk for environmental exposure. In cases when the transport devices maintain the pressure by cooling, the cooling systems may fail, giving higher pressure and leakage through safety valves.

Ethylene is ubiquitous in the environment, arising from both natural and man made sources. Major natural sources are emissions from vegetation of all types, where it functions as a plant hormone.

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The main anthropogenic sources are from combustion of gas, fuel, coal and biomass. The highest exposure concentrations to humans are due to ethylene from car motors. The total global ethylene emission has been estimated to be  $18\text{--}45 \cdot 10^6$  t/y, of which approximately 74 % is released from natural sources and 26 % from anthropogenic sources. Emission from fuel oil combustion is estimated to  $1.54 \cdot 10^6$  t/y, equal to approximately 4 % of total global emissions.

## 2.1 Production volumes, uses and release

Production plants and processing plants for ethylene are often sited together. A medium sized production plant has a capacity of ca. 400 000 tons/y and production runs 365 days/y. Fraction released corresponds to ca. 0.06 % of the production. As plants for processing and use of ethylene are, as already mentioned, assumed to be directly connected to the production site by pipelines, these activities do not contribute significantly to the emissions.

## 3. ENVIRONMENT

### 3.1 Environmental Exposure

#### 3.1.1 General Discussion

Fugacity calculation using a six compartment global reference model shows that emitted ethylene almost exclusively is distributed to air. The most recent methods give a calculated lifetime of 1.45 days for ethylene in the atmosphere, this is the sum of all relevant loss pathways. Other calculations gave lifetimes in the range of 0.37-4 days. Biodegradation in water is however slower with half-lives in the range of 1-28 days, and even slower under anaerobic conditions where half-lives are 3 to 112 days.

No bioaccumulation is expected as the calculated Log  $P_{ow}$  is low ( $\log P_{ow} = 1.13$ ) and the vapour pressure is high. This is confirmed in the fugacity calculation below.

#### 3.1.2 Predicted Environmental Concentration

##### Air

Environmental levels will depend on both anthropogenic and natural sources in the surroundings, but highest levels are measured in urban areas and this is considered due to combustion of fuel, coal and gas. Ethylene concentrations in ambient air at rural and remote sites worldwide are generally in the range of  $< 1 - 5 \text{ mg/m}^3$ . In urban and indoor air contaminated with combustion products, ethylene concentrations typically range from a few up to about  $50 \text{ mg/m}^3$ . However, in extreme cases values above  $1000 \text{ mg/m}^3$  have been measured in heavy traffic.

Local environmental concentration in air due to industrial production and use (production and use usually coexists) can be exemplified by a refinery with a production capacity of 365 000 tons/y. Production is continuous with emissions 365 days/y. Local emissions at the site have been measured to be 24 kg/h equal to 210 tons/y or 0.057 % of total production and use. According to TGD(96) this gives a local concentration of  $160 \text{ g/m}^3$  at a distance of 100 m from the source.

From these observations, the maximum Predicted Environmental Concentration is estimated to be regionally ( $PEC_{\text{regional}}$ )  $5 \text{ mg/m}^3$  and locally ( $PEC_{\text{local}}$ ) for traffic  $1000 \text{ mg/m}^3$  and for industrial production and use  $160 \text{ g/m}^3$ , and general urban areas  $50 \text{ g/m}^3$ .

##### Water

The marine aquatic level of ethylene has been measured in a set of 452 samples. This gave a baseline (average) concentration of 6.0 mg/l, but increasing up to 44 mg/l in heavily exposed areas.

### Environmental partitioning

A fugacity level I calculation, using a six compartment (air, water, soil solids, suspended sediments and fish) model has been conducted using the global reference model of OECD. Default values for the environmental parameters were not changed. Entered generic parameters were: melting point - 169.15 °C, vapour pressure 4.27 MPa, water solubility 200 g/m<sup>3</sup>, log<sub>10</sub>P<sub>OW</sub> 1.13, half-life in air 56 hours, half-life in water, soil and sediment 672 hours. This gave the following distribution:

in air	99.99915 %,
in water	8.27·10 <sup>-4</sup> %,
in soil solids	9.88·10 <sup>-6</sup> %
in sediment solids	2.20·10 <sup>-7</sup> %
in suspended sediments	6.87·10 <sup>-9</sup> %
in fish	5.58·10 <sup>-10</sup> %

This means that for all practical purposes, emitted ethylene is distributed to air only.

The fate of atmospheric ethylene emitted from natural and anthropogenic sources has been estimated by several methods. The most recent published estimates indicate that about 85.4 % was destroyed in the troposphere by reaction with OH radical, and 14.5 % in the reaction with O<sub>3</sub>. The remaining 0.07 % was transported into the stratosphere. The atmospheric lifetime of ethylene was estimated to be 1.45 days. Other estimates of lifetime of ethylene in the atmosphere due to the combined effect of O<sub>3</sub> reaction with ethylene and OH reactions are in the range of 0.37 and 4 days.

## **3.2 Effects on the Environment**

### **3.2.1 Aquatic effects**

There are limited experimental data on the toxicity of ethylene to fish and invertebrates. A rather old study concluded that ethylene concentrations above 22 mg/l might be lethal to orange-spotted sunfish after 1 hour exposure. Calculated values (QSAR) indicate LC<sub>50</sub> values from 50 to 120 mg/l for different fish species after 4 days exposure. Calculated NOEC for fish after 28 days exposure (Fathead minnow) is 13 mg/l.

Calculated LC<sub>50</sub> values for Daphnia range from 53 - 153 mg/l. NOEC after 16 days was 37.4 mg/l.

In order to estimate the toxicity of ethylene to aquatic plants, a growth inhibition test to the alga *Selenastrum capricornutum* was undertaken. Nominal ethylene concentration in the growth medium ranged from 8.2 to 131 mg/l. During the 72 hr exposure period there was a loss of ethylene in the range of 64-91 %, however in calculation of results the mean measured ethylene concentration was used. EC<sub>50</sub> for the growth inhibition based on biomass was calculated to be 40 mg/l. Based on the specific growth rate (m), the 0 - 72 hr EC<sub>50</sub> was calculated to be 72 mg/ml. The highest NOEC was 13.9 mg/l.

The results agree fairly well with the calculated (QSAR) EC<sub>50</sub> value for growth of algae after 48 hours exposure of 122.5 mg/l.

This is a rather important finding, since it demonstrates that the aquatic algae do not show the high susceptibility to ethylene as terrestrial plants. This coincides with ethylene having no function as a plant hormone in aquatic algae, which are single cell organisms.

### **3.2.2 Terrestrial effects**

Available data show that higher plants are highly susceptible to ethylene. Ethylene is a natural plant hormone produced by plants at all stages in growth in varying amounts. The susceptibility of a plant is also dependant on stage of growth. Ethylene plays an important role in flowering, fruit ripening,

senescence and abscission. Exposure of high concentrations of ethylene can have deleterious effects on plants if it occurs at the wrong time for plants. Adverse effects that may occur are inhibition of photosynthesis and growth, epinastic curling and shedding of flowers and leaves. Among the more sensitive agricultural or horticultural crops are peas, potatoes and oats where retardation effects were observed at concentrations in the range 8-50 mg/m<sup>3</sup>. The most susceptible non-woody plant reported are African marigold which reacts epinasty at 1.2 g/m<sup>3</sup> (0.001 ppm) and Cattleya orchid with septal tissue collapse at 2.3 mg/m<sup>3</sup> (0.002 ppm) ethylene for 24 hours. The table below summarizes some of the observed effects described in literature. The observations are grouped according to the seriousness of effects.

Summary table of effects of ethylene exposure to vascular plants. Exotic and tropical plants are not included. Epinasty=leaf curling, Abcission=loss

Effects	exposure time	Concentration µg m <sup>-3</sup>
1) None or small long term effects:		
Epimasty, Lemon		25-50
Epimasty, tomato	3-4 h	46
Epimasty, <i>Chenopodium</i>		60
Epimasty, Potato	16 h	60
2) Effects that may cause long term effects		
Inhib growth, sweet pea, (NOEC)	2 d	12
Abcission flower, Carnation	2d	58
Inhibition of photosynth. Pea (NOEL)	2 h	115
Abcission flower, Snapdragon	1h	575
3) Long term effects:		
Decreased amount flowers, Oats	100d	8
Growth inhibition, Potato	28 d	27
Yield reduction, Tomato	28 d	50
Growth retardation, Pea		116
Yield reduction, Garden cress (30 %)	14 d	115
Yield reduction, Cotton	30 d	700

### 3.2.3 Other effects

*E. coli B* bacteria were treated with ethylene by passing the gas through a bacterial suspension at constant rate for 10 minutes. After exposure, the suspensions were plated on agar medium and incubated for 24 hours at 37°C. Survival of colonies from gas treated cells was 79 ±1.3 % of controls. The survival of the *E. coli* Sd-4 strain after the same treatment was 84.2 ±1.6 % compared to controls. It was concluded that treatment seemed to have little if any effect on the survival of both bacteria strains.

## 3.3 Initial Assessment for the Environment

### 3.3.1 Aquatic compartment

The solubility of ethylene in water is comparatively low (200 mg/l at 15 °C), and the observed levels even lower. A baseline level of 6.0 mg/l has been measured, and even in heavily exposed areas the observed levels are only about 38-44 mg/l. Compared to the no effect concentrations for fish and algae of about 13 mg/l this gives a PEC/PNEC of 3.4·10<sup>-3</sup> and no reason for concern.

### 3.3.2 Terrestrial compartment



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When evaluating the terrestrial compartment, ethylene is present in air, but not in other compartments. The predicted environmental concentrations have been estimated to be  $PEC_{\text{regional}} = 5 \text{ mg/m}^3$  and  $PEC_{\text{local}} = 50 \text{ mg/m}^3$  (burning/urban),  $160 \text{ g/m}^3$  (industrial) and  $1000 \text{ g/m}^3$  (heavy traffic).

$PEC_{\text{regional}}$  is on the same level as those effecting susceptible plants ( $2.3 \text{ g/m}^3$ ), however the observed effects are not of long term significance.  $PEC_{\text{local}}$  (burning/urban) of  $50 \text{ g/m}^3$  is above the level known to affect agricultural plants ( $8\text{-}50 \text{ g/m}^3$ ) after long exposure periods. This is a problem localised to urban areas and locations with much traffic or burning of fossil hydrocarbons. In these areas, ethylene from fuel combustion is known to interact with other air pollutants like  $\text{SO}_2$ ,  $\text{NO}_x$ , ozone etc. The importance of agriculture in such areas are low.  $PEC/PNEC$  for local industrial emissions are also high, with most ratios being greater than 1 when comparing with effect concentrations in the summary table in section 3.2.2. However it applies only for within 100 m of site and as for urban areas the agricultural importance in these areas are low. With ethylene exposure concentrations of 1000 ppm as is the case for areas with heavy traffic, even quite short exposure periods may give long term effects. The awareness of the intoxication of plants in urban areas with ethylene is not new, but the present assessment further focuses on this problem.

## 4. HUMAN HEALTH

### 4.1 Human Exposure

Ethylene was in general use as an anaesthetic for many years. It has been replaced by more modern anaesthetics, mostly due to the high explosion risk. Today, elevated exposure of humans is limited to a low number of workers at ethylene production plants, and those involved in transport of ethylene.

#### 4.1.1 Occupational exposure

Personal and stationary monitoring of ethylene in a company where ethylene was used for controlling the ripening of bananas showed air concentrations to be in the range of 0.02-3.35 ppm (0.02 -  $3.85 \text{ mg/m}^3$ ). In a study on exposure of firefighters, samples taken during the "knockdown" phase of a fire showed a concentration of 46 ppm ( $53 \text{ mg/m}^3$ ) ethylene.

A study was carried out among workers at a Swedish petrochemical plant in order to assess the amounts and effects of ethylene exposure. The study was carried out in two parts, part one in 1989 and part two in 1993. Eight workers exposed to high levels of ethylene ( $4 \text{ mg/m}^3$ ) and 3 workers exposed to lower levels ( $0.1\text{-}0.3 \text{ mg/m}^3$ ) were compared to nine controls exposed to  $0.01 \text{ mg/m}^3$ . Part two of the study, which included four workers, was designed to more accurately determine exposure level which had a mean of  $4.5 \text{ mg/m}^3$ .

#### 4.1.2 Consumer exposure

No consumer products contain or release ethylene, thus consumer exposure is not relevant.

#### 4.1.3 Indirect exposure via the environment

Humans will be exposed to ethylene in the air which has been estimated to maximum  $PEC_{\text{regional}} = 2 \text{ mg/m}^3$  and  $PEC_{\text{local}} = 50 \text{ mg/m}^3$ . In-door air is anticipated also to contain increased levels of ethylene.

### 4.2 Effects on Human Health

#### a) Mode of action of the chemical, toxicokinetics and metabolism

No clear evidence of toxic effects of exposure of humans or animals to ethylene has been reported. However, it has been shown in studies, both in animals and in humans that inhaled ethylene can be metabolised to ethylene oxide. This metabolism is of concern since ethylene oxide is a potent alkylating agent, a carcinogen and a genotoxicant, and hence more toxic than ethylene. About 5 - 10 % of ethylene inhaled by rats has been reported to be converted to ethylene oxide, depending upon the concentration of ethylene in the inhaled air.

Part of the ethylene oxide formed from ethylene has been shown to react with nucleophilic sites in DNA as well as in haemoglobin. The extent of adduct formation with haemoglobin has been used to monitor the ethylene exposure in animals and in humans after occupational exposure. The oxidation of ethylene to ethylene oxide and subsequent alkylation of DNA and proteins identifies a possible mechanism of potential toxic effects of ethylene in humans.

Epidemiological as well as experimental data concludes that ethylene oxide is a carcinogen, and this is also the conclusion of the IARC working group. Thus ethylene oxide is classified as a carcinogen.

It has been demonstrated that ethylene is acute hepatotoxic to rats pre-treated with polychlorinated biphenyl (PCB) probably due to the induction of hepatic mixed function oxidases which catalyse the oxidation of ethylene to ethylene oxide. This indicate that combined exposure to inducers of mono-oxygenases and ethylene may cause a health hazard in humans.

#### **b) Acute toxicity**

The acute toxicity of inhaled ethylene is low, but very high concentrations may cause asphyxia due to oxygen displacement. The lethal concentration for mice in air is estimated to be 950,000 ppm (1093 g/m<sup>3</sup>). When male rats were exposed to 10, 25 or 57 · 10<sup>3</sup> ppm (11.5, 28.8 or 65.6 g/m<sup>3</sup>) for 4 hours, all groups showed increased serum pyruvate and liver weight.

#### **c) Repeated dose toxicity**

The toxicity of ethylene has been tested in a 90 days inhalation study on 4 exposed and one control groups of 30 rats (15 males, 15 females). The animals were exposed 6 hours/day 5 days/week for 13 weeks. The different groups were exposed to 0; 300; 1,000; 3,000 or 10,000 ppm (0, 345, 1150, 3450 or 11,500 mg/m<sup>3</sup>). There were no differences between controls and treated rats with respect to total weights, weight change, food consumption, haematology, clinical chemistry, gross pathology or histopathology. Male rats in the control, 300 ppm and 10,000 ppm groups showed red deposits or red discharge around the nose, whereas the males in the 1000 ppm group had red deposits around the eyes. Amongst the female rats, a red deposit was observed around left eye of one 300 ppm rat and alopecia around both ears of one 1000 ppm rat. Compared with the controls, the liver weights in several groups of exposed rats were significantly lower. There was however, no dose response relationship for this weight reduction and the cause was unknown. Ethylene appeared to have a low toxicity in rats when administered up to 10,000 ppm (11,500 mg/m<sup>3</sup>). This is considered a no effect level (NOEL) for the 90 days study.

In a small explorative study, where a group of six male Sprague-Dawley albino rats were exposed to a continuous flow of 60% (600,000 ppm) ethylene in oxygen as inhalation for 6 days, effects could be seen on several haematology parameters. There was a significant reduction in thrombocyte count (-19.3%) and leukocyte count (-48.2%). A reduction was also seen in the bone marrow cellularity (-30%). With this very high concentration, clear signs of toxicity were seen. However, the relevance of these studies to human health is unclear since very high doses were used.

#### **d) Reproduction developmental toxicity**

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The potential effects of ethylene inhalation on rat reproduction and on growth and development of the offspring has been studied in a combined reproduction/development toxicity screening test, conducted according to GLP. Four groups of rats (10 females and 10 males per group) were dosed by head only inhalation for 6 hours daily with: air only (control); 200, 1000 or 5000 ppm of ethylene (corresponding to 0, 230, 1150 or 5750 mg/m<sup>3</sup>). This dosing regime was calculated to give about 80, 400 and 2000 mg/kg/day of ethylene for the three dosed groups respectively. The test substance was administered to parent animals for two weeks prior to mating, during the mating period and until the day prior to necropsy of the males (minimum 28 days) or until day 20 of gestation for the females. The females were allowed to litter and rear their offspring to day 4 *post-partum*, when they and their offspring were killed.

Morbidity, mortality, clinical condition, weight and food intake were observed throughout the study, and mating was carefully observed. For each female, litter data and also observations for each offspring were recorded. At termination of the study, all animals were subject to macroscopic examination for structural or pathological changes. Ovaries, testes and epididymides of the control and high dose animals were subject to a histopathological examination.

There were no deaths attributable to the test article, and body weight gain was not adversely affected during the pre-pairing, gestation or lactation periods. The treatment had no effect on fertility or fecundity and all females became pregnant. Litter size, sex ratio, mean pup weight and pup growth and clinical condition were not adversely affected by treatment.

Necropsy revealed no macroscopic finding suggestive of toxicity due to test substance administration. There was no evidence of any toxic effect on the testis due to test substance administration and there were no other microscopic findings suggestive of toxicity due to the exposure.

In conclusion, head-only administration of ethylene at nominal concentrations of 200; 1,000 or 5,000 ppm was without evidence of toxicity or adverse effects on male and female reproductive performance, fertility, pregnancy, maternal and suckling behaviour and growth and development of the offspring from conception to Day 4 post-partum. The highest dose was concluded to be a no effect level (NOEL) for the reproduction/development screening test in rats.

#### **e) Genetic toxicity**

##### **Bacterial test *in vitro***

Ethylene at atmospheric concentrations up to 20 % gave no indication of mutagenic potential when tested in one strain of *Salmonella typhimurium* in the presence or absence of a liver metabolic activation system (S9) (Ames test). Previous testing with four *Salmonella* strains in the presence and absence of S9 have also given negative results. Ethylene also showed no genotoxic activity in *Escherichia coli*.

##### **Non-bacterial *in vitro* test**

The effect of ethylene on chromosome structures was tested in an *in vitro* cytogenetics assay using duplicate cultures of CHO cells. The test was conducted according to GLP. Treatments covering a broad range of doses, separated by narrow intervals, were performed both in the absence and presence of metabolic activation system (rat liver post-mitochondrial fraction) from Aroclor 1254 induced animals. The highest dose level used, approximately 280.5 mg/l, was equivalent to a concentration of 10 mM.

A preliminary range-finding study was performed to investigate the toxic effects of ethylene on CHO cells. In this study, treatment in the absence and presence of S9 was for 3 hours only followed by a 17 hour recovery period prior to harvest (3+17). The dose levels for the main study were selected by evaluating the effect of ethylene on mitotic index. The treatment regimes used in the range-finding study were used in the main study. Chromosome aberrations were analysed at three consecutive dose levels. No mitotic inhibition (reduction in mitotic index) was observed at the highest concentration chosen for

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analysis (280.5 mg/l) in either the absence or presence of S-9. Treatment of cultures with ethylene in the absence and presence of S-9 resulted in frequencies of cells with structural aberrations that were similar to, and not significantly different from, those seen in concurrent negative control cultures. Frequencies seen in treated cultures fell within the normal range.

It is concluded that ethylene did not induce chromosome aberrations in cultured Chinese hamster ovary cells exposed to a concentration of 280.5 mg/l in the absence and presence of S9.

#### **Genetic toxicity *in vivo***

Ethylene did not induce micronuclei formation in bone marrow cells of rats or mice exposed to up to 3000 ppm (3500 mg/m<sup>3</sup>) for 6 h/day, five days/week for four weeks.

#### **f) Carcinogenicity**

The potential carcinogenicity of ethylene has been tested in a two years study with rats (Fischer 344 inbred). In the study, 960 rats were randomly divided into 4 groups of 120 animals of each sex and exposed 6 hr/day, 5 days/week to 0 (control), 300, 1000 and 3000 ppm (0, 345, 1150 or 3450 mg/m<sup>3</sup>) for up to 24 months.

During the course of the study there were observations of hair loss, deposits on and around the nose and eyes and gross eye abnormalities, but there were no obvious differences among the treatment groups.

There was an overall increase in the number of animals exhibiting gross tissue masses for the test groups as compared with the control group, although this trend was not statistically significant. The spontaneous mortality (15.7 %) was roughly equal in all treated groups. The final body weights and total weight changes for treated males were higher than those in the control groups, but no dose-related pattern was seen.

There were no statistically significant difference among any of the treatment groups on any of the haematology, blood chemistry or other parameters investigated. No gross or histopathologic tissue changes attributable to the effects of the test material were observed in any of the treated rats. The summary reports only few findings which could indicate any carcinogenic effect of the treatment. However, based on the rate of formation of the carcinogen ethylene oxide and its possible role in ethylene toxicity, the study did not have statistical power to detect an increased frequency of tumour formation.

The latest evaluation by the IARC working group (1994) concludes that there is inadequate evidence in humans and in experimental animals for the carcinogenicity of ethylene. Overall, ethylene was evaluated as not classifiable as to its carcinogenicity to humans.

#### **g) Human data**

The inhalation pharmacokinetics of ethylene was investigated in human volunteers at atmospheric concentrations of up to 50 ppm by gas uptake in a closed spirometer system, and the uptake, exhalation and metabolism could be described by first-order kinetics [64]. The clearance due to uptake was low, only 5.6 %, while the rest was only exhaled without entering the blood stream. Clearance due to metabolism was 36 % of systematically available ethylene. The biological half-life of ethylene was 0.65 hours. The alveolar retention of ethylene at steady state was calculated to be 2 %. The low uptake rate of ethylene was considered due to its low solubility in blood.

There have been two preliminary but independent reports of increased miscarriage rates among women working in the petrochemical industry. Elevated ethylene concentrations were mentioned as a possible reason, but this has not been confirmed. No firm conclusions can be drawn from these reports.

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A preliminary study found no increase in lung cancer incidence in 31 workers exposed to ethylene (at unspecified levels) at a US petrochemical factory. However, due to the limited number of exposed workers in this study no conclusions regarding ethylene not causing cancer can be drawn.

A study of workers at an US petrochemical plant found that an increased risk of developing brain cancer was associated with exposure to (unspecified levels of) a number of chemicals including ethylene. However, the investigators were unconvinced that the association reflected a causal relationship.

### 4.3 Initial Assessment for Human Health

#### Assessment from acute and repeated dose toxicity studies

It is assumed that the maximum occupational exposure is  $5 \text{ mg/m}^3$  and the maximum exposure to the general population via the environment is  $50 \text{ mg/m}^3$ . The NOEL of  $11,500 \text{ mg/m}^3$  in the 90 days inhalation study in rats gives safety margins of 2300 and  $2.3 \cdot 10^5$ , which indicates no hazard concern.

#### Assessment from the reproduction/development toxicity study in rats

No signs of reproductive or development toxicity of ethylene was observed in the inhalation test with rats. The highest concentration used ( $5750 \text{ mg/m}^3$ ) is taken as the NOEL and this gives a safety margin of 1150 for occupational exposure and  $1.15 \cdot 10^5$  for general exposure indicates minimal hazard concern.

#### Assessment from genetic toxicity and carcinogenicity studies

Ethylene has been shown to be without genetic toxicity in bacterial test *in vitro*, in mammalian cells *in vitro* and in the mouse micronucleus test *in vivo*. Also a full two years carcinogenicity study has been conducted in rats. As all the tests are negative, there is no reason to indicate that ethylene should imply a high risk of genetic toxicity or carcinogenicity to humans. However, the fact that ethylene is metabolised, both in humans and experimental animals, to the carcinogenic genotoxicant ethylene oxide is of some concern.

#### Assessment from mode of action and mechanism of toxicity

In the case of ethylene, a possible mechanism for a toxic potential in humans has been identified, but few signs of toxicity have been observed. This is related to the fact that ethylene gives rise only to minute doses of ethylene oxide. The maximum conversion of ethylene to ethylene oxide in humans is estimated to 4 %, while about 1 % has been measured.

## 5. CONCLUSIONS AND RECOMMENDATIONS

### 5.1 Conclusions

Ethylene is, due to its physical and chemical properties released mainly into the atmospheric compartment. About three quarters of atmospheric ethylene originates from natural sources, while one quarter is from anthropogenic sources. The main anthropogenic release is from burning of hydrocarbons and biomass.

It has been well documented through relevant toxicity studies that the minute amounts measured in water implies no environmental hazard to the organisms in this compartment.

In the terrestrial compartment, the vegetation has proven highly susceptible to ethylene, probably through a mechanism related to the hormone function of ethylene in plants. Ethylene levels in urban areas have reached levels which inhibit growth of certain plant species. This is of concern both for decorative plants and agriculture in exposed areas. This ethylene is not due to emission of ethylene from ethylene production, but rather to incomplete burning of coal and petrol products.

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Relevant studies on ethylene have indicated a low toxicity and no risk to human health has been identified neither from occupational exposure nor exposure of general public, either exposed directly or indirectly via the environment. Metabolic studies in animals and man have revealed that ethylene is metabolized to ethylene oxide which is known to have carcinogenic and mutagenic effects. However, IARC has in its evaluation in 1994 concluded that there is inadequate evidence both in humans and in experimental animals for the carcinogenicity of ethylene.

## **5.2 Recommendations**

No further testing of ethylene toxicity is recommended.

It is recommended to do a closer monitoring of the environmental ethylene levels in urban and polluted regions with respect to its potential intoxication of vegetation, but this goes beyond the OECD HVP Chemical Programme.

## **6. REFERENCES**

References are not given in the SIDS initial assessment report. It is referred to those given in the Full SIDS Dossier.

***SIDS DOSSIER ON ETHYLENE***

**Summary of Responses to the OECD Request for  
Available Data on HPV Chemicals**

**SIDS PROFILE**

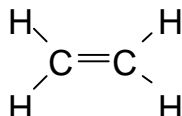
1.01 A	CAS NO.	74-85-1
1.01 C	CHEMICAL NAME	Ethylene
1.01 G	STRUCTURAL FORMULA	$  \begin{array}{c}  \text{H} \quad \quad \text{H} \\  \diagdown \quad \diagup \\  \text{C} = \text{C} \\  \diagup \quad \diagdown \\  \text{H} \quad \quad \text{H}  \end{array}  $
	<b>OTHER CHEMICAL IDENTITY INFORMATION</b>	
1.5	QUANTITY	Millions metric tonnes per year: (capacity for 1996) Norway: 0.4 World: 83.0
1.7	USE PATTERN	Chemical industry; as raw material for synthesis of chemicals, petrochemicals and resins. Minor quantities used for fruit ripening and as anaesthetic gas.
1.9	SOURCES OF EXPOSURE	Fuel, coal and gas combustion. Leakage from chemical industry. Rural areas: < 1 - 5 µg/m <sup>3</sup> (0.9 - 4.3 ppb) Heavy traffic areas: up to 1.0 mg/m <sup>3</sup> (0.9 ppm) Petrochemical plants: up to 5 mg/m <sup>3</sup> (4.3 ppm)
ISSUES FOR DISCUSSION (IDENTITY, IF ANY)	No further testing required	



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**1. GENERAL INFORMATION**

- A. **CAS number:** 74-85-1
- B. **Name (IUPAC):** Ethylene
- C. **Name (OECD):** Ethylene
- F. **Molecular formula:** CH<sub>2</sub>CH<sub>2</sub>
- G. **Structural formula:**



- H. **Substance group:** Industrial chemical; as raw material for synthesis of chemicals, petrochemicals and resins.
- J. **Molecular Weight:** 28.05

**1.02 OECD INFORMATION**

- A. **Sponsor Country:** Norway
- B. **Lead Organisation:**  
Norwegian Pollution Control Authority (SFT),  
P.O. Box 8100 Dep.,  
N-0032 Oslo  
NORWAY

**Contact person:**  
Marit Kopangen

Tel.: +47 22 573400  
Fax.: +47 22 676706

- C. **Name of responder:**  
Noretyl ANS,  
Petrochemical division,  
Norsk Hydro ANS,  
N-0240 Oslo  
NORWAY

**1.1 GENERAL SUBSTANCE INFORMATION**

- A. **Type of Substance:** Organic, hydrocarbon
- B. **Physical state** ( at 20 °C and 1.013 hPa): Gaseous
- C. **Purity:**

- 1) High purity : > 99.9 %
- 2) Commercial purity : about 99.9 %

## 1.2 SYNONYMS

Ethene, acetene, bicarburetted hydrogen, olefiant gas, elayl.

## 1.3 IMPURITIES

Western Europe product, (ppm range):

Methane + ethane (50-200), propylene and heavier (7-200), CO<sub>2</sub> (2.2-50), H<sub>2</sub>(0.1-10), O<sub>2</sub> (0.6-10), acetylene (1.4-10), total sulphur (1-10), water (0.6-20) and CO (0.15-10) [3].

## 1.4 ADDITIVES

None known.

## 1.5 QUANTITY

More than 1,000,000 tonnes per annum.

Capacity for 1996 [2]:

Norway: 405,000 tonnes

World: 83,000,000 tonnes

## 1.6 LABELLING AND CLASSIFICATION

**EEC:** Fx, R12 (Extremely flammable).

S 2 (Keep out of reach of children.

S 9 (Keep container in well-ventilated place)

S 16 (Keep away from sources of ignition - No smoking)

S 33 (Take precautionary measures against static discharges)

**Norway:** F, R13 (Extremely flammable liquid gas)

S 9-16-33

According to IARC Monograph Volume 60, (1994):

Ethylene: The agent is not classifiable as to its carcinogenicity to humans [3].

## 1.7 USE PATTERN

Ethylene is the petrochemical product produced in largest quantities world-wide. More than 95% of the annual commercial production of ethylene is currently based on steam cracking of petroleum hydrocarbons [4].

About 80 % of the ethylene consumed in US, Western Europe and Japan is used for production of ethylene oxide, ethylene dichloride and low density, linear low density and high density polyethylene. Significant amounts are also used to make ethylbenzene, alcohols, olefins, acetaldehyde and vinylacetate. Most of these products are further processed into products such as film, blow and injection moulding, extrusion coating, cable insulation and PVC. Minor quantities have been used as anaesthetic gas, for fruit ripening and for welding and cutting metals.

## A. General

	<b>Type of use:</b>	<b>Category:</b>
a)	Main industrial use	Use in closed systems Chemical Industry: used in synthesis Raw material
b)	Main industrial use	Non dispersive use Agricultural Industry As fruit ripener

**B. Uses in Consumer Products**

Not known

**1.8 OCCUPATIONAL EXPOSURE LIMIT VALUE**

No exposure limits have been recommended in most countries, but Switzerland established a time-weighted average occupational exposure limit of 11 500 mg/m<sup>3</sup> [3].

**1.9 SOURCES OF EXPOSURE**

Ethylene is ubiquitous in the environment, arising from both natural and man made sources. Major sources are as a natural product from vegetation of all types [5].

The main anthropogenic sources are from combustion of gas, fuel, coal and biomass. Maximal exposure of ethylene to humans is considered to be through fossil combustion by vehicles. The total ethylene emission from the global surface has been estimated to be 18-45 · 10<sup>6</sup> t/y, of which approximately 74% is released from natural sources and 26 % from anthropogenic sources. Emission from oil combustion is estimated to 1.54 · 10<sup>6</sup> t/y [5]. Ethylene produced and consumed in chemical industry is kept in closed systems and the production facility is normally next door to the factory using ethylene as a raw material. Exposure to ethylene from industrial sources are thus mainly due to uncontrolled leakage or blow outs. Such events occur at a rate of once every 2.0 · 10<sup>6</sup> t/y of produced ethylene and may result in an immediate release of about 1 ton.

**1.10 ADDITIONAL REMARKS****A. Option for disposal**

Incineration.

**B. Other remarks**

No data.

**2. PHYSICAL-CHEMICAL DATA****2.1 MELTING POINT**

-169.15 °C [4]

**2.2 BOILING POINT**

-103.71 °C [4]

**2.3 DENSITY**

$d = 0.57 \text{ g/cm}^3$  at boiling point [4].  
Gas density at STP 1.2603 g/l [4].  
Density relative to air 0.9686 [4].

## 2.4 VAPOUR PRESSURE

4.27 MPa at 0 °C [4].

## 2.5 PARTITION COEFFICIENT $\log_{10}P_{ow}$

$\log_{10}P_{ow} = 1.13$  (calculated) [6].

## 2.6 WATER SOLUBILITY

### A. Solubility

According to Merck Index, "One volume of ethylene gas dissolves in 4 vol of water at 0°C" [7].  
One volume of ethylene gas dissolves in 9 volumes of water at 25 °C [8].  
Solubility: 131 mg/l at 20°C [9].  
At 15 °C the solubility in water is 200 mg/l [10].

### B. pH Value, pKa Value

No data available. There is no chemical evidence to suggest a reaction between dissolved ethylene and water and pH remains unchanged.

## 2.7 FLASH POINT

- 136.11 °C [11].

## 2.8 AUTO FLAMMABILITY

Autoignition temp: 543°C [7].  
Ignition temp: 425-527°C [4].

## 2.9 FLAMMABILITY

Extremely flammable - liquefied gas.

## 2.10 EXPLOSIVE PROPERTIES

**Explosive limits in air** (0.1 MPa and 20°C) [4] :  
Lower explosive limit (LEL): 2.75 vol %  
Upper explosive limit (UEL): 28.6 vol %

## 2.11 OXIDIZING PROPERTIES

No information

## 2.12 OXIDATION:REDUCTION POTENTIAL

No information.

## 2.13 ADDITIONAL DATA

### A. Partition co-efficient between soil/sediment and water (Kd).

No information

### B. Other data

#### Conversion factor for ethylene in air:

1 ppm in air =  $1.15 \text{ mg/m}^3 = 912 \text{ nl/l}$  [1,4]

#### Odour threshold:

Odour low:  $299 \text{ mg/m}^3$

Odour high:  $4600 \text{ mg/m}^3$  [12]

## 3. ENVIRONMENTAL FATE AND PATHWAYS

### 3.1 STABILITY

#### 3.1.1 STABILITY IN AIR

The fate of atmospheric ethylene emitted from natural and anthropogenic sources has been estimated by Sawada and Totsuka, 1986 [5]. They concluded that 89 % was destroyed in the troposphere by reaction with OH radical, and 8 % in the reaction with O<sub>3</sub>. The remaining 3 % was transported into the stratosphere. The atmospheric lifetime of ethylene was estimated to be between 2 and 4 days.

Indirect calculation of photodegradation with O<sub>3</sub> as a sensitizer gave a lifetime of 9.4 days [13]. Using OH as the sensitizer a lifetime of 2.7 days was calculated [14].

The following lifetimes are according to Howard, P.H. et al (1991) [15]: Handbook of environmental degradation rates:

		<u>Lifetimes:</u>
Air:	High:	3.36 days
	Low:	0.37 days

This is based upon combined, measured photooxidation rate constants for ·OH and O<sub>3</sub>.

If the calculation procedures for organic compounds in atmosphere of Atkinson, R. (1996) [75] are used the following depletion rates are found:

		<u>Lifetimes</u>
Air	Due to ·OH reaction	1.7 days
	Due to O <sub>3</sub> reaction	10 days
	Due to stratospheric removal	1900 days

Stratospheric removal can be calculated according to IPCC (1995) [76], assuming a similar removal of ethylene as CO.

#### 3.1.2 STABILITY IN WATER

No data available

### 3.1.3 STABILITY IN SOIL

No data available

### 3.2 MONITORING DATA (ENVIRONMENT)

Rudolph and Johnen, [16] did more than 200 in situ measurements of ethylene and other selected Light Atmospheric Hydrocarbons during, a cruise from Puerto Madryn (Argentina) to Bremerhaven (Germany) in 1987. The measuring locations were remote with low biological activity in the surrounding ocean areas. The ethylene level, expressed as mixing ratio was in the range 10-30 ppt (12-35 ng/m<sup>3</sup>) in the southern hemisphere and in the northern hemisphere a factor of 2 higher. The observed ethylene levels were primarily a result of oceanic emissions and the differences were indicated to be caused by changes in oceanic phytoplankton concentration.

The oceanic distribution of ethylene and other low molecular weight (LMW) hydrocarbons has been studied by Swinnerton and Lamontagne, 1974 [17]. They analyzed 452 water samples from the open ocean and near shore for LMW hydrocarbons and found a baseline (average) ethylene of: 4.8 nanoliters/litre (6.0 µg/l) . Upper values were: Mississippi R. Delta ; 35.0 nl/l (44 µg/l) and Miami dockside; 30.0 nl/l (38 µg/l).

Fuel, coal and gas combustion. Leakage from chemical industry. Rural areas: < 1 - 5 µg/m<sup>3</sup>, heavy traffic areas: up to 1.0 mg/m<sup>3</sup> [1, 3].

During burning of wood (white pine) an ethylene concentration of about 50 ml/m<sup>3</sup> (63 mg/m<sup>3</sup>) was measured in the smoke [18].

### 3.3 TRANSPORT AND DISTRIBUTION BETWEEN ENVIRONMENTAL COMPARTMENTS INCLUDING ESTIMATED ENVIRONMENTAL CONCENTRATIONS AND DISTRIBUTION PATHWAYS

In their study of the dynamics of atmospheric ethylene, Sawada and Totsuka, [5] estimated the following emissions of ethylene (in 10<sup>6</sup> t/y):

<u>Natural:</u>		
Terrestrial	23.3	(65.8 %)
Aquatic	<u>2.9</u>	( 8.2 %)
	<u>Sum</u> 26.2	(74.0 %)
<u>Anthropogenic:</u>		
Fuel oil combustion	1.5	(4.28 %)
Coal combustion	0.42	(1.20 %)
Leakage from Industri	0.03	(0.09 %)
Sjøpel forbrenning	0.10	(0.29 %)
Biomass burning	<u>7.10</u>	(20.1 %)
	<u>Sum</u> 9.19	(26.0 %)

**Total Natural + Anthropogenic = 35.4 · 10<sup>6</sup> t/y**

#### Atmospheric depletion of ethylene:

Ethylene reacts with OH radical to form an adduct which in the presence of O<sub>2</sub> and NO<sub>x</sub> forms formaldehyde. The products of reaction of ethylene with O<sub>3</sub> are mostly CO, CO<sub>2</sub>, H<sub>2</sub>O and CH<sub>2</sub>O. Some ethylene is also transported into the stratosphere [76]. Using the most recent

estimates [75] of the depletion rates (lifetime) of ethylene in the atmosphere due to these processes give:

:

	lifetime (days)	
Reaction with ·OH radical	1.7	
Reaction with O <sub>3</sub>	10	
into the stratosphere	1900	
total lifetime in atmosphere	1.45	
<u>Ethylene sinks (removal capacity, 10<sup>6</sup> tons/y):</u>		
Reaction with ·OH radical	44.4	(85.4%)
Reaction with O <sub>3</sub>	7.5	( 14.5 %)
Into stratosphere	<u>0.036</u>	( 0.07 %)
<u>Sum</u>	<u>52.0</u>	

The ethylene transported into the stratosphere will eventually react with O<sub>3</sub> with the production of a krüer molecule, which again may react with NO regenerating O<sub>3</sub>. ethylene is therefore not suspected of being a potential ozone depletor.

### 3.3.1 TRANSPORT

Physical properties of ethylene indicate that it will rapidly move into the atmosphere from any type of release.

### 3.3.2 THEORETICAL DISTRIBUTION (FUGACITY CALCULATION)

A fugacity level I calculation, using a six compartment model (air, water, soil solids, sedimented solids, suspended sediments and fish) was conducted using the global reference model of OECD [19]. Default values for the environmental parameters were not changed. Entered generic parameters were: melting point - 169.15 °C, vapour pressure 4.27 MPa, water solubility 200 g/m<sup>3</sup>, log<sub>10</sub>P<sub>ow</sub> 1.13, half-life in air 56 hours, half-life in water, soil and sediment 672 hours. This gave the following distribution:

in air	99.99915 %,
in water	8.27·10 <sup>-4</sup> %,
in soil solids	9.88·10 <sup>-6</sup> %
in sedimented solids	2.20· 10 <sup>-7</sup> %.
in suspended sediments	6.87· 10 <sup>-9</sup> %
in fish	5.58·10 <sup>-10</sup> %

This means that for all practical purposes, emitted ethylene is distributed to air only.

### 3.4 IDENTIFICATION OF MAIN MODE OF DEGRADABILITY IN ACTUAL USE

See 3.3

### 3.5 BIODEGRADATION

Also a number of research orientated studies were designed to examine the oxidation/hydroxylation and epoxidation of various hydrocarbons by microorganisms isolated from soil, fresh water systems or other natural systems and pure cultures. Generally, results of these studies show that ethylene is subject to biodegradation by various microorganisms and that ethylene oxide and ethylene glycol are most likely initial degradation products [21].

Aqueous biodegradation rates have been estimated both for aerobic and anaerobic conditions [15]:

Aerobic half-life:	High: 672 hours
	Low: 24 hours
Anaerobic half life:	High: 2688 hours
	Low: 96 hours

### 3.6 BOD<sub>5</sub>, COD OR RATIO BOD<sub>5</sub>/COD

No data available

### 3.7 BIOACCUMULATION

Ethylene is not expected to bioaccumulate because of  $\text{Log}_{10} P_{\text{ow}} = 1.13$ . BCF (Bioconcentration factor) is calculated (QSAR) to be 4 on the basis of the toxic action of nonpolar molecules in the freshwater fish Fathead minnow (*pimephales promelas*), exposure duration 2.00 - 304 days [22].

### 3.8 ADDITIONAL REMARKS

No data.

## 4. ECOTOXICOLOGICAL DATA

### 4.1 ACUTE TOXICITY TO FISH

Little is known about the acute toxicity of ethylene to fish, but the "Water Quality Criteria, California State Water Resources Control Board, 1963" [23] refers to two reports of toxicity of ethylene to Orange-spotted sunfish from 1917 [24] and 1921 [25]. The findings were the following:

Lethal conc after 1 hour :	22 - 25 mg/l [24]
Lethal conc after $\geq 1$ hour :	22 - 65 mg/l [25]

Calculated (QSAR) values reported in the database Ecotoxicity Profile database [26]:

Fathead minnow ( <i>Pimephales promelas</i> )	4 days LC <sub>50</sub> 116 mg/l
Bluegill, ( <i>Lepomis macrochirus</i> )	4 days LC <sub>50</sub> 85 mg/l
Channel catfish, ( <i>Ictalurus punctatus</i> )	4 days LC <sub>50</sub> 50 mg/l
Rainbow trout, Donaldson trout, ( <i>Onchorhynchus mykiss</i> )	4 days LC <sub>50</sub> 55 mg/l

Calculated (QSAR) values reported by Leeuwen et. al. [27]:  
Fathead minnow (*Pimephales promelas*) 4 days LC<sub>50</sub> 120 mg/l

### 4.2 ACUTE TOXICITY TO AQUATIC INVERTEBRATES

#### A. *Daphnia*

Calculated (QSAR) value reported in the database Ecotoxicity Profile [26]:

Water flea, ( <i>Daphnia magna</i> )	48 hours	LC <sub>50</sub> 53 mg/l
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Calculated (QSAR) value according to Leeuwen et. al. [27]:  
 Daphnid 48 hours LC<sub>50</sub> 153 mg/l

#### B. Other aquatic organisms

No data available.

### 4.3 TOXICITY TO ALGAE

A growth inhibition test with *Selenastrum capricornutum* was performed according to OECD 201 and conducted according to GLP guidelines in 1996 [74]. The 5 nominal test concentrations in the growth medium ranged from 8.2 to 131 mg/l. During the 72 hr exposure period there was a loss of ethylene, however the mean measured ethylene concentrations (mean of zero time and 72 h measurement) were used for calculation of growth inhibition. Actual test concentrations (mean) were therefore: 3.3, 7.8, 13.9, 32 and 58mg/l. Loss of ethylene during the 72 hr incubation period ranged from 64 to 91 %. EC<sub>50</sub> for the growth inhibition based on reduction in biomass compared to control, was calculated to be 40 mg/l (95 % conf. lim.36-46 mg/l). Based on the specific growth rate ( $\mu$ ) the 0 - 72 hr EC<sub>50</sub> was calculated to be 72 mg/l (95 % conf. lim. could not be calculated due to that the EC<sub>50</sub> value was outside the range of the test). The highest NOEC was 13.9 mg/l. The results agree fairly well with QSAR calculation for *Selenastrum capricornutum* which gave an EC<sub>50</sub> after 48 hour value of 122.5 mg/l [27].

### 4.4 TOXICITY TO BACTERIA

*E.coli* bacteria were treated with ethylene by passing the gas through a bacterial suspension at constant rate for 10 minutes. After 24 hours exposure, the suspensions were plated on agar medium and incubated for 24 hours at 37 °C. Survival of colonies from gas treated cells was 79 ± 1.3 % of controls. The survival of the *E. coli* Sd-4 strain after the same treatment was 84.2 ± 1.6 % compared to controls. It was concluded that treatment seemed to have little if any effect on the survival of both bacteria strains [28].

### 4.5 CHRONIC TOXICITY TO AQUATIC ORGANISMS

#### 4.5.1 CHRONIC TOXICITY TO FISH

Calculated (QSAR) value reported in the database Ecotoxicity Profile [26]:  
 Fathead minnow, (*Pimephales promelas*) 32 days MATC 15.3 mg/l

Calculated (QSAR) value according to Leeuwen et. al. [27]:  
 Fathead minnow, (*Pimephales promelas*) 28 days NOEC 13 mg/l

#### 4.5.2 CHRONIC TOXICITY TO AQUATIC INVERTEBRATES

Calculated (QSAR) value according to Leeuwen et. al. [27]:  
 Daphnia 16 days NOEC 37.4 mg/l

### 4.6 TOXICITY TO TERRESTRIAL ORGANISMS

#### 4.6.1 TOXICITY TO SOIL DWELLING ORGANISMS

No data available

#### 4.6.2 TOXICITY TO TERRESTRIAL PLANTS

A large and diverse literature exists on the effects of ethylene on vascular plants, including several hundred observations of ethylene exposure and effects. This is mainly due to the fact that ethylene acts as a plant hormone, regulating a whole range of different reactions. Most of these reactions can be categorised as growth regulation and include such effects as defloration, ripening, inhibition of elongation, leaf loss and senescence [9,11, 29, 30, 31, 32]. While most of these effects are non reversible, they do not all constitute effects that reduce a plants fitness nor growth and reproduction. One may categorise the effects into 3 groups based on assumed long term effects, where long term effects are associated with reduced fitness, growth or reproduction. In the table below exotic and tropical plants have been excluded in order to present data that give a more realistic view of risks associated with exposure in industrial areas.

Summary table of effects of ethylene exposure to vascular plants. Exotic and tropical plants are not included. Epinasty=leaf curling, Abcission=loss

Effects	exposure time	concentration $\mu\text{g m}^{-3}$	Ref
1) None or small long term effects:			
Epinasty, Lemon		25-50	[77]
Epinasty, tomato	3-4 h	46	[9]
Epinasty, <i>Chenopodium</i>		60	[9]
Epinasty, Potato	16 h	60	[9]
2) Effects that may cause long term effects			
Inhib growth, sweet pea, (NOEC)	2 d	12	[77]
Abcission flower, Carnation	2d	58	[77]
Inhibition of photosynth. Pea (NOEL)	2 h	115	[77]
Abcission flower, Snapdragon	1h	575	[33]
3) Long term effects:			
Decreased amount flowers, Oats	100d	8	[77]
Growth inhibition, Potato	28 d	27	[77]
Yield reduction, Tomato	28 d	50	[77]
Growth retardation, Pea		116	[9]
Yield reduction, Garden cress (30 %)	14 d	115	[77]
Yield reduction, Cotton	30 d	700	[9]

Among the more sensitive agricultural or horticultural crops are peas, potatoes, tomatoes and oats where retardation effects were observed at concentrations in the range 8-50  $\mu\text{g/m}^3$  (7-40 ppb) . The most susceptible non-woody plant reported, African marigold reacts with leaf epinasty (downward curling of leaves at 1.16  $\mu\text{g/m}^3$  (1.0 ppb) ethylene [9], the Cattleya orchid, reacts with sepal tissue collapse (loss of flower) at 2.3  $\mu\text{g/m}^3$  (2.0 ppb) after ethylene exposure for 24 hours [33].

#### 4.6.3 TOXICITY TO OTHER NON MAMMALIAN TERRESTRIAL SPECIES (INCLUDING AVIAN)

No data

#### 4.7 BIOLOGICAL EFFECTS MONITORING (INCLUDING BIOMAGNIFICATION)

No data

#### 4.8 BIOTRANSFORMATION AND KINETICS IN ENVIRONMENTAL SPECIES

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No data

#### **4.9 ADDITIONAL REMARKS**

No data

### **5. TOXICITY**

#### **5.1. ACUTE TOXICITY**

##### **5.1.1 ACUTE ORAL TOXICITY:**

Not relevant. Ethylene is a gas with a low boiling point (-103.71 °C).

##### **5.1.2 ACUTE INHALATION TOXICITY**

The acute toxicity of ethylene is low, but very high concentrations may cause asphyxia due to oxygen displacement. The lethal ethylene concentration in air to mice is thus estimated to be 950,000 ppm. [34].

When male rats were exposed to 10, 25 or 57·10<sup>3</sup> ppm for 4 hours, all groups showed increased serum pyruvate and liver weight [35]. Non of the studies were GLP.

##### **5.1.3 ACUTE DERMAL TOXICITY**

Not relevant. Very little ethylene is likely to be absorbed through the skin because of ethylene's low solubility in fat and low boiling point.

##### **5.1.4 ACUTE TOXICITY, OTHER ROUTES OF ADMINISTRATION**

No information

#### **5.2 CORROSIVENESS/IRRITATION**

##### **5.2.1 SKIN IRRITATION/CORROSION**

There is no evidence to suggest that the liquid ethylene gas is irritant, but it might cause frost injuries.

##### **5.2.2 EYE IRRITATION**

There is no evidence to suggest that the liquid ethylene gas is irritant, but it might cause frost injuries.

#### **5.3 SKIN SENSITISATION**

No data.

#### **5.4 REPEATED DOSE TOXICITY**

The toxicity of ethylene has been tested in a 90 days inhalation study on 4 exposed and one control groups of 30 rats (15 males, 15 females) [36]. The animals were exposed 6 hours/day 5

days/week for 13 weeks. The exposure groups were T-I: 300 ppm, T-II: 1,000 ppm, T-III: 3,000 ppm and T-IV: 10,000 ppm. The study was not conducted according to GLP, but the study held a high scientific standard and a quality assurance statement was issued. There were no differences between controls and treated rats with respect to total weights, weight change, food consumption, haematology, clinical chemistry, gross pathology or histopathology. Male rats in the control, T-I and T-IV groups showed red deposits or red discharge around the nose, whereas the male T-II had red deposits around the eyes. Amongst the female rats, a red deposit was observed around the left eye of one T-I rat and alopecia around both ears of one T-II rat. Compared with the controls, the liver weights in several groups of exposed rats were significantly lower. There was, however, no dose response relationship for this weight reduction and the cause was unknown. Ethylene was not toxic to rats when administered under a stratified regimen of exposure up to 10,000 ppm.

In an explorative non-GLP study, where a group of six male Sprague-Dawley albino rats (50-60 g) were exposed to a continuous flow of 60% ethylene in oxygen as inhalation for 6 days, effects could be seen on several haematology parameters [37]. There were significant reductions in thrombocyte count (-19.3%) and leukocyte count (-48.2%). A reduction was also seen in the bone marrow cellularity (-30%).

During chronic tests on rats (newborn) exposed to a concentration of 2.62 ppm (continuous as inhalation) for 90 days, a delay in coat appearance, dentition, eye opening and circulation hypotension, cholinesterase activity inhibition, subordination disruption were reported [38]. There were no information on the quality of the study.

In rats treated by inhalation with a concentration of 100 ppm for 70 days, a change in the reflex nerve impulses, a decrease of cholinesterase activity and a reduction of the blood pressure were observed [39]. There were no information on the quality of the study.

## 5.5 GENETIC TOXICITY IN VITRO

### A. Bacterial test

Ethylene at atmospheric concentrations up to 20 % gave no indication of mutagenic potential in *Salmonella typhimurium* in the presence or absence of a metabolic activation system (Ames test) [40]. The study was not conducted according to GLP, and only one (TA 100) of the four bacterial test strains recommended in the guidelines was tested. Previous testing with the full range of *Salmonella* strains in the presence and absence of a metabolic activation system have also given negative results [41, 42]. Ethylene showed no genotoxic activity in *Escherichia coli*. [28].

### B. Non-bacterial in vitro test

The effect of ethylene on chromosomes was tested in an in vitro cytogenetics assay using duplicate cultures of CHO cells [71]. The methodology in this study complies with GLP and the OECD Test Guideline 473, "Genetic Toxicology: In vitro Mammalian Cytogenetic Test". Treatments covering a broad range of doses, separated by narrow intervals, were performed both in the absence and presence of metabolic activation (S9) from Aroclor 1254 induced rats. The highest dose level used, approximately 280.5 mg/ml, was equivalent to a concentration of 10 mM, corresponding to about 25 % of ethylene.

Due to the explosive properties of the test article when mixed with air, it was not possible to achieve the maximum concentration required by the Regulatory Guidelines using air as carrier gas. Nitrogen was therefore used as carrier gas, which allowed higher doses to be achieved. There are, however, technical problems associated with continuous treatment in a nitrogen atmosphere, and short (3 hour) pulse treatments were the only practical option.

A preliminary range-finding study was performed to investigate the toxic effects of ethylene on CHO cells. In this trial, treatment in the absence and presence of S9 lasted for 3 hours only followed by a 17 hours recovery period prior to harvest (3+17). The dose levels for the main study were selected by evaluating the effect of ethylene on mitotic index.

The treatment regimes used in the range-finder were repeated in the main study. Chromosomal aberrations were analyzed at three consecutive dose levels. No mitotic inhibition (reduction in mitotic index) was observed at the highest concentration chosen for analysis (280.5 µg/ml) in either the absence or presence of S9.

Appropriate negative (carrier gas) controls were included in the test system in both experiments under each treatment condition. Untreated controls were also included in the main study. The proportion of cells with structural aberrations in the negative and untreated cultures fell within historical solvent control ranges. 4-Nitroquinoline 1-oxide and cyclophosphamide were employed as positive controls in the absence and presence of liver S9 respectively. Cells receiving these were sampled in the main study, 20 hours after the start of treatment; both compounds induced statistically significant increases in the proportion of cells with structural aberrations.

Treatment of cultures with ethylene in the absence and presence of S9 resulted in frequencies of cells with structural aberrations that were similar to, and not significantly different from, those seen in concurrent negative controls. Frequencies seen in treated cultures fell within the normal range.

It is concluded that ethylene did not induce chromosome aberrations in cultured Chinese hamster ovary cells exposed to a concentration of 10 mM (25 %) in the absence and presence of S9.

## 5.6 GENETIC TOXICITY IN VIVO

The effects on micronucleus formation in bone marrow cells of rats and mice have been studied following ethylene inhalation [43]. Each group consisted of 10 animals of each of the two species and they were dosed with concentrations of 0; 40; 1,000 and 3,000 ppm for 6 hours/ day, 5 days a week for 4 weeks. An ethylene oxide control group with both species was exposed using the same conditions at a concentration of 200 ppm. Bone marrow was collected approximately 24 hours after the final exposure. Ethylene did not produce, statistically significant, exposure related increases in the frequencies of micronucleated polychromatic erythrocytes in the bone marrow of either rats or mice, while ethylene oxide exposure resulted in significant increases in the frequencies in both species. It is not stated if the study was conducted according to GLP.

Absorption, distribution, elimination of ethylene and formation of haemoglobin and DNA adducts were studied in rats after inhalation of 300 ppm ethylene for 12 hours/day for 3 consecutive days [44]. DNA adduct formation was measured in liver and lymphocytes and haemoglobin adducts determined in erythrocytes. The adduct formation with ethylene was compared to other alkenes and adduct formation decreased with increasing number of carbon atoms in the molecule. This was an explorative study not conducted according to GLP.

Alkylation of 7-guanine was measured in DNA from liver spleen and testis of mice 14 hours after exposure by inhalation of <sup>14</sup>C-ethylene at an initial concentration of 11 ppm for 8 hours [45]. The degree of alkylation was much higher in the liver than in the other tissues. This study was an explorative non-GLP study.

## 5.7 CARCINOGENICITY

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The potential carcinogenicity of ethylene has been tested in a two years study with rats (Fischer - 344 inbred) [46]. The study was conducted prior to OECD Guideline 451 for carcinogenicity testing (1981), but still the study comply with this guideline except for some minor points. In the study, 960 rats were randomly divided into 4 groups of 120 animals of each sex and exposed 6 hr/day, 5 days/week to 0(control); 300; 1,000 and 3,000 ppm for up to 24 months.

During the course of the study there were observations of hair loss, deposits on and around the nose and eyes and gross eye abnormalities, but there were no obvious differences among the different treatment groups.

There was an overall increase in the number of animals exhibiting gross tissue masses for the test groups as compared with the control group, although this trend was not statistically significant. The spontaneous mortality (15.7 %) was roughly equal in all treated groups. The final body weights and total weight changes for treated males were higher than those in the control groups, but no dose-related pattern was seen.

There were no statistically significant differences among any of the treatment groups on any of the haematology, blood chemistry or other parameters investigated.

No gross or histopathologic tissue changes attributable to the effects of the test material were observed in any of the treated rats. The summary reports only few findings which could indicate any carcinogenic effect of the treatment, but lacks a conclusion at this point.

In a publication from the carcinogenicity study [41], it was concluded that the results provided "no evidence that ethylene at these concentrations causes chronic toxicity or is oncogenic in Fischer - 344 rats". However, this publication and the summary have later been criticised [47] since they do not discuss the mononuclear cell leukaemia described in the full report. It was claimed that the number of animals affected (out of 90) rose from 12 and 8 in the male and female control groups to 21 and 11, respectively in the groups receiving 3,000 ppm. On the other hand, it has been stated that mononuclear cell leukemia may occur in F344 rats at a background incidence > 75 %, and that a further increase in exposed animals is difficult to interpret with respect to human cancer development.

When the carcinogenic risk of ethylene was evaluated by the International Agency for Research on Cancer (IARC) in 1979 [1], no data were available to the working group on the carcinogenicity or mutagenicity of the substance in animals and humans. In supplement 7 published in 1987 [48] it is still summarised that no adequate data were available and ethylene is stated to be not classifiable as to its carcinogenicity to humans. The latest evaluation of ethylene by the IARC working group (1994) concludes that there is inadequate evidence in humans and in experimental animals for the carcinogenicity of ethylene [3]. Overall, ethylene was evaluated as not being classifiable as to its carcinogenicity to humans.

In the Ecotoxicity Profile database it is stated to be no information in the QSAR system which would suggest that this chemical is a potential carcinogen or mutagen [26].

In another recent evaluation of ethylene as a cancer risk factor it was concluded that it was a risk factor of concern [49]. This conclusion was based on the observed metabolism of ethylene to ethylene oxide, a compound which has been shown to be both mutagenic and carcinogenic. The linearity hypothesis for dose response relationship can not be applied in this case, since there is a saturation of the metabolism of ethylene. The findings from administration of high doses to animals can thus not be extrapolated to the human exposure level.

The carcinogenic potential of ethylene has also been reviewed in the BIBRA Bulletin [50]. This review concludes also on the basis of metabolic production of ethylene oxide that it is timely with

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a detailed reconsideration of the possible carcinogenic risks of inhaling ethylene. The evaluation also calls for re-evaluation of the need for a specific industrial limit of ethylene.

## 5.8 TOXICITY TO REPRODUCTION

The potential effects of ethylene inhalation on male and female rat reproduction and on growth and development of the offspring has been studied [70]. The experimental study was carried out according to GLP (OECD Guideline 421; Reproduction/Development Toxicity Screening Test).

Four groups of rats (10 females and 10 males per group) were dosed by head only inhalation for 6 hours daily; air only (control); 200; 1,000 or 5,000 ppm of ethylene (corresponding to 0; 230; 1,150 or 5,750 mg/m<sup>3</sup>). This dosing regime was calculated to give about 80; 400 and 2,000 mg/kg/day of ethylene for the three dosed groups respectively. Since the uptake from the lungs most likely is in the range of 5-10 %, the absorbed dose probably was substantially less than the figures given above.

The test material was administered to parent animals for two weeks prior to mating, during the mating period and until the day prior to necropsy for the males (minimum 28 days) and until day 20 of gestation for the females. The females were allowed to litter and rear their offspring to day 4 post-partum, when they and their offspring were killed.

Morbidity, mortality, clinical condition, weight and food intake were observed throughout the study, and mating was carefully observed. For each female, litter data and also observations for each offspring were recorded. At termination of the study, all animals were subject to macroscopic examination for structural or pathological changes. Ovaries, testes and epididymides of the control and high dose animals were subject to a histopathological examination.

There were no deaths attributable to the test article, and body weight gain was not adversely affected during the pre-pairing, gestation or lactation periods. The treatment had no effect on fertility or fecundity and all females became pregnant. Litter size, sex ratio, mean pup weight and pup growth and clinical condition were not adversely affected by treatment.

Necropsy revealed no macroscopic finding suggestive of toxicity due to test article administration. There was no evidence of any toxic effect on the testis due to test substance administration and there were no other microscopic findings suggestive of toxicity due to test article administration.

In conclusion, head-only administration of ethylene at nominal concentrations of 200; 1,000 or 5,000 ppm was without evidence of toxicity or adverse effects on male and female reproductive performance, fertility, pregnancy, maternal and suckling behaviour and growth and development of the offspring from conception to Day 4 post-partum.

## 5.9 DEVELOPMENTAL TOXICITY/TERATOGENICITY

It is referred to the experimental study [70] carried out according to the OECD Guideline 421; Reproduction/Development Toxicity Screening Test. The study is summarised under point 5.8 above.

## 5.10 OTHER RELEVANT INFORMATION

- A. **Specific toxicities (neurotoxicity, immunotoxicity etc.)**  
No data

**B. Toxicodynamics, toxico-kinetics**

Cowles, A.L. et al [51], studied the uptake and distribution of four inhalation anaesthetics in dogs. In a series of 21 experiments, 13 large mongrel dogs were ventilated with a constant concentration of ethylene (1.4 % = 12 g/m<sup>3</sup>) and three other inhalation anaesthetics. Concentrations of the anaesthetic were measured by gas chromatography in alveolar gas, arterial blood, brain, muscle and central venous blood. The average times necessary for the partial pressure of ethylene to reach 50 % of the inspired partial pressure (1.4 %) were: alveolar gas, <2.0 min; arterial blood, <2.0 min; brain, 3.7 min; muscle, 8.2 min and central venous, 5.2 min.

**Biotransformation of ethylene to ethylene oxide**

Ehrenberg et. al, 1977 [52] showed that <sup>14</sup>C-labelled ethylene was metabolized to ethylene oxide when administered to male CBA mice by inhalation. This metabolism is of significant concern since ethylene oxide is a potent alkylating agent, a carcinogen and a genotoxicant, and hence more toxic than ethylene. The amount of epoxide formed was quantitatively determined from the degree of alkylation of cysteine and histidine residues in haemoglobin.

In a later study from the same laboratory [45], it was shown that ethylene oxide alkylated nucleophilic sites of mouse DNA. Since the ratio between the degree of alkylation of DNA and that of haemoglobin was the same when exposed to ethylene and ethylene oxide, it was concluded that the latter was the reactive intermediate formed from ethylene *in vivo*. A comparison of the degrees of alkylation obtained per unit exposure of ethylene oxide and ethylene, showed that at low levels of ethylene, about 8% of the inhaled amount was metabolized to ethylene oxide. The rate of ethylene oxidation followed saturation kinetics with increasing ethylene concentration. At 218 ppm ethylene, the oxidation rate was half of the maximal rate ( $K_m$  value). It was estimated that the maximal rate of metabolism ( $V_{max}$ ) of ethylene corresponds to exposure to an air level of 4 ppm of ethylene oxide.

After exposing rats to automotive engine exhaust, Tännqvist et. al., 1988 [53] identified alkylated amino acids in haemoglobin. These resulted from conversion of about 5-10 % of inhaled ethylene and propylene to their respective epoxides which again alkylated the nucleophilic sites in haemoglobin. This quantification of the fraction of ethylene to be oxidised form agreed very well with the conversion factor of around 8 % found for the mouse in the above mentioned study [45].

Results from Tännqvist and Ehrenberg in 1990, estimate that in humans, some 6 % of inhaled ethylene in mainstream smoke is converted to ethylene oxide in smokers [54] and some 3 % in non-smokers [55].

Metabolic conversion of ethylene to ethylene oxide results in the formation of adducts to DNA and proteins, and this offers a means for identifying ethylene exposure *in vivo*. Determination of haemoglobin adducts using the N-alkyl Edman method has proven valuable [53]. This method has been used for monitoring adduct formation after ethylene exposure from different sources [49].

**Toxicity of ethylene oxide**

Ethylene oxide causes dose-related increases in the incidence of gliomas, peritoneal mesotheliomas and mononuclear cell leukemias in F 344 rats and lymphomas and adenomas/adenocarcinomas of the lung, uterus, harderian gland and mammary gland in B6C3F1 mice (for a review see Walker et. al., 1990 [56]).

Epidemiologic data on ethylene oxide support the anticipation that ethylene oxide is a carcinogenic agent. When mortality and incidence of cancer in totally 733 workers exposed to



ethylene oxide were assessed, 8 cases of leukaemia and 6 cases of stomach cancer occurred, while the expected numbers were 0.8 and 0.65 respectively [57].

In vivo as well as in vitro, ethylene oxide is seen to react both with amino acid residues in proteins and with the purine bases in DNA. When mouse, human or rat erythrocytes were exposed to ethylene oxide, the main reaction products with haemoglobin were 2-hydroxyethylations of cysteines, N-terminal valine, imidazole nitrogens of histidines and carboxylic groups [58]. The main reaction product after reaction with calf thymus DNA was N-7-(2-hydroxyethyl) guanine, whereas O-6-(2-hydroxyethyl)guanine was only about 0.5 % of this. Species differences were also observed, as rat and mouse erythrocytes were more susceptible to alkylation than the human erythrocytes.

The alkylation of DNA-bases with ethylene oxide has been studied further after exposure of rats to ethylene oxide by inhalation [59, 56, 60]. The main alkylation site both in vivo and in vitro is the N-7 position in guanine, resulting in 7-(2-hydroxyethyl) guanine, and this modification is probably the reason for its carcinogenic and mutagenic effects.

The IARC working group evaluated ethylene oxide in 1994 and came to the overall conclusion that it was carcinogenic to humans [61]. This was mainly based on the evidence for carcinogenicity from experimental studies in animals.

#### **Effects of PCB-pre-treatment on ethylene toxicity and biotransformation**

It has been demonstrated that ethylene, as well as halogenated ethylenes are acute hepatotoxic in rats pretreated with polychlorinated biphenyl (PCB) [62]. The hepatotoxicity was evident as increased serum alanine- $\alpha$ -ketoglutarate transaminase (SAKT) and sorbitol dehydrogenase (SDH) in rats pretreated with PCB and exposed to 20,000 ppm ethylene for 4 hours. Without pretreatment with PCB, ethylene and halogenated ethylenes are not acute toxic. From these findings it was suggested that the acute toxicity was mediated through epoxide intermediates formed by hepatic mixed function oxidases induced by the PCB pre-treatment.

When rats were exposed to ethylene in a closed desiccator jar chamber, the rate of metabolic elimination of the compound is influenced by pretreatment with PCB (single dose of Aroclor 1254, 500 mg/kg in oil 6 days prior to the experiment) [63]. Biotransformation of ethylene lead to ethylene oxide which was exhaled.

The effects of PCB pre-treatment and high exposure levels of ethylene, due to induction of mono-oxygenases and increased formation of ethylene oxide, demonstrates that the toxicity of ethylene is of concern for organisms also exposed to mono-oxygenase inducers. However, it should be kept in mind that the concentrations used are far above actual exposure levels.

### **5.11 EXPERIENCE WITH HUMAN EXPOSURE**

Ethylene was in general use as an anaesthetic for many years. It has been replaced by more modern anaesthetics, mostly due to the high explosion risk. Chronic injury in humans resulting from prolonged and repeated exposure to low concentrations of ethylene (less than 2.5 %) was not reported in "Patty's Industrial Hygiene and Toxicology (1981)" [11].

#### **Inhalation pharmacokinetics**

The inhalation of ethylene was investigated in human volunteers at atmospheric concentrations of up to 50 ppm. The uptake, exhalation and metabolism could be described by first-order kinetics [64]. The clearance due to uptake was low, only 5.6 %, while the rest was exhaled without entering the blood stream. Clearance due to metabolism was 36 % of systemic available ethylene. The biological half-life of ethylene was 0.65 hours. The alveolar retention of ethylene at steady

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state was calculated to be 2 %. The low uptake rate of ethylene was considered due to its low solubility in blood.

### **Reproduction effects**

In a preliminary study, the miscarriage rate (six out of 15 pregnancies) amongst Swedish women who had worked in the local petrochemical industry was higher than that seen in 1549 women outside the industry. Ethylene was the main product in four of the five local petrochemical plants. No data were provided on occupational levels but measurements made in areas surrounding the plants indicated that ethylene was present in concentrations up to tenfold higher than the other pollutants (propylene, ethane, propane and phenol) [65].

A brief abstract notes that there was a higher than expected rate of miscarriage and gynaecological disease among female operatives of a polyethylene plant who were exposed to ethylene concentrations in the range of about 40-60 ppm and high levels of noise [66].

### **Carcinogenicity**

A preliminary study found no increase in lung cancer incidence in 31 workers exposed to ethylene (at unspecified levels) at a US petrochemical factory [67].

A study of workers at an US petrochemical plant found that an increased risk of developing brain cancer was associated with exposure to (unspecified levels of) a number of chemicals including ethylene. However, the investigators were unconvinced that the association reflected a casual relationship [68].

### **Work Place Exposure**

Personal and stationary monitoring of ethylene in a company where this gas was used for controlling the ripening of bananas showed air concentrations to be in the range of 0.02-3.35 ppm (0.02 - 3.85 mg/m<sup>3</sup>), with an estimated average concentration of 0.3 ppm (0.35 mg/m<sup>3</sup>). In a study on exposure of fire-fighters, samples taken during the "knockdown" phase of a fire showed a concentration of 46 ppm (53 mg/m<sup>3</sup>) ethylene, while none was detected during the "overhaul" phase [3]

A study was carried out among workers at a Swedish petrochemical plant using measurements of haemoglobin adducts formed from ethylene oxide for monitoring of ethylene exposure [69]. The study was carried out in two parts, part one in 1989 and part two in 1993. Eight workers exposed to high levels of ethylene ( 4 mg/m<sup>3</sup>) and 3 workers exposed to low levels (0.1 -0.3 mg/m<sup>3</sup>) were compared to nine controls exposed to 0.01 mg/m<sup>3</sup>. All exposed workers showed elevated levels of haemoglobin adducts and adduct formation was dose-related. The results indicated that about 1 % of the inhaled ethylene was metabolized to ethylene oxide.

The second part of the study, which included four workers, was designed to more accurately determine the exposure levels, which turned out to have a mean of 4.5 mg/m<sup>3</sup>. The results confirmed part one, showing that about 1 % of inhaled ethylene was metabolized to ethylene oxide and the maximum fraction to be converted was estimated to be 4 %.

The peak level of ethylene reported for human exposure is about 50 ppm ( 57.5 mg/m<sup>3</sup> ), while 3.5 ppm ( 4.0 mg/m<sup>3</sup> ) has been characterized as a high average level for longer term exposure. The conversion will then correspond to maximum 2 ppm (3.6 mg/m<sup>3</sup>) of ethylene oxide for the peak level and to maximum 0.14 ppm (0.25 mg/m<sup>3</sup>) for the high averaged level. Given occupational exposure limit levels for ethylene oxide (time-weighted averages) are 1.8 mg/m<sup>3</sup> (Denmark, Japan, USA, Norway) and 2.0 mg/m<sup>3</sup> (France, Canada, Sweden) [3].

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**6 REFERENCES**

1. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Some monomers, plastics and synthetic elastomers, and acrolein. 1979, Vol. 19:157-86.
2. CMAI, World Light Olefin Analysis, 1996.
3. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Some Industrial Chemicals. 1994, Vol. 60:45-71.
4. Granton RL, Roger DJ. Ullmann`s encyclopedia of industrial chemistry.1987;10:45-93.
5. Sawada S, Totsuka T. Natural and anthropogenic sources and fate of atmospheric ethylene. Atmospheric Environment. 1986;20:821-32
6. Hanch C, Leo A. Substituent Constants for Correlation Analysis in Chemistry and Biology, John Wiley & Sons, New York, 1979.
7. The Merck Index, 10th edn, 1983, Merck & Co.
8. HSDB Database, 1991.
9. Verschueren K. Handbook of Environment Data on Organic Chemicals. Second edition. 1983.
10. Seidell A. Solubilities of organic compounds. 1941;2:96.
11. Clayton GD, Clayton FE. Patty`s industrial hygiene and toxicology. 1981. Vol 2b. Toxicology 3rd edn. John Wiley & sons.
12. Ruth JH. Odour thresholds and irritation levels of several chemical substances: a review. Am Ind Hyg Assoc J. 1986;47:A142-51.
13. Atkinson R, Carter WPL. Chem Rev. 1984;84:437-70.
14. Atkinson R. Journal of Phys and Chem Reference Data. 1989, Monograph no. 1.
15. Howard PH. et. al. Handbook of environmental degradation rates. 1991, Lewis Publ. Inc.
16. Rudolph J, Johnen FJ. Measurements of light atmospheric hydrocarbons over the Atlantic in regions of low biological activity. 1990;
17. Swinnerton JW, Lamontagne RA. Oceanic distribution of low-molecular-weight hydrocarbons. Baseline measurements. Environ Sci & Technol. 1974;8:657-63.
18. O'Mara MM. J Fire Flammability. 1974;5:34-53.
19. Mackay D, Paterson S, Shin WY. Generic Models for Evaluation of the Regional Fate of Chemicals. Chemosphere 1992; 24: 695-717.
20. BIODEG database, Jan. 1993, version 3.03.U.S EPA, OTC, Washington DC.
21. EUCLID Dataset, Ethylene, BASF AG, May 1994.

- 
22. Veith GD, Kosian P. Estimating bioconcentration potential from octanol/water partition coefficients. in Mackay D et al. (eds.) Physical behaviour of PCBs in the Great Lakes. Ann Arbor Science Publishers, Ann Arbor, Mi. 1983:269-82.
  23. Mcku JE, Wolf HW (eds). Water quality criteria. US Department of Commerce, NTIS. 1963;186.
  24. Sheford VE. An experimental study of the effects of gas waste upon fishes with special reference to stream pollution. Bulletin of the Illinois State Laboratory of Natural History. 1917;11:379.
  25. Gutsell JS. Danger to fisheries from oil and tar pollution of waters. Bureau of Fisheries. Document 910, 1921.
  26. Ecotoxicity Profile Data Base (EPA), Environ. Res. Lab. Duluth, Cont.;SCI. Out. Prog 18/720.
  27. Van Leeuwen et al. Predictions of aquatic toxicity of High-Production-Volume-Chemicals. Part B. Publikatiereeks Stoffen, Veiligheid, Straling br 1993/9B. Ministerie van Volkshuisvesting, Ruimtelijke Ordening en Milieubeheer. 1993.
  28. Landry MM, Fuerst R. Gas ecology of bacteria. Dev Ind Microbiol. 1968;9:370-81
  29. Bilthoven SW, Bont PFH, Janus JA, Rab E. Exploratory report ethylene. Report no. 710401010, National Institute of Public Health and Environmental Protection, The Netherlands. 1991.
  30. Sloof W, Bont PFH, Janus JA, Rab E. Exploratory report ethylene. Report no 710401010, National Institute of Public Health and Environmental Protection, The Netherlands. 1991.
  31. Smith WH. Air pollution and forests - interaction between air contaminants and forest ecosystems. Springer Verlag 1981;26.
  32. Rademaker BC, Guiné EP, Van de Plassche EJ. The derivation of preliminary maximum permissible concentrations of volatile compounds in air. RIVM report no.: 679101009. 1993. National Institute of Public Health and Environmental Protection, Postbus 1, 3720 BA Bilthoven, The Netherlands.
  33. Davidson OW. Effects of Ethylene on Orchid Flowers. Proc Amer Soc Hort Sci 1949;53:440.
  34. Flury. Arch Exp Pathol Pharmacol. 1928;138:65.
  35. Gaeb S, Cochrane WP, Parlar H, Korte F. Zeitschrift für Naturforschung. 1975;2.
  36. Chemical Industry Institute of Toxicology, Research Triangle Park, North Carolina, USA. CIIT summary report, a ninety day inhalation toxicology study in albino rats exposed to atmospheric ethylene gas. 1977.
  37. Fink B.R. Toxicity of anesthetics. Williams & Wilkins Co. Baltimore, 1968.
  38. Krasovitskaya ML, Mabyarova LK. Gig Sanit 1968;33:5-7.
  39. Krasovitskaya ML, Mabyarova LK. Beol Deistive I Gig 1966.

- 
40. Victorin K, Ståberg M. A method for studying the mutagenicity of some gaseous compounds in *Salmonella typhimurium*. *Environ mol Mutag*. 1988;11:65 & 79.
  41. Hamm TE jr, Guest D, Dent JG. Chronic toxicity and oncogenicity bioassay of inhaled ethylene in Fisher-344 rats. *Fundamental and Appl Toxicol*. 1984;4:473-8.
  42. Huges TJ. et al. *Chemical Abstracts*. 1984;101:85417t.
  43. Vergenes JS, Pritts IM. Effects of ethylene on micronucleus formation in the bone marrow of rats and mice following four weeks of inhalation exposure. *Mutat Res*. 1994; 324: 87-91.
  44. Eide I. et al. Uptake, distribution and formation of haemoglobin and DNA adducts after inhalation of C2-C8 1-alkene (olefins) in the rat. *Carcinogenesis*. 1995;16: 1603-9.
  45. Segerbäk D. Alkylation of DNA and haemoglobin in the mouse following exposure to ethene and ethene oxide. *Chem Biol Interactions*. 1983;45:139-51.
  46. Chemical Industry Institute of Toxicology, Research Triangle Park, North Carolina, USA. CIIT report, two year chronic inhalation toxicology study with ethylene in F 344 rats, 1979.
  47. Rostron C. Ethylene metabolism and carcinogenicity. *FD Chem. Toxic* 19xx;24:70.
  48. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Overall evaluations of carcinogenicity: an updating of IARC monographs volumes 1 to 42. IARC, Suppl 7, 1987.
  49. Tännqvist M. Is ambient ethene a cancer risk factor ? *Environ Health Perspectives*. 1994;102 (Suppl 4):157-160.
  50. Hopkins J. The carcinogenic potential of ethylene. *BIBRA Bulletin*. 1993;32 (Oct.): 245-7.
  51. Cowles AL, Borgstedt HH, Gillies AJ. The uptake and distribution of four inhalation anesthetics in dogs. *Anaesthesiology*. 1972;36:558-70.
  52. Ehrenberg L, Osterman-Galkar S, Segerbäk D, Svensson K, Calleman CJ. Evaluation of genetic risks of alkylating agents. iii. Alkylation of haemoglobin after metabolic conversion of ethene to ethene oxide *in vivo*. *Mutation Res*. 1977;45:175-84.
  53. Tännqvist M, Kautiainen A, Gatz RN, Ehrenberg L. Haemoglobin adducts in animals exposed to gasoline and diesel exhausts 1. Alkene. *J Appl Toxicol*. 1988;8:159-70.
  54. Tännqvist M, Ehrenberg L. Approaches to risk assessment of automotive engine exhausts. In: Vaino H, Sorsa M, McMichael AJ (eds.) *Complex Mixtures and Cancer Risk*. Lyon IARC, 1990:277-87.
  55. Tännqvist M et. al. Ethylene oxide doses in ethene-exposed fruit store workers. *Scand J Work Environ Health*. 1989;15:436-8.
  56. Walker VE, Fennell TR, Boucheron JA, Fedtke N, Ciroussel F, Swenberg JA. Macromolecular adducts of ethylene oxide: a literature review and a time-course study on the formation of 7-(2-hydroxyethyl) guanine following exposures of rats by inhalation. *Mutation Res*. 1990;233:151-64.

- 
57. Hogstedt C, Aringer L, Gustavsson A. Epidemiologic support for ethylene oxide as a cancer-causing agent. *JAMA* 1986;255:1575-8.
  58. Segerbäck D. Reaction products in haemoglobin and DNA after in vitro treatment with ethylene oxide and n-(2-hydroxyethyl)-n-nitrosurea. *Carcinogenesis*. 1990;11:307-12.
  59. Young LT, Habraken Y, Ludlum DB, Santella RM. Development of monoclonal antibodies recognizing 7-(2-hydroxyethyl) guanine and imidazole ring-opened 7-(2-hydroxyethyl)guanine. *Carcinogenesis* 1990;11:1685-9.
  60. Ffist U, Marczynski B, Kasemann RP. Determination of 1-(2-hydroxyethyl) guanine with gas chromatography/mass spectrometry as a parameter for genotoxicity of ethylene oxide. *Arch Toxicol*. 1989;Suppl.13:250-3.
  61. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Some Industrial Chemicals. 1994, Vol. 60:73-159.
  62. Conolly RB, Jaeger RJ. Acute hepatotoxicity of ethylene and halogenated ethylenes after PCB pretreatment. *Environmental Health Perspectives*. 1977;21:131-5.
  63. Filser JG, Bolt HM. Exhalation of ethylene oxide by rats on exposure to ethylene. *Mutation Res*. 1983;120:57-60.
  64. Filser JG, Denk B, Tönqvist M, Kessler W, Ehrenberg L. Pharmacokinetics of ethylene in man; body burden with ethylene oxide and hydroxyethylation of haemoglobin due to endogenous and environmental ethylene. *Arch Toxicol*. 1992;66: 157-63.
  65. Axelsson G, Molin I. *Int J Epidemiol*. 1988;17:363.
  66. Yakubova ZN. *Chemical Abstracts*. 1976;87:28224w.
  67. Bond GG, et al. *Am J Epidemiol*. 1986;124:53.
  68. Leffingwell SS, et al. *Neuroepidemiology*. 1983;2:179.
  69. Tönqvist M, Granath F. Studier av doser av etylenoxid i etenexponerad personal vid Borealis/Neste. Slutrapport. [Studies of doses of ethylene oxide in personnel exposed to ethylene at Borealis/Neste. Final Report.]. May 17, 1994.
  70. Aveyard L. Ethylene: Inhalation (Head-only) Reproduction/Development Toxicity Study in the Rat. Corning Hazleton Report No. 1458/2-1050, April 1996.
  71. Riley S. Ethylene: Induction of Chromosome Aberrations in Cultured Chinese Hamster Ovary (CHO) cells. Corning Hazleton Report No. 1458/1-1052, April 1996.
  72. Victorin K. Uppdaterad hälsoriskbedömning av etenoxid, eten och propen. Institutet för miljömedicin - Karolinska institutet, IMM-Rapport 8/92, Stockholm, 1992. [Updated medical risk assessment of ethylene oxide, ethylene and propene. Institute of environmental medicine].
  73. BIBRA Tox Profile on Ethylene, 1993
  74. Mattock SD. Ethylene: Inhibition of growth to the Alga *Selenastrum capricornutum*. Corning Hazleton Report No. 1458/3-1018, May 1996.

75. Atkinson, R. Kinetics and mechanisms of gas-phase reactions of the hydroxyl radical with organic compounds under atmospheric conditions. *Chem. Rev.* 85:69-201. 1986.
76. IPCC, *Climate change 1994*. 1995.
77. Van der Eerden, L.J. *Luchtkwaliteitsevaluatie met behulp van indicatorplanten en agrarische gewassen in de omgeving van industrieterrein Moerdijk*. IPO report 279. 1981.

***EXTRACT FROM IRPTC LEGAL FILE***



File: 17.01 LEGAL

rn : 300046

systematic name:Ethene  
 common name :ethylene  
 reported name :ETHYLENE  
 cas no :74-85-1  
 area : CAN  
 rtecs no :KU5340000  
 type : REG

subject	specification	descriptor
AIR	OCC	TLV

Appendix E - simple asphyxiant. Prescribed by the Canada Occupational Safety and Health Regulations, under the Canada Labour Code (administered by the Department of Employment and Immigration). The regulations state that no employee shall be exposed to a concentration of an airborne chemical agent in excess of the value for that chemical agent adopted by ACGIH (American Conference of Governmental Industrial Hygienists) in its publication entitled: "Threshold Limit Value and Biological Exposure Indices for 1985-86". The regulations also state that the employer shall, where a person is about to enter a confined space, appoint a qualified person to verify by means of tests that the concentration of any chemical agent or combination of chemical agents will not result in the exposure of the person to a concentration in excess of the value indicated above. These regulations prescribe standards whose enforcement will provide a safe and healthy workplace.  
 entry date: OCT 1994 effective date: 24MCH1994

amendment: CAGAAK, CANADA GAZETTE PART II, 128 , 7 , 1513 , 1994

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File: 17.01 LEGAL

rn : 301890

systematic name:Ethene  
 common name :ethylene  
 reported name :ETHYLENE  
 cas no :74-85-1  
 area : CAN  
 rtecs no :KU5340000  
 type : REG

subject	specification	descriptor
TRNSP LABEL PACK		CLASS RQR

Applies to ethylene or ethylene, compressed. Schedule II, List II - Dangerous Goods other than Explosives: PIN (Product Identification No.):  
 UN1962. Class (2.1): Flammable gas. Special provisions: 102. Passenger Vehicles: Prohibited. Passenger Ship: Prohibited. Prescribed by the Transportation of Dangerous Goods Regulations, under the Transportation of Dangerous Goods Act (administered by the Department of Transport). The act and regulations are intended to promote safety in the transportation of dangerous goods in Canada, as well as provide comprehensive regulations applicable to all modes of transport across Canada. These are based on United Nations recommendations. The act and regulations should be consulted for details. Information is entered under the proper shipping name found in the regulations; this may include general groups of chemical substances.  
 entry date: OCT 1994 effective date: 02DEC1993

amendment: CAGAAK, CANADA GAZETTE PART II, 127 , 25 , 4056 , 1993

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File: 17.01 LEGAL

rn : 301891

systematic name:Ethene  
 common name :ethylene  
 reported name :ETHYLENE  
 cas no :74-85-1  
 area : CAN  
 rtecs no :KU5340000  
 type : REG

subject	specification	descriptor
TRNSP		CLASS
LABEL		RQR
PACK		

Applies to ethylene, refrigerated liquid. Schedule II, List II - Dangerous Goods other than Explosives: PIN (Product Identification No.):UN1038. Class (2.1): Flammable gas. Special provisions: 102. Consumer Commodity: Prohibited. Limited Quantity: Prohibited. Passenger Vehicles:Prohibited. Passenger Ship: Prohibited. Prescribed by the Transportation of Dangerous Goods Regulations, under the Transportation of Dangerous Goods Act (administered by the Department of Transport). The act and regulations are intended to promote safety in the transportation of dangerous goods in Canada, as well as provide comprehensive regulations applicable to all modes of transport accross Canada. These are based on United Nations recommendations. The act and regulations should be consulted for details. Information is entered under the proper shipping name found in the regulations; this may include general groups of chemical substances.  
 entry date: OCT 1994 effective date: 02DEC1993

amendment: CAGAAK, CANADA GAZETTE PART II, 127 , 25 , 4056 , 1993

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File: 17.01 LEGAL

rn : 305377

systematic name:Ethene  
 common name :ethylene  
 reported name :ETHYLENE  
 cas no :74-85-1  
 area : CAN  
 rtecs no :KU5340000  
 type : REG

subject	specification	descriptor
TRNSP		CLASS
LABEL		RQR
PACK		

Applies to ethylene, acetylene and propylene in mixtures, refrigerated liquid. Schedule II, List II - Dangerous Goods other than Explosives: PIN (Product Identification No.): UN3138. Class (P): Prohibited. Prescribed by the Transportation of Dangerous Goods Regulations, under the Transportation of Dangerous Goods Act (administered by the Department of Transport). The act and regulations are intended to promote safety in the transportation of dangerous goods in Canada, as well as provide comprehensive regulations applicable to all modes of transport accross Canada. These are based on United Nations recommendations. The act and regulations should be consulted for details. Information is entered under the proper shipping name found in the regulations; this may include general groups of chemical substances.

entry date: OCT 1994

effective date: 02DEC1993

amendment: CAGAAK, CANADA GAZETTE PART II, 127 , 25 , 4056 , 1993

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File: 17.01 LEGAL

rn : 402419

systematic name:Ethene  
 common name :ethylene  
 reported name :ETHYLENE  
 cas no :74-85-1  
 area : CZE  
 rtecs no :KU5340000  
 type : REG

subject	specification	descriptor
AIR	EMI	MXL

GENERAL EMISSION LIMIT: 150 MG/M3 (IT APPLIES TO THE SUM OF ACETONE, ALKYLALCOHOLS, BIPHENYL, 2-BUTANONE, BUTYL ACETATE, DIBUTYLEETHER, DIETHYLEETHER, DIPHENYLEETHER, DICHLORODIFLUOROMETHANE, 1,2-DICHLOROETHYLENE, DICHLOROMETHANE, DIISOPROPYLEETHER, DIMETHYLEETHER, ETHYL ACETATE, ETHYLENE GLYCOL, 4-HYDROXY-4-ETHYL-2-PENTANONE, CHLOROETHANE, METHYL BENZOATE, 4-METHYL-2-PENTANOL, N-METHYLPYRROLIDONE, OLEFINS (EXCEPT 1,3-BUTADIENE), PARAFINS (EXCEPT METHANE) AND TRICHLOROFLUOROMETHANE IF THEIR MASS FLOW > 3 KG/H).  
 entry date: DEC 1994  
 effective date: 1SEP1992

title: PROVISION OF FEDERAL COMMITTEE FOR ENVIRONMENT TO ACT NO. 309 FROM 9 JULY 1991 ON AIR PROTECTION AGAINST AIR POLLUTANTS  
 original : SZCFR\*, SBIRKA ZAKONU CESKE A SLOVENSKE FEDERATIVNI REPUBLIKY(COLLECTION OF THE LAW OF CZECH AND SLOVAK FEDERAL REPUBLIC), , 84 , 2061 , 1991  
 amendment: SZCFR\*, SBIRKA ZAKONU CESKE A SLOVENSKE FEDERATIVNI REPUBLIKY(COLLECTION OF THE LAW OF CZECH AND SLOVAK FEDERAL REPUBLIC), , 84 , 2398 , 1992

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File: 17.01 LEGAL

rn : 402820

systematic name:Ethene  
 common name :ethylene  
 reported name :ETHYLENE  
 cas no :74-85-1  
 area : CZE  
 rtecs no :KU5340000  
 type : REG

subject	specification	descriptor
TRNSP		RQR

ROAD TRANSPORT OF THE SUBSTANCE IN THE QUANTITY > 1000 KG IS ALLOWED ONLY WITH THE PERMISSION GIVEN BY RELEVANT AUTHORITY.  
 entry date: AUG 1994  
 effective date: 01APR1992

title: THE DECREE OF FEDERAL MINISTRY OF TRANSPORT NO. 122 WHICH PROMULGATE THE ACT ON ROAD TRANSPORT  
 original : SZCSR\*, SBIRKA ZAKONU CESKOSLOVENSKE SOCIALISTICKE REPUBLIKY(COLLECTION OF THE LAW OF CZECHOSLOVAK SOCIALIST REPUBLIC), , 24 , 606 , 1979  
 amendment: SZCFR\*, SBIRKA ZAKONU CESKE A SLOVENSKE FEDERATIVNI REPUBLIKY





File: 17.01 LEGAL

rn : 1122614

systematic name:Ethene  
 common name :ethylene  
 reported name :ETHYLENE  
 cas no :74-85-1  
 area : RUS  
 rtecs no :KU5340000  
 type : REG

subject	specification	descriptor
AIR	OCC	MAC CLASS

CLV: 100MG/M3 (VAPOUR) HAZARD CLASS: IV  
 entry date: MAY 1990  
 effective date: 01JAN1989

amendment: GOSTS\*, GOSUDARSTVENNYI STANDART SSSR (STATE STANDARD OF USSR), 12.1.005 , , , 1988

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File: 17.01 LEGAL

rn : 1123448

systematic name:Ethene  
 common name :ethylene  
 reported name :ETHYLENE  
 cas no :74-85-1  
 area : RUS  
 rtecs no :KU5340000  
 type : REG

subject	specification	descriptor
AQ	SURF	MAC CLASS

0.5MG/L HAZARD CLASS: III  
 entry date: JUL 1990  
 effective date: 1JAN1989

amendment: SPNPV\*, SANITARNYE PRAVILA I NORMY OKHRANY POVERKHNOSTNYKH VOD OT ZAGRIAZNENIA (HEALTH REGULATION AND STANDARDS OF SURFACE WATER PROTECTION FROM CONTAMINATION), 4630-88 , , , 1988

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File: 17.01 LEGAL

rn : 1317115

systematic name:Ethene  
 common name :ethylene  
 reported name :ETHYLENE  
 cas no :74-85-1  
 area : USA  
 rtecs no :KU5340000  
 type : REG

subject	specification	descriptor
USE		RSTR
FOOD	ADDIT	RSTR
STORE		RSTR
MANUF		RSTR
PACK		RSTR



; Summary - FACILITIES THAT EXCEEDED A MANUFACTURING, IMPORTATION, OR PROCESSING THRESHOLD OF 25,000 LBS OR THE USE OF 10,000 LBS FOR THIS CHEMICAL MUST REPORT TO EPA ANY RELEASES OF THE CHEMICAL (OR CATEGORY CHEMICAL) TO AIR, LAND, WATER, POTW, UNDERGROUND INJECTION, OR OFF SITE TRANSFER. THIS REGULATION COVERS STANDARD INDUSTRIAL CLASSIFICATION

(SIC) CODES 20-39 ONLY).

entry date: OCT 1991

effective date: 1987

title: SUPERFUND AMENDMENTS AND REAUTHORIZATION ACT, TITLE III. EPCRA SECTION 313 LIST OF TOXIC SUBSTANCES

original : CFRUS\*, CODE OF FEDERAL REGULATIONS, 40 , 372 , 65 , 1988

amendment: CFRUS\*, CODE OF FEDERAL REGULATIONS, 40 , 372 , 65 , 1988

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File: 17.01 LEGAL

rn : 1340625

systematic name:Ethene

common name :ethylene

reported name :ETHYLENE

cas no :74-85-1

rtecs no :KU5340000

area : USA

type : REC

subject	specification	descriptor
AIR	OCC	TLV

SIMPLE ASPHYXIANT; Summary - THIS THRESHOLD LIMIT VALUE IS INTENDED FOR USE IN THE PRACTICE OF INDUSTRIAL HYGIENE AS A GUIDELINE OR RECOMMENDATION IN THE CONTROL OF POTENTIAL HEALTH HAZARDS.

entry date: DEC 1991

effective date: 1989

title: THRESHOLD LIMIT VALUES

original : ACGIH\*, AMERICAN CONFERENCE OF GOVERNMENT INDUSTRIAL HYGIENISTS, , , 11 , 1989

amendment: ACGIH\*, AMERICAN CONFERENCE OF GOVERNMENT INDUSTRIAL HYGIENISTS, , , 11 , 1991

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File: 17.01 LEGAL

rn : 1408383

systematic name:Ethene

common name :ethylene

reported name :ETHYLENE

cas no :74-85-1

rtecs no :KU5340000

area : EEC

type : REG

subject	specification	descriptor
FOOD		RQR
GOODS		MXL
GOODS		PRMT

THE SUBSTANCE IS INCLUDED IN THE LIST OF AUTHORIZED MONOMERS AND OTHER STARTING SUBSTANCES, WHICH SHALL BE USED FOR THE MANUFACTURE OF PLASTICS AND ARTICLES INTENDED TO COME INTO CONTACT WITH FOODSTUFFS. THE USE OF THE SUBSTANCE IS SUBJECT TO THE RESTRICTIONS SPECIFIED THEREIN. PLASTIC MATERIALS AND ARTICLES SHALL NOT TRANSFER THEIR CONSTITUENTS TO FOODSTUFFS IN QUANTITIES EXCEEDING 10MG/DM2 OF SURFACE AREA OF MATERIAL OR ARTICLE OR 60 MG/KG OF FOODSTUFFS IN THE SPECIFIED CASES.

VERIFICATION OF COMPLIANCE WITH THE MIGRATION LIMITS SHALL BE CARRIED



OUT IN ACCORDANCE WITH DIRECTIVES 82/711/EEC AND 85/572/EEC.  
 entry date: SEP 1995 effective date: 01JAN1991

title: COMMISSION DIRECTIVE OF 23 FEBRUARY 1990 RELATING TO PLASTICS MATERIALS AND ARTICLES INTENDED TO COME INTO CONTACT WITH FOODSTUFFS (90/128/EEC)  
 original : OJEC\*\*, OFFICIAL JOURNAL OF THE EUROPEAN COMMUNITIES, L75 , 19 , 1990  
 amendment: OJEC\*\*, OFFICIAL JOURNAL OF THE EUROPEAN COMMUNITIES, L90 , 26 , 1993

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File: 17.01 LEGAL

rn : 1421775

systematic name:Ethene  
 common name :ethylene  
 reported name :ETHYLENE  
 cas no :74-85-1 rtecs no :KU5340000  
 area : EEC type : REG

subject	specification	descriptor
CLASS		CLASS
LABEL		RQR
PACK		RQR

CLASS: F+ - EXTREMELY FLAMMABLE; EXTREMELY FLAMMABLE (R 12). LABEL: F+ - EXTREMELY FLAMMABLE; EXTREMELY FLAMMABLE (R 12); (KEEP OUT OF THE REACH OF CHILDREN (S 2)); KEEP CONTAINER IN A WELL-VENTILATED PLACE (S 9); KEEP AWAY FROM SOURCES OF IGNITION - NO SMOKING (S 16); TAKE PRECAUTIONARY MEASURES AGAINST STATIC DISCHARGES (S 33).  
 entry date: AUG 1994 effective date: JAN1994

title: COUNCIL DIRECTIVE 67/548/EEC OF 27 JUNE 1967 ON THE APROXIMATION OF THE LAWS, REGULATIONS AND ADMINISTRATIVE PROVISIONS RELATING TO THE CLASSIFICATION, PACKAGING AND LABELLING OF DANGEROUS SUBSTANCES  
 original : OJEC\*\*, OFFICIAL JOURNAL OF THE EUROPEAN COMMUNITIES, 196 , 1 , 1967  
 amendment: OJEC\*\*, OFFICIAL JOURNAL OF THE EUROPEAN COMMUNITIES, L 13 , 1 , 1994

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File: 17.01 LEGAL

rn : 1601163

systematic name:Ethene  
 common name :ethylene  
 reported name :ETHYLENE  
 cas no :74-85-1 rtecs no :KU5340000  
 area : UN type : REC

subject	specification	descriptor
TRNSP		CLASS
LABEL		
PACK		

HAZARD CLASS: 2.1 = FLAMMABLE GAS. PACKING METHOD: M. (APPLIES TO REFRIGERATED LIQUID ETHYLENE). UN NO.1038.  
 entry date: SEP 1994 effective date: 1993

title: RECOMMENDATIONS ON THE TRANSPORT OF DANGEROUS GOODS  
 amendment: !UNTDG\*, UN TRANSPORT OF DANGEROUS GOODS, RECOMMENDATION PREPARED BY THE COMMITTEE OF EXPERTS ON THE TRANSPORT OF

DANGEROUS GOODS, , , 19 , 1993

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File: 17.01 LEGAL

rn : 1601490

systematic name:Ethene  
 common name :ethylene  
 reported name :ETHYLENE  
 cas no :74-85-1  
 area : UN

rtecs no :KU5340000  
 type : REC

subject	specification	descriptor
TRNSP		CLASS
LABEL		
PACK		

HAZARD CLASS: 2.1 = FLAMMABLE GAS. (APPLIES TO COMPRESSED ETHYLENE). UN  
 UN NO. 1962.

entry date: SEP 1994 effective date: 1993

title: RECOMMENDATIONS ON THE TRANSPORT OF DANGEROUS GOODS  
 amendment: !UNTDG\*, UN TRANSPORT OF DANGEROUS GOODS, RECOMMENDATION  
 PREPARED BY THE COMMITTEE OF EXPERTS ON THE TRANSPORT OF  
 DANGEROUS GOODS, , , 19 , 1993

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File: 17.01 LEGAL

rn : 1602034

systematic name:Ethene  
 common name :ethylene  
 reported name :ETHYLENE  
 cas no :74-85-1  
 area : UN

rtecs no :KU5340000  
 type : REC

subject	specification	descriptor
TRNSP		CLASS
LABEL		
PACK		

HAZARD CLASS: 2.1 = FLAMMABLE GAS. PACKING METHOD: M. (APPLIES TO  
 O ETHYLENE, ACETYLENE AND PROPYLENE IN MIXTURES, REFRIGERATED LIQUID,  
 CONTAINING AT LEAST 71.5 PERCENT ETHYLENE WITH NOT MORE THAN 22.5  
 PERCENT ACETYLENE AND NOT MORE THAN 6 PERCENT PROPYLENE). UN NO. 3138.

entry date: SEP 1994 effective date: 1993

title: RECOMMENDATIONS ON THE TRANSPORT OF DANGEROUS GOODS  
 amendment: !UNTDG\*, UN TRANSPORT OF DANGEROUS GOODS, RECOMMENDATION  
 PREPARED BY THE COMMITTEE OF EXPERTS ON THE TRANSPORT OF  
 DANGEROUS GOODS, , , 19 , 1993

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File: 17.01 LEGAL

rn : 1604990

systematic name:Ethene  
 common name :ethylene  
 reported name :ETHYLENE  
 cas no :74-85-1  
 area : IMO

rtecs no :KU5340000  
 type : REC

subject	specification	descriptor

TRNSP LABEL PACK	MARIN	CLASS
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HAZARD CLASS: 2(2.1) = FLAMMABLE GAS. (APPLIES TO REFRIGERATED LIQUID ETHYLENE). UN NO. 1038.

entry date: SEP 1994 effective date: 1991

title: INTERNATIONAL MARITIME DANGEROUS GOODS CODE (IMDG CODE)  
 amendment: IMCOC\*, IMO DANGEROUS GOODS CODE, RECOMMENDATION PREPARED BY THE MARITIME SAFETY COMMITTEE, 26-91 , , 10086 , 1991

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File: 17.01 LEGAL

rn : 1604991

systematic name: Ethene  
 common name : ethylene  
 reported name : ETHYLENE  
 cas no : 74-85-1 rtecs no : KU5340000  
 area : IMO type : REC

subject	specification	descriptor
TRNSP LABEL PACK	MARIN	CLASS

HAZARD CLASS: 2(2.1) = FLAMMABLE GAS. (APPLIES TO COMPRESSED ETHYLENE). UN NO. 1962.

entry date: SEP 1994 effective date: 1991

title: INTERNATIONAL MARITIME DANGEROUS GOODS CODE (IMDG CODE)  
 amendment: IMCOC\*, IMO DANGEROUS GOODS CODE, RECOMMENDATION PREPARED BY THE MARITIME SAFETY COMMITTEE, 26-91 , , 10085 , 1991

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File: 17.01 LEGAL

rn : 1604992

systematic name: Ethene  
 common name : ethylene  
 reported name : ETHYLENE  
 cas no : 74-85-1 rtecs no : KU5340000  
 area : IMO type : REC

subject	specification	descriptor
TRNSP LABEL PACK	MARIN	CLASS

HAZARD CLASS: 2\*2.1) = FLAMMABLE GAS. (APPLIES TO ETHYLENE, ACETYLENE AND PROPYLENE MIXTURES, REFRIGERATED LIQUID, CONTAINING AT LEAST 71,5 % ETHYLENE WITH NOT MORE THAN 22.5 % ACETYLENE AND NOT MORE THAN 6% PROPYLENE). UN NO. 3138.

entry date: SEP 1994 effective date: 1991

title: INTERNATIONAL MARITIME DANGEROUS GOODS CODE (IMDG CODE)  
 amendment: IMCOC\*, IMO DANGEROUS GOODS CODE, RECOMMENDATION PREPARED BY THE MARITIME SAFETY COMMITTEE, 26-91 , , 10084 , 1991