

HPA Compendium of Chemical Hazards

Petrol

Key Points

Fire

- Highly flammable
- Mixtures of petrol vapour and air may explode
- In the event of a fire involving petrol, use normal foam and normal fire kit with breathing apparatus

Health

- Serious lung injury may occur if droplets of petrol are inhaled (e.g. if vomiting occurs after ingestion)
- Harmful
- Inhalation may cause headache, dizziness and drowsiness.
- Often no symptoms occur following ingestion. In some cases, nausea, vomiting and diarrhoea may occur
- Petrol vapour may be irritating to the eyes and lungs
- Prolonged skin exposure to petrol may cause a variety of skin conditions
- Long-term exposure to high levels of petrol is associated with a range of disorders affecting the nervous system
- Petrol does not affect human reproduction or development
- There is currently no evidence that petrol causes cancer in humans

Environment

- Avoid release into the environment
- Inform the Environment Agency of substantial releases

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Petrol

General information

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Health

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- Harmful
- Inhalation may cause headache, dizziness and drowsiness.
- Often no symptoms occur following ingestion. In some cases, sickness and diarrhoea may occur
- Petrol vapour may be irritating to the eyes and lungs
- Prolonged skin exposure to petrol may cause a variety of skin conditions
- Long-term exposure to high levels of petrol is associated with a range of disorders affecting the nervous system
- Petrol does not affect human reproduction or development
- There is currently no evidence that petrol causes cancer in humans

Environment

- Avoid release into the environment
- Inform the Environment Agency of substantial releases

Background

Petrol is a complex mixture of chemicals and is manufactured by blending different products obtained from the distillation of crude oil with performance-enhancing chemicals.

'Petrol' was first used as a product name by a London chemical company (Carless, Capel & Leonard) at the end of the nineteenth century. The term 'Petrol' is an abbreviation of 'petroleum', derived from the Greek words 'petros' (meaning 'rock' or 'stone') and 'oleum' ('oil'). Petrol has also been sold as 'motor spirit', 'petroleum spirit', 'mogas' and 'gasoline' (often shortened to 'gas').



Nearly 50 thousand litres (11 thousand gallons) of petrol are used every hour in the UK.

Petrol is not particularly toxic and accidental poisoning is very rare. However, if petrol is swallowed, medical advice should be obtained immediately as there is a risk of lung damage if vomiting occurs resulting in droplets of petrol being inhaled.

Frequently Asked Questions

What is petrol?

Petrol is a complex mixture of hydrocarbons produced by mixing fractions obtained from the distillation of crude oil with brand-specific additives to improve performance. Under normal conditions, it is a volatile liquid with a characteristic odour.

What is petrol used for?

In the UK, petrol is mainly used as a fuel for light road vehicles (cars, motorbikes and small vans) and small appliances (lawnmowers, cement mixers, etc.). In smaller (“two-stroke”) engines, petrol is mixed with oil to produce a fuel mixture that reduces engine wear.

How does petrol get into the environment?

Substantial quantities of petrol are found in the environment only as a result of accidental release from an industrial site or transport vehicle. There are no natural sources of petrol. Vehicle engines may emit a very small amount of un-burnt petrol in exhaust fumes.

If there is petrol in the environment will I have any adverse health effects?

The presence of petrol in the environment does not always lead to exposure. Clearly, in order for it to cause any adverse health effects you must come into contact with it. You may be exposed by breathing, eating, or drinking the substance or by skin contact. Following exposure to any chemical, the adverse health effects you may encounter depend on several factors, including the amount to which you are exposed (dose), the way you are exposed, the duration of exposure, the form of the chemical and if you were exposed to any other chemicals.

A short, one-off exposure to petrol vapour will not normally cause any long-term health effects. Breathing large quantities of petrol vapour may cause signs of drunkenness, such as dizziness, unsteadiness and slurred speech. Drinking petrol may cause non-specific signs and symptoms of poisoning such as dizziness, headache and vomiting. A severe form of lung damage called pneumonitis (pronounced ‘new-mown-eye-tus’) may occur if liquid petrol is inhaled directly onto the lungs, for example, whilst manually siphoning a tank or from inhaling vomit after swallowing petrol. This is why it is important not to make someone sick if they have swallowed petrol and to seek immediate medical advice.

Can petrol cause cancer?

Petrol is classified by the International Agency for Research on Cancer (IARC) as being a possible carcinogen (cancer-causing chemical) mainly on the basis of long-term animal studies. However, there is not thought to be any risk of cancer from short-term, occasional exposures.

Can petrol affect or damage the unborn child?

There is no evidence to suggest that exposure of a mother to petrol may harm the unborn child. However, as with all chemicals, it is obviously best to avoid unnecessary contact.

What should I do if I am exposed to petrol?

You should remove yourself from the source of exposure.

If you have ingested petrol you should not make yourself sick. If you have ingested a small amount of petrol and you do not have any symptoms (e.g. choking, coughing, vomiting) you should be observed at home under supervision for 6 hours. If you do develop any symptoms you should attend hospital.

If you have inhaled petrol seek medical advice.

If you have got petrol on your skin, remove soiled clothing, wash the affected area with lukewarm water and soap for at least 10 – 15 minutes and seek medical advice.

If you have got petrol in your eyes, remove contact lenses, irrigate the affected eye with lukewarm water for at least 10 – 15 minutes and seek medical advice.

Petrol

Incident management

Key Points

Fire

- Highly flammable
- Vapour/air mixtures are explosive
- Low flash point
- In the event of a fire involving petrol, use normal foam and normal fire kit with breathing apparatus

Health


- Toxicity occurs if petrol is inhaled while being ingested; aspiration may cause serious lung injury
- Harmful
- Ingestion causes nausea, vomiting and abdominal pain. Systemic symptoms include drowsiness, lethargy, ataxia, convulsions, cardiac arrhythmias, coma and respiratory collapse
- Aspiration into the lungs causes pneumonitis. Signs and symptoms may progress over 24 – 48 hours
- Inhalation may cause nausea, vomiting, headache, dizziness, respiratory tract irritation, euphoria, delirium, tremor, lethargy, ataxia and drowsiness.
- Dermal exposure can cause irritation, drying, cracking, erythema and blistering. Rarely systemic toxicity may arise
- Ocular exposure may cause irritation to the eyes causing an immediate stinging and burning sensation with lacrimation

Environment

- Avoid release into the environment
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Hazard Identification

Standard (UK) Dangerous Goods Emergency Action Codes^(a)

UN	1203	Motor spirit, gasoline or petrol		
EAC	3YE	Use normal foam. Wear normal fire kit in combination with breathing apparatus*. Spillages and decontamination run-off should be prevented from entering drains and watercourses. Substance can be violently or explosively reactive. There may be a public safety hazard outside the immediate area of the incident**.		
APP	-			
Hazards	Class	3	Flammable liquid	
	Sub risks	-		
HIN	33	Highly flammable liquid (flash-point below 23°C)		

UN – United Nations number; EAC – Emergency Action Code; APP – Additional Personal Protection; HIN - Hazard Identification Number




* Normal fire fighting clothing i.e. fire kit (BS EN 469), gloves (BS EN 659) and boots (HO specification A29 and A30) in combination with self-contained open circuit positive pressure compressed air breathing apparatus (BS EN 137).

** People should stay indoors with windows and doors closed, ignition sources should be eliminated and ventilation stopped. Non-essential personnel should move at least 250 m away from the incident.

^a Dangerous Goods Emergency Action Code List, HM Fire Service Inspectorate, Publications Section, The Stationery Office, 2009.




Chemical Hazard Information and Packaging for Supply Classification^(a)

Gasoline, Natural, Low Boiling Point Naptha

Classification	Carc. Cat. 2	Category 2 Carcinogen	
	Muta. Cat 2	Category 2 Mutagen	
	Xn	Harmful	
Risk phrases	R45	May cause cancer	
	R46	May cause heritable genetic damage	
	R65	Harmful: may cause lung damage if swallowed	
Safety phrases	S45	In case of accident or if you feel unwell seek medical advice immediately (show the label where possible)	
	S53	Avoid exposure – obtain special instructions before use	

^a Annex VI to Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures- Table 3.2.
<http://esis.jrc.ec.europa.eu/index.php?PGM=cla> (accessed 11/2011)

Globally Harmonised System of Classification and Labelling of Chemicals (GHS)^{(a)}*

Hazard Class and Category	Carc. 1B	Carcinogenicity, category 1B	
	Muta. 1B	Germ cell mutagenicity, category 1B	
	Asp. Tox. 1	Aspiration hazard, category 1	
Hazard Statement	H350	May cause cancer	
	H340	May cause genetic defects	
	H304	May be fatal if swallowed and enters airways	
Signal Words	DANGER		

* Implemented in the EU on 20 January 2009.

^a Annex VI to Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures- Table 3.1.
<http://esis.jrc.ec.europa.eu/index.php?PGM=cla> (accessed 11/2011)

Physicochemical Properties

CAS number	-
Molecular weight	-
Empirical formula	Mixture of C ₄ to C ₁₂ hydrocarbons
Common synonyms	Gasoline
State at room temperature	Liquid
Volatility	Highly volatile
Specific density	Liquid: 0.7 at 16 °C (water = 1) Vapour: 3 – 4 at 20 °C (air = 1); Vapours are heavier than air at room temperature
Flammability	Highly flammable
Lower explosive limit	1.3%
Upper explosive limit	7.1%
Water solubility	Practically insoluble in water
Reactivity	Low flashpoint. Vapour/air mixtures are explosive
Reaction or degradation products	Data not available
Odour	Characteristic odour

References^(a,b)

^a WHO / UN / ILO International programme on Chemical Safety: International Chemical Safety Card 1400: Gasoline, 2001.

^b The Merck Index (14th Edition), Entry 4372: Gasoline, 2006.

Threshold Toxicity Values

EXPOSURE VIA INHALATION / INGESTION		
ppm	mg m⁻³	SIGNS AND SYMPTOMS
-	-	Data not available

Published Emergency Response Guidelines

Emergency Response Planning Guideline (ERPG) Values^(a)

	Listed value (ppm)	Calculated value (mg m ⁻³)
ERPG-1*	200 [^]	-
ERPG-2**	1000	-
ERPG-3***	4000 ^{^^}	-

* Maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to 1 hr without experiencing other than mild transient adverse health effects or perceiving a clearly defined, objectionable odour.

** Maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to 1 hr without experiencing or developing irreversible or other serious health effects or symptoms which could impair an individual's ability to take protective action.

*** Maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to 1 hr without experiencing or developing life-threatening health effects.

[^]Odour should be detectable near ERPG-1

^{^^} 10 – 49% LEL (Lower Explosive Limit of 14,000 ppm)

Acute Exposure Guideline Levels (AEGs)

	ppm				
	10 min	30 min	60 min	4 hr	8 hr
AEGL-1[†]	Data not available				
AEGL-2^{††}					
AEGL-3^{†††}					

[†] The level of the chemical in air at or above which the general population could experience notable discomfort.

^{††} The level of the chemical in air at or above which there may be irreversible or other serious long-lasting effects or impaired ability to escape.

^{†††} The level of the chemical in air at or above which the general population could experience life-threatening health effects or death.

^a American Industrial Hygiene Association (AIHA). 2010 Emergency Response Planning Guideline Values and Workplace Environmental Exposure Level Guides Handbook, Fairfax, VA. (accessed 01/2011).

Exposure Standards, Guidelines or Regulations

Occupational standards

WEL	LTEL(8 hour reference period): No guideline value specified
	STEL(15 min reference period): No guideline value specified

Public health guidelines

DRINKING WATER QUALITY GUIDELINE	No guideline value specified
AIR QUALITY GUIDELINE	No guideline value specified
SOIL GUIDELINE VALUE AND HEALTH CRITERIA VALUES	No guideline value specified

WEL – Workplace exposure limit; LTEL - Long-term exposure limit; STEL – Short-term exposure limit

Health Effects

Major routes of exposure^(a)

- Toxic via ingestion or inhalation.

Immediate signs or symptoms of acute exposure^(b,c,d,e)

- Inhalation may cause nausea, vomiting, headache, dizziness, respiratory tract irritation, euphoria, delirium, tremor, lethargy, ataxia and drowsiness. In severe cases renal impairment, non-cardiogenic pulmonary oedema, haemolytic anaemia, amnesia, coma, convulsions and cardiopulmonary arrest. There may be sudden death due to cardiac arrhythmias (in particular ventricular fibrillation). Direct inhalation of aerosols also may cause death due to bradycardia or cardiac arrest.
- Ingestion causes nausea, vomiting and abdominal pain. Rarely diarrhoea, haematemesis and melaena can occur. Aspiration into the lungs causes pneumonitis with initial choking, gasping, coughing and haemoptysis. Signs and symptoms may progress over 24 – 48 hours with wheeze, breathlessness, hyperventilation, dyspnoea, tachypnoea, bronchospasm, hypoxia, cyanosis, fever and leukocytosis. Pulmonary oedema (may be delayed for 24 – 72 hours). In severe cases shock and cardiorespiratory arrest can occur. Rare complications include pleural effusions or pneumatoceles, lipoid pneumonia, emphysema, pneumothorax and pneumomediastinum.
- Systemic symptoms include drowsiness, lethargy, ataxia, convulsions, cardiac arrhythmias, coma and respiratory collapse. In rare cases elevated LFTs, renal failure, intravascular haemolysis and disseminated intravascular coagulation may occur.
- Dermal exposure can cause irritation, drying and cracking due to defatting action. There may be transient pain with erythema, blistering necrosis, partial thickness burns and possibly full thickness burns. Rarely systemic toxicity may arise.
- Ocular exposure may cause irritation to the eyes causing an immediate stinging and burning sensation with lacrimation.

TOXBASE - <http://www.toxbase.org> (accessed 01/2011)

^a TOXBASE: Petrol, 05/2010.

^b TOXBASE: Petroleum distillates – inhalation, 03/2010.

^c TOXBASE: Petroleum distillates – features and management, 04/2010.

^d TOXBASE: Petroleum distillates – skin contact, 03/2010.

^e TOXBASE: Eye irritants, 05/2002.

Decontamination and First Aid

Important Notes

- Ambulance staff, paramedics and emergency department staff treating chemically-contaminated casualties should be equipped with Department of Health approved, gas-tight (Respirex) decontamination suits based on EN466:1995, EN12941:1998 and prEN943-1:2001, where appropriate.
- Decontamination should be performed using local protocols in designated areas such as a decontamination cubicle with adequate ventilation.
- Flammability warning: prevent exposure to all sources of ignition such as naked flames, electrical equipment, oxidising chemicals and the smoking of tobacco products.

Dermal Exposure^(a,b)

- Remove patient from exposure.
- The patient should remove all clothing and personal effects.
- Double-bag soiled clothing and place in a sealed container clearly labelled as a biohazard.
- Gently blot away any adherent liquid from the patient.
- Wash hair and all contaminated skin with copious amounts of water (preferably warm) and soap for at least 10-15 minutes. Decontaminate open wounds first and avoid contamination of unexposed skin.
- Pay special attention to skin folds, axillae, ears, fingernails, genital areas and feet.
- Burns totalling more than 15% of body surface area in adults (> 10% in children) will require standard fluid resuscitation as for thermal burns.
- Cover affected area with a clean non-adherent dressing.
- Apply other supportive measures as indicated by the patient's clinical condition.

Ocular Exposure^(c)

- Remove patient from exposure.
- Remove contact lenses if necessary and immediately irrigate the affected eye thoroughly with water or 0.9% saline for at least 10-15 minutes.
- Patients with corneal damage or those whose symptoms do not resolve rapidly should be referred for urgent ophthalmological assessment.

Inhalation^(d)

- Remove patient from exposure.
- Ensure a clear airway and adequate ventilation.
- Give oxygen to symptomatic patients.
- If the patient has clinical features of bronchospasm treat conventionally with nebulised bronchodilators and steroids. (The use of epinephrine should be avoided).

TOXBASE - <http://www.toxbase.org> (accessed 01/2011)

^a TOXBASE: Petrol, 05/2010.

^b TOXBASE: Petroleum distillates – skin contact, 03/2010.

^c TOXBASE: Eye irritants, 05/2002.

^d TOXBASE: Petroleum distillates – inhalation, 03/2010.

- Monitor pulse, blood pressure, oxygen saturation, conscious level and respiratory rate.
- Perform 12 lead ECG and monitor cardiac rhythm.
- Apply other supportive measures as indicated by the patient's clinical condition.

Ingestion^(a)

- Ensure a clear airway and adequate ventilation.
- Give oxygen to symptomatic patients.
- Gastric lavage should NOT be undertaken.
- Monitor pulse, respiratory rate, oxygen saturation, conscious level and temperature.
- Perform a 12-lead ECG and monitor cardiac rhythm in symptomatic patients.
- Treat bronchospasm conventionally with nebulised bronchodilators and steroids. (The use of epinephrine should be avoided).
- Intubation and mechanical ventilation may be required in severe cases.
- Apply other supportive measures as indicated by the patient's condition.

^a TOXBASE: Petroleum distillates – features and management, 03/2010.

Petrol

Toxicological overview

Key Points

Kinetics and metabolism

- As petrol is a mixture of chemicals, there is no definitive ADME (absorption, distribution, metabolism and excretion) data

Health effects of acute exposure

- Exposure to petrol vapour in confined or poorly ventilated areas may cause rapid onset of unconsciousness
- The main hazard associated with petrol is chemical pneumonitis that may arise following aspiration of vomitus (secondary to ingestion) or inhalation of aerosol (or aspiration of liquid) during manual siphoning
- Inhalation may cause dizziness, excitement and in-coordination
- Ingestion may cause nausea, vomiting and diarrhoea
- Petrol vapour may be irritating to the eyes and respiratory system

Health effects of chronic exposure

- Prolonged skin exposure to petrol may cause a variety of dermatitic conditions and is generally a result of inadequate or inappropriate use of personal protective equipment
- Chronic exposure to high levels (particularly arising from recreational inhalation) is associated with a range of neurological disorders
- Petrol does not have a measurable effect on human reproduction or development
- There is currently no unequivocal evidence to link petrol with the incidence of cancer in humans but there is limited evidence for carcinogenicity in animals

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Toxicological Overview

Petrol is a complex mixture of aliphatic and aromatic hydrocarbons derived from blending fractions of crude oil with brand-specific additives. The actual composition of petrol will vary according to the source of crude oil, the manufacturing process and between batches. A representative composition of European petrol is given at Annex I.

Unless otherwise stated, this document pertains to unleaded petrol and not products of combustion or individual chemical components (e.g. benzene, toluene, xylene, butadiene, etc). Vapour concentrations are expressed as ppm and refer to total hydrocarbon present. However, it should be noted that this conventional measure of concentration introduces a source of error and should be considered at best an approximation, as the average molecular weight (on which the calculation of ppm is based) may vary according to temperature, brand or batch of technical product.

Summary of Health Effects

At low doses, petrol vapour is irritating to the eyes, respiratory tract and skin. Exposure to higher concentrations of vapour may produce CNS effects such as staggered gait, slurred speech and confusion. Very high concentrations may result in rapid unconsciousness and death due to respiratory failure [1].

Prolonged dermal exposure to liquid petrol or inhalation of vapour has been associated with renal dysfunction, attributed to lipid degeneration of the proximal convoluted tubules and glomeruli [2, 3], the clinical manifestations of which include haematuria, proteinuria and myoglobinuria. A late-onset autoimmune glomerulonephritis has also been described [4].

Pulmonary sequelae following inhalation of petrol vapour or secondary to pulmonary elimination of volatile hydrocarbons (following ingestion or dermal absorption) include persistent atelectasis [2] and petachial haemorrhage [1]. This may be associated with concomitant 'hydrocarbon hepatitis' secondary to vascular endothelial damage, possibly due to hydrocarbon-induced degeneration of fatty tissue [4-6].

The critical health effect of petrol is chemical pneumonitis, arising from aspiration of liquid petrol or inhalation of petrol-contaminated vomitus [7].

Kinetics and metabolism

Petrol is a complex mixture of hydrocarbons and so there is no definitive ADME data available for animals or humans. The onset of local or systemic effects following dermal, oral and inhalation exposure indicates that these are all potential routes of entry for petrol vapour or liquid.

Dermal exposure to petrol can be retrospectively identified following solvent extraction of hydrocarbons from the skin surface. However, the profile of hydrocarbons found in the systemic circulation after cutaneous exposure is markedly different to that extracted from the skin [8], indicating selective uptake and distribution of individual hydrocarbon components.

There is some evidence to suggest that petrol exposure may alter hepatic enzyme activity in rats and humans [9, 10], although the clinical relevance of such observations has not been established.

The predominant route of elimination for volatile components of petrol is considered to be via expired air. This assumption is based on clinical observation; there are no experimental studies to confirm this route of elimination in humans.

Sources and route of exposure

Petrol contains mixture of volatile hydrocarbons and so inhalation is the most common form of exposure [11]. Petrol vapour can reach supra-lethal concentrations in confined or poorly ventilated areas, although such exposures are rare [12-14]. A representative sample of petrol vapour concentrations under different exposure scenarios are summarised in Table 1. It should be noted that the chemical composition (hydrocarbon profile) of petrol vapour differs substantially from the corresponding liquid (Annex I). Petrol vapour is predominantly (>70%) composed of light (C₄ & C₅) hydrocarbons [15] whereas liquid petrol contains mainly (>80%) C₆₋₁₂ compounds [16]. The intentional inhalation of vapour ('sniffing' or 'huffing') has been extensively documented [17-22].

Table 1: Representative vapour concentrations, expressed in parts per million (ppm) or mass of total hydrocarbons per unit volume (mg m⁻³) under different conditions.

Vapour concentration (ppm)	Scenario	Notes	Ref
25,000	Air above open barrel in unventilated out-house on 'hot' day.	Environmental conditions not reported.	[12]
5 – 320	Air around tanker during bulk-loading.	Environmental conditions not reported.	[23]
2 – 100	Air around petrol pump in service station.		[24]
1 – 5	Air within petrol service station.	Temperature varied from 4.5 – 25°C. Recovered petrol components were predominantly (72%) C ₄ and C ₅ aliphatic hydrocarbons.	[25]
174	Worker at bulk loading facility.	Average exposure values. Environmental conditions not reported. Vapour concentration reported as the sum of all detected hydrocarbon constituents.	[16]
13	Road tanker driver.		
4	Service station worker.		

It is generally considered that dermal absorption of petrol does not contribute significantly to systemic signs of toxicity.

Accidental ingestion of petrol by adults is often the result of siphoning petrol tanks whereas the incidence of petrol ingestion in children is relatively low [7, 12].

Health Effects of Acute / Single Exposure

Human Data

General toxicity

The acute health risks involved in handling and using petrol are minimal, provided that the product(s) are used in accordance with appropriate health and safety practices [26].

The main health effect associated with petrol exposure is chemical pneumonitis, resulting from pulmonary aspiration of vomitus following ingestion [27]. A rare complication of petrol intoxication may be cardiac arrhythmia and ventricular fibrillation, attributed to increased myocardial sensitivity to endogenous catecholamines [28].

Vascular endothelial damage of the lung, liver, kidney and spleen has been described following severe intoxication with associated renal lipid degeneration confined mainly to the proximal tubules [1, 6].

Inhalation

Petrol vapour is readily detectable by most individuals at concentrations below 1 to 2 ppm [29] (as reviewed by [7]) although prior exposure within 24 hours or chronic, occupational exposure may increase the olfactory threshold [30].

It has been suggested that the concentration of petrol vapour is the primary determinant of acute toxicity rather than duration of exposure [31]. This assumption is based on exposures of less than 30 minutes and relates to one study of three dogs dosed with different petrol products [32].

The minimum concentration of petrol vapour required to elicit a mild response (cough) is probably less than 140 ppm (Table 2), although there are no human studies which have determined the exact threshold for this effect.

Following inhalation, effects on the CNS are readily apparent above 900 ppm within a few minutes, the morbidity of which resembles alcohol intoxication (dizziness, excitement, incoordination, etc.).

In sufficient concentration (>10,000 ppm), petrol may act as an anaesthetic, sometimes resulting in immediate loss of consciousness [1]. Indeed, the rapidity of this effect has been implicated as a significant factor in fatal incidents [12-14, 31].

Ingestion

Ingestion of petrol may cause acute, generalised signs of GI tract irritation, including nausea, vomiting, colic and diarrhoea [7]. The relatively low oral toxicity of petrol is comparable to that of other petrochemical products such as kerosene (2 – 17 g kg⁻¹) [33] and ingestion of 7.5 g kg⁻¹ has been reported to be the lethal dose in adult humans in the absence of pulmonary effects caused by aspiration of ingested petrol or vomitus [1].

A critical, delayed health effect associated with ingestion of petrol is chemical pneumonitis, resulting from aspiration of petrol during the swallowing process or of vomitus following emesis [34].

Table 2: Summary of the human inhalation toxicity of petrol vapour. *Refers to estimated concentrations based on post-incident measurements: See also Annex II.

Study type	Concentration (ppm)	Duration	Temperature	Effect(s)	Ref
Human volunteer studies	140 – 270	8 h	23 °C	Mild irritation (coughing, sore throat), conjunctival hyperaemia.	[30]
	200	0.5 h		Threshold level for eye irritation.	[35]
	900	8 h	22 °C	Mild CNS effects (dizziness). Tolerable.	[30]
	2,600	1 h	n/s	Onset of neuromuscular effects (incoordination)	[30]
	> 10,700	< 5 min	n/s	Rapid onset of dizziness and 'drunkenness' (ataxia, confusion). Threshold level for onset of anaesthetic effects.	[30]
Case studies	8,000 – 35,000*	Minutes	'hot'	Death occurred sometime within 45 minutes of initial exposure.	[12]
	5,000 – 16,000*	Minutes	n/s	Death occurred sometime within five minutes of exposure.	[31]

Dermal / ocular exposure

Mild ocular irritation may follow prolonged (8 h) exposure to low concentrations (140 ppm) of petrol vapour [30] or shorter (30 minute) exposure to vapours above 200 ppm [35]. More pronounced signs of ocular toxicity (lacrimation) occur above concentrations of 1000 ppm [35]. One study attributed ocular irritation to the presence of hydrocarbon component(s) present in the most volatile fraction of petrol: “The substance causing eye irritation was distilled from gasoline at extremely low temperatures and in very small amounts” [30].

Skin lesions resulting from acute exposure to liquid petrol are rare and generally result only from prolonged contact (hours) with undiluted product [5]. Thus, the extent of skin injury is primarily related to the duration of the exposure rather than concentration. Petrol “burns” resemble scalding, with an initial erythema leading to blister formation [5]. Prolonged dermal exposure (45 minutes – 12 hours) may lead to partial or full thickness burns with associated loss of epidermis and coagulation necrosis [3, 6]. Discolouration of the skin (brown / yellow / gold) has been observed and attributed to dye additives.

Dermal exposure to petrol is not considered to be a major factor in systemic toxicity [1]: this is based upon the assumption that skin contamination will occur concomitantly to inhalation of petrol vapour (which is considered the predominant route of entry). However, several clinical reports have suggested that dermal exposure may substantially contribute to systemic toxicity [2, 6] and it has been recommended that debridement of contaminated skin

may limit continued systemic absorption in cases where prolonged dermal exposure has occurred [2]. Systemic toxicity resulting from dermal exposure has been noted with other hydrocarbon mixtures such as diesel [36].

Neurotoxicity

As with other hydrocarbon solvents, petrol has anaesthetic (narcotic) properties (Table 2). Petrol also contains a number of potentially neurotoxic chemicals including n-hexane, benzene, butadiene, toluene, ethylbenzene, xylene and trimethyl pentane [37]. The approximate concentration of each constituent in liquid petrol and vapour are given in Table 3.

Table 3: Average concentration of potentially neurotoxic constituents of liquid petrol and vapour [11, 38]. Numbers in brackets refer to range of values. Vapour values expressed as percentage of total hydrocarbons recovered from air samples obtained during the manual filling of cars (conditions and duration not reported).

Chemical	Concentration (%w/w)	
	Liquid	Vapour
Benzene	2.5 (0.2 – 4.7)	1.77 (0 – 5.4)
1,3-Butadiene	<0.1	0.65 (0 – 4.6)
Ethylbenzene	2.6 (1.0 – 5.4)	.009 (0 – 0.1)
n-Hexane	2.5 (0.8 – 5)	1.37 (0 – 6.5)
Toluene	11.4 (2.7 – 21.0)	1.63 (0 – 7.1)
Xylene	10.6 (5.8 – 15.8)	0.48 (0 – 2.1)

Historically, lead (as tetraethyl lead; TEL) has been identified as the principal component of petrol responsible for neurological deficits following intentional (recreational) inhalation or massive acute exposure. However, European legislation has prohibited the use of TEL in petrol since January 2000 [39]. Therefore, there is currently a paucity of human data pertaining to the acute neurological effects of current (unleaded) petrol products other than narcosis.

Delayed effects following an acute exposure

A variety of delayed, neurological deficits have historically been associated with the acute inhalation of petrol vapour, including peripheral neuritis, impairment of memory, paresthesia, ataxis and epilepsy [1]. A late-onset autoimmune glomerulonephritis has also been observed following acute exposure [4].

There is limited evidence to suggest that long-term pulmonary residual effects may occur following chemical pneumonitis (as a result of aspiration-induced pneumonitis) [27, 40], the effects of which are of unknown clinical relevance [41].

Animal and In-Vitro Data

General toxicity

The acute toxicity of petrol in a variety of animal species is broadly consistent with that reported in humans, being predominantly associated with CNS, pulmonary and renal effects [42]. Data on LD₅₀ and skin and eye irritation are shown in Table 4.

Table 4: Acute toxicity data for petrol [43].

Test	Species	Result
Acute oral (LD ₅₀)	Rat	13.6 g kg ⁻¹
Sensitisation	Guinea pig	Not sensitising
Primary dermal irritancy	Rabbit	slight
Acute dermal	Rabbit	No mortalities
Primary eye	Rabbit	Non irritating

Health Effects of Chronic / Repeated Exposure

Human Data

General toxicity

Dysfunction of the central nervous system is the predominant pathological condition associated with chronic exposure to high levels and such effects arising from frequent, recreational exposure ('sniffing' or 'huffing') have been extensively documented [17-22, 44-49]. There is currently insufficient evidence to unequivocally link chronic (occupational) exposure to petrol with other pathological conditions [50]. This may be because petrochemical workers are potentially exposed to a wide range of chemicals in addition to other confounding factors [42].

Historically, lead has been identified as the principal component of petrol responsible for neurotoxicity [51] and studies have demonstrated a link between lead body burden and neurological deficits as a result of petrol abuse ('sniffing' or 'huffing') [52]. However, it should be noted that the volatility of tetraethyl lead (TEL) is relatively low (0.4 mm Hg at 25°C) and so prolonged dermal exposure associated with the practice of petrol sniffing is likely to be the predominant route of entry for TEL rather than inhalation [7]. Since 2000, petrol has only been commercially available in 'unleaded' form within the UK and most of Europe. This policy limits the concentration of lead in marketable petrol to less than 0.005 g L⁻¹ as defined at Annex I of the 1998 EU Directive [39].

Whilst there is a known association between chronic petrol exposure and renal cancer in male rats [53-55], there is currently no evidence to link petrol exposure and renal cancer in humans [56, 57]. It is generally accepted that the susceptibility of male rats is mediated via a specific protein (α -2-microglobin) which is absent in other mammals [54, 58].

Genotoxicity

At relatively high concentrations (1000 – 2500 ppm) in cell culture medium, petrol was mutagenic in *Drosophila melanogaster* [59].

Negative results have been reported when petrol was investigated for its ability to induce gene mutations in bacteria (*Salmonella* assay) and mammalian cells using the mouse lymphoma assay [60]. Negative results were also obtained in another mammalian cell assay for gene mutation using a human lymphoblastoid cell line [61]. Negative results were reported in an *in vivo* bone marrow assay for clastinogenicity in the rat [60].

There is a report of positive results being obtained using the UDS (unscheduled DNA synthesis) assay: limited studies *in vitro* (essentially only one dose level used) gave a positive result using rat hepatocytes and marginal effect in human and mouse hepatocytes. In the same report, negative results were obtained from *in vivo* UDS assays in the rat, but a slight increase in UDS was observed in mice [62].

Overall, it can be concluded that petrol does not have significant mutagenic activity.

Carcinogenicity

Epidemiological studies have not demonstrated a statistically significant link between cancer and occupational exposure to petrol [63-71]. However, the IARC has classified petrol as “possibly carcinogenic to humans” (Group 2B) mainly on the basis that there was inadequate evidence for the carcinogenicity in humans but there was limited evidence for the carcinogenicity in experimental animals. It was also noted that certain components of petrol are known or possible human carcinogens such as benzene and 1,3-butadiene. [57].

Petrol is assigned the Risk Phrase R45 (“may cause cancer”) under the Chemical Hazard Information and Packaging for Supply (CHIPS) Regulations.

Reproductive and developmental toxicity

No reports specifically pertaining to the human reproductive or developmental toxicity of petrol were identified. The NOAEL for reproductive toxicity in a two-generation rat study was reported to be 20 000 mg m⁻³ [72].

Petrol is not classified under CHIPS (Chemical Hazards Information and Packaging Supply) regulations as a reproductive or developmental hazard.

References

- [1] Machle, W. (1941). gasoline intoxication. *Journal of the American Medical Association* **117**, 1965-1971.
- [2] Hansbrough, J. F., Zapata-Sirvent, R., Dominic, W., Sullivan, J., Boswick, J. and Wang, X. W. (1985). Hydrocarbon contact injuries. *J Trauma* **25**, 250-2.
- [3] Schneider, M. S., Mani, M. M. and Masters, F. W. (1991). Gasoline-induced contact burns. *J Burn Care Rehabil* **12**, 140-3.
- [4] Simpson, L. A. and Cruse, C. W. (1981). Gasoline immersion injury. *Plast Reconstr Surg* **67**, 54-7.
- [5] Binns, H., Gursel, E. and Wilson, N. (1978). Gasoline contact burns. *Jacep* **7**, 404-5.
- [6] Walsh, W. A., Scarpa, F. J., Brown, R. S., Ashcraft, K. W., Green, V. A., Holder, T. M. and Amoury, R. A. (1974). Gasoline immersion burn. *N Engl J Med* **291**, 830.
- [7] Toxicology update. Gasoline. (1989). *J Appl Toxicol* **9**, 203-10.
- [8] Hieda, Y., Tsujino, Y. and Takeshita, H. (2005). Skin analysis to determine causative agent in dermal exposure to petroleum products. *J Chromatogr B Analyt Technol Biomed Life Sci* **823**, 53-9.
- [9] Harman, A. W., Frewin, D. B. and Priestly, B. G. (1981). Induction of microsomal drug metabolism in man and in the rat by exposure to petroleum. *Br J Ind Med* **38**, 91-7.
- [10] Rao, G. S. and Pandya, K. P. (1980). Hepatic metabolism of heme in rats after exposure to benzene, gasoline and kerosene. *Arch Toxicol* **46**, 313-7.
- [11] Cecil, R., Ellison, R. J., Larnimaa, K., Margary, S. A., Mata, J. M., Morcillo, L., Muller, J.-M., Peterson, D. R. and Short, D. (1997). exposure profile: gasoline. *CONCAWE Report* **97/52**.
- [12] Aiden, R. (1958). petrol-vapour poisoning. *British Medical Journal* **ii**, 369-370.
- [13] Ainsworth, R. (1960). petrol-vapour poisoning. *British Medical Journal* **1**, 1547-1548.
- [14] Takamiya, M., Niitsu, H., Saigusa, K., Kanetake, J. and Aoki, Y. (2003). A case of acute gasoline intoxication at the scene of washing a petrol tank. *Leg Med (Tokyo)* **5**, 165-9.
- [15] Halder, C. A., Van Gorp, G. S., Hatoum, N. S. and Warne, T. M. (1986). Gasoline vapor exposures. Part II. Evaluation of the nephrotoxicity of the major C4/C5 hydrocarbon components. *Am Ind Hyg Assoc J* **47**, 173-5.
- [16] Carter, M., Claydon, M., Giacometti, D., Money, C., Pizzella, G., Margary, A. and Viinanen, R. (2002). A survey of european gasoline exposures for the period 1999 - 2001. *CONCAWE Report* **9/02**.
- [17] Edminster, S. C. and Bayer, M. J. (1985). Recreational gasoline sniffing: acute gasoline intoxication and latent organolead poisoning. Case reports and literature review. *J Emerg Med* **3**, 365-70.

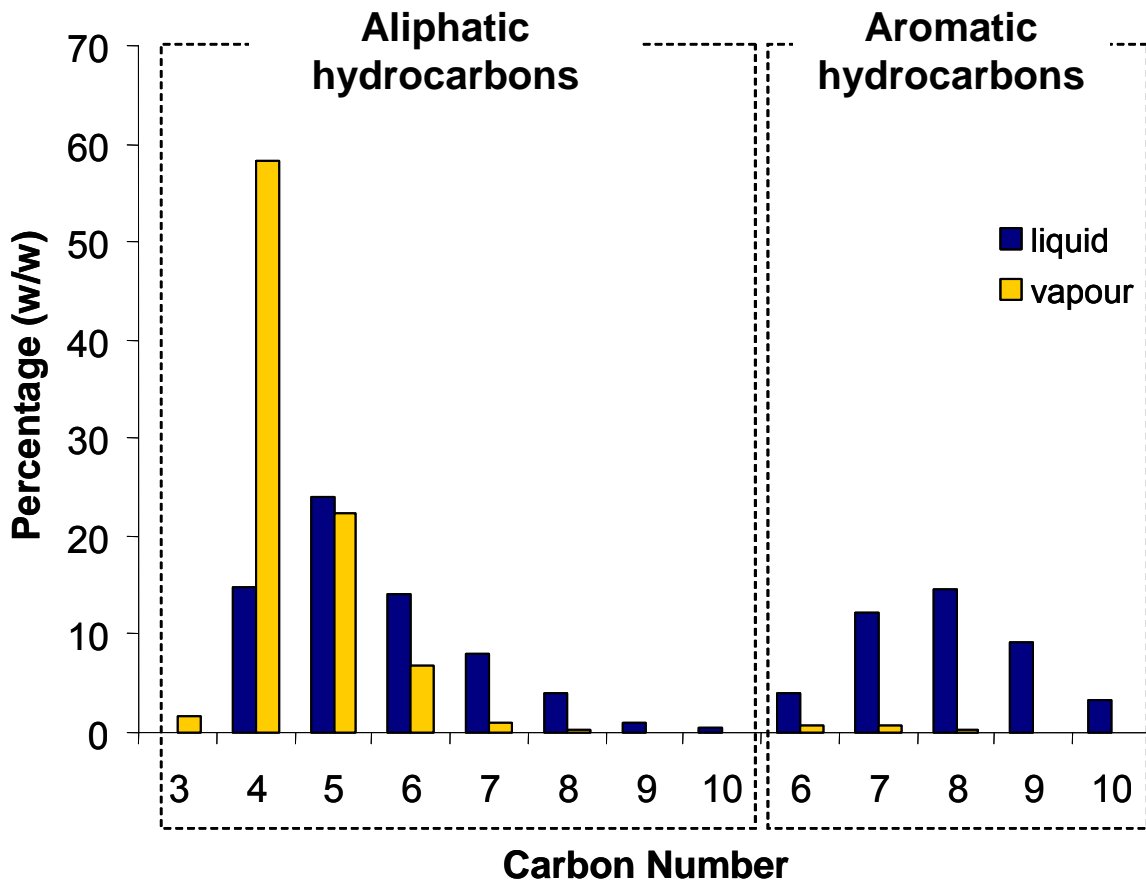
- [18] Cairney, S., Maruff, P., Burns, C. and Currie, B. (2002). The neurobehavioural consequences of petrol (gasoline) sniffing. *Neurosci Biobehav Rev* **26**, 81-9.
- [19] Flanagan, R. J. and Ives, R. J. (1994). Volatile substance abuse. *Bull Narc* **46**, 49-78.
- [20] Fortenberry, J. D. (1985). Gasoline sniffing. *Am J Med* **79**, 740-4.
- [21] Remington, G. and Hoffman, B. F. (1984). Gas sniffing as a form of substance abuse. *Can J Psychiatry* **29**, 31-5.
- [22] Poklis, A. and Burkett, C. D. (1977). Gasoline sniffing: a review. *Clin Toxicol* **11**, 35-41.
- [23] Periago, J. F. and Prado, C. (2005). Evolution of occupational exposure to environmental levels of aromatic hydrocarbons in service stations. *Ann Occup Hyg* **49**, 233-40.
- [24] McDermott, H. J. and Vos, G. A. (1979). Service station attendants' exposure to benzene and gasoline vapors. *Am Ind Hyg Assoc J* **40**, 315-21.
- [25] Kearney, C. A. and Dunham, D. B. (1986). Gasoline vapor exposures at a high volume service station. *Am Ind Hyg Assoc J* **47**, 535-9.
- [26] Henry, J. A. (1998). Composition and toxicity of petroleum products and their additives. *Hum Exp Toxicol* **17**, 111-23.
- [27] Litovitz, T. and Greene, A. E. (1988). Health implications of petroleum distillate ingestion. *Occup Med* **3**, 555-68.
- [28] Litovitz, T. (1988). Myocardial sensitization following inhalation abuse of hydrocarbons. *Occup Med* **3**, 567-568.
- [29] Amooore, J., Von Burg, R. and Whittemore, I. (1983). Detectibility of Gasoline by its Odor. The Toxicologist. 22nd Annual Meeting of the Society of Toxicologists. March 6-11.
- [30] Drinker, P., Yaglou, C. P. and Warren, M. F. (1943). The threshold toxicity of gasoline vapour. *J Ind Hyg Toxicol* **25**, 225-232.
- [31] Wang, C. C. and Irons, G. V., Jr. (1961). Acute gasoline intoxication. *Arch Environ Health* **2**, 714-6.
- [32] Haggard, H. W. (1920). the anaesthetic and convulsant effects of gasoline vapour. *J Pharmacol Exp Ther* **16**, 401-404.
- [33] Chilcott, R. P. (2006). Kerosene: toxicological overview. *Health Protection Agency compendium of chemical toxicity*.
- [34] Banner, W., Jr. and Walson, P. D. (1983). Systemic toxicity following gasoline aspiration. *Am J Emerg Med* **1**, 292-4.
- [35] Davis, A., Schafer, L. J. and Bell, Z. G. (1960). The effects on human volunteers of exposure to air containing gasoline vapor. *Arch Environ Health* **1**, 548-54.
- [36] Barrientos, A., Ortuno, M. T., Morales, J. M., Tello, F. M. and Rodicio, J. L. (1977). Acute renal failure after use of diesel fuel as shampoo. *Arch Intern Med* **137**, 1217.

- [37] Ritchie, G. D., Still, K. R., Alexander, W. K., Nordholm, A. F., Wilson, C. L., Rossi, J., 3rd and Mattie, D. R. (2001). A review of the neurotoxicity risk of selected hydrocarbon fuels. *J Toxicol Environ Health B Crit Rev* **4**, 223-312.
- [38] Coker, D. T., Christian, F., Claydon, M. F., Irvine, D., Portail, C., Tindle, P., Webb, C. L. F. and Eyres, A. R. (1987). A Survey of exposures to gasoline vapour. *CONCAWE Report* **4/87**.
- [39] EU (1998). Directive 98/70/EC of the European parliament and of the Council: Amendment to Council Directive 93/12/EEC. *Official Journal of the European Communities* **350/59**.
- [40] Eade, N. R., Taussig, L. M. and Marks, M. I. (1974). Hydrocarbon pneumonitis. *Pediatrics* **54**, 351-7.
- [41] Seymour, F. K. and Henry, J. A. (2001). Assessment and management of acute poisoning by petroleum products. *Hum Exp Toxicol* **20**, 551-62.
- [42] International Agency for the Research on Cancer (IARC) (1989). Occupational exposures in petroleum refining: Crude oil and major petroleum fuels. IARC Monographs on the evaluation of carcinogenic risks to humans. Volume 45. Lyon.
- [43] Beck, L. S., Hepler, D. I. and Hansen, K. L. (1984) The acute toxicity of selected hydrocarbons. Princeton Scientific Publishers, Inc.,
- [44] Cairney, S., Maruff, P., Burns, C. B., Currie, J. and Currie, B. J. (2004). Neurological and cognitive impairment associated with leaded gasoline encephalopathy. *Drug Alcohol Depend* **73**, 183-8.
- [45] Cairney, S., Maruff, P., Burns, C. B., Currie, J. and Currie, B. J. (2005). Neurological and cognitive recovery following abstinence from petrol sniffing. *Neuropsychopharmacology* **30**, 1019-27.
- [46] Goodheart, R. S. and Dunne, J. W. (1994). Petrol sniffer's encephalopathy. A study of 25 patients. *Med J Aust* **160**, 178-81.
- [47] Maruff, P., Burns, C. B., Tyler, P., Currie, B. J. and Currie, J. (1998). Neurological and cognitive abnormalities associated with chronic petrol sniffing. *Brain* **121 (Pt 10)**, 1903-17.
- [48] Poklis, A. (1976). Death resulting from gasoline "sniffing": a case report. *J Forensic Sci Soc* **16**, 43-6.
- [49] Valpey, R., Sumi, S. M., Copass, M. K. and Goble, G. J. (1978). Acute and chronic progressive encephalopathy due to gasoline sniffing. *Neurology* **28**, 507-10.
- [50] Harrington, J. M. (1987). The health experience of workers in the petroleum manufacturing and distribution industry. *CONCAWE report number 2/87*.
- [51] Kovarik, W. (2005). Ethyl-leaded gasoline: how a classic occupational disease became an international public health disaster. *Int J Occup Environ Health* **11**, 384-97.
- [52] Seshia, S. S., Rjani, K. R., Boeckx, R. L. and Chow, P. N. (1978). The neurological manifestations of chronic inhalation of leaded gasoline. *Dev Med Child Neurol* **20**, 323-34.

- [53] Halder, C. A., Holdsworth, C. E., Cockrell, B. Y. and Piccirillo, V. J. (1985). Hydrocarbon nephropathy in male rats: identification of the nephrotoxic components of unleaded gasoline. *Toxicol Ind Health* **1**, 67-87.
- [54] Olson, M. J., Garg, B. D., Murty, C. V. and Roy, A. K. (1987). Accumulation of alpha 2u-globulin in the renal proximal tubules of male rats exposed to unleaded gasoline. *Toxicol Appl Pharmacol* **90**, 43-51.
- [55] Short, B. G., Burnett, V. L., Cox, M. G., Bus, J. S. and Swenberg, J. A. (1987). Site-specific renal cytotoxicity and cell proliferation in male rats exposed to petroleum hydrocarbons. *Lab Invest* **57**, 564-77.
- [56] Trump, B. F., Lipsky, M. M., Jones, T. W., Heatfield, B. M., Higginson, J., Endicott, K. and Hess, H. B. (1984) An Evaluation of the significance of experimental hydrocarbon toxicity in man. Princeton Scientific Publishers Inc., New Jersey
- [57] IARC (1989). Occupational Exposure in Petroleum Refining; Crude Oil and Major Petroleum Fuels. *IARC Monogr Eval Carcinog Risks Hum* **45**.
- [58] Olson, M. J., Johnson, J. T. and Reidy, C. A. (1990). A comparison of male rat and human urinary proteins: implications for human resistance to hyaline droplet nephropathy. *Toxicol Appl Pharmacol* **102**, 524-36.
- [59] Nylander, P. O., Olofsson, H., Rasmuson, B. and Svahlin, H. (1978). Mutagenic effects of petrol in *Drosophila melanogaster* I. Effects of benzene and 1,2-dichloroethane. *Mutat Res* **57**, 163-7.
- [60] Conaway, C. C., Schreiner, C. A. and Cragg, S. T. (1984) Mutagenicity evaluation of petroleum hydrocarbons. Princeton Scientific Publishers Inc, New Jersey
- [61] Richardson, K. A., Wilmer, J. L., Smith-Simpson, D. and Skopek, T. R. (1986). Assessment of the genotoxic potential of unleaded gasoline and 2,2,4-trimethylpentane in human lymphoblasts in vitro. *Toxicol Appl Pharmacol* **82**, 316-22.
- [62] Loury, D. J., Smith-Oliver, T., Strom, S., Jirtle, R., Michalopoulos, G. and Butterworth, B. E. (1986). Assessment of unscheduled and replicative DNA synthesis in hepatocytes treated in vivo and in vitro with unleaded gasoline or 2,2,4-trimethylpentane. *Toxicol Appl Pharmacol* **85**, 11-23.
- [63] Dement, J. M., Hensley, L. and Gitelman, A. (1997). Carcinogenicity of gasoline: a review of epidemiological evidence. *Ann N Y Acad Sci* **837**, 53-76.
- [64] Enterline, P. E. (1993). Review of new evidence regarding the relationship of gasoline exposure to kidney cancer and leukemia. *Environ Health Perspect* **101 Suppl 6**, 101-3.
- [65] Enterline, P. E. and Viren, J. (1985). Epidemiologic evidence for an association between gasoline and kidney cancer. *Environ Health Perspect* **62**, 303-12.
- [66] McLaughlin, J. K. (1993). Renal cell cancer and exposure to gasoline: a review. *Environ Health Perspect* **101 Suppl 6**, 111-4.
- [67] McLaughlin, J. K., Blot, W. J., Mandel, J. S., Schuman, L. M., Mehl, E. S. and Fraumeni, J. F., Jr. (1983). Etiology of cancer of the renal pelvis. *J Natl Cancer Inst* **71**, 287-91.

- [68] McLaughlin, J. K., Blot, W. J., Mehl, E. S., Stewart, P. A., Venable, F. S. and Fraumeni, J. F., Jr. (1985). Petroleum-related employment and renal cell cancer. *J Occup Med* **27**, 672-4.
- [69] McLaughlin, J. K., Mandel, J. S., Blot, W. J., Schuman, L. M., Mehl, E. S. and Fraumeni, J. F., Jr. (1984). A population--based case--control study of renal cell carcinoma. *J Natl Cancer Inst* **72**, 275-84.
- [70] Norell, S., Ahlbom, A., Olin, R., Erwald, R., Jacobson, G., Lindberg-Navier, I. and Wiechel, K. L. (1986). Occupational factors and pancreatic cancer. *Br J Ind Med* **43**, 775-8.
- [71] Rushton, L. and Alderson, M. R. (1983). Epidemiological survey of oil distribution centres in Britain. *Br J Ind Med* **40**, 330-9.
- [72] McKee, R. H., Dally, S., Dmytrasz, B. A., Gonnet, J. F., Hagemann, R. E., Mackerer, C. R., Nessel, C. S., Priston, R. A. and Riley, A. J. (2000). An assessment of the reproductive toxicity of gasoline vapour. CONCAWE Report number 00/53. Brussels.

Annex I: Average composition of (European) petrol liquid and vapour.



This document will be reviewed no later than 3 years or sooner if substantive evidence becomes available.