DEPARTMENT OF ENVIRONMENTAL AFFAIRS AND TOURISM

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IMPACTS OF AIR POLLUTION

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ACRONYMS

APPA	Atmospheric Pollution Prevention Act (No. 45 of 1965)
AQA	National Environmental Management: Air Quality Act (No. 39 of 2004)
ATSDR	Agency for Toxic Substances and Disease Registry (USA)
CCINFO	Canadian Centre for Occupational Health and Safety Information Systems
CFCs	Chlorofluorocarbons
CH_4	Methane
CO	Carbon monoxide
CO_2	Carbon dioxide
GHG	Greenhouse gas
IRIS	Integrated Risk Information System (USA EPA)
NO	Nitric oxide
NO_2	Nitrogen dioxide
NO_x	Oxides of nitrogen
O_3	Ozone
PAN	PeroxyacetylNitrate
Pb	Lead
PM	Particulate matter
$PM_{2.5}$	Particulate matter smaller than or equal to 2.5µm in diameter
PM_{10}	Particulate matter smaller than or equal to 10µm in diameter
Ppb	Parts per billion
Ppm	Parts per million
SO_2	Sulphur dioxide
TSP	Total suspended particulates
US-EPA	United States Environmental Protection Agency
VOCs	Volatile organic compounds
WHO	World Health Organisation

GLOSSARY OF TERMS

Alveolar membranes
Alveolar membranes are thin membranes which are comprised of simple squamous epithelium cells. They form part of the respiratory membrane (alveolar membrane + endothelial membrane + fused basement membranes). The oxygen concentration within each air sac or alveolus is high, so oxygen diffuses across the alveolar membrane into the pulmonary capillary.

Ambient air
Considered to be the air in the environment excluding indoor air.

Anthropogenic sources
Pollution sources that are related to human activities.

Bronchoalveolar Lavage
Washing out of the lungs with saline (sodium chloride solution) for diagnostic or therapeutic purposes

Capillary membranes
A thin layer of tissue lining the minute blood vessels

Carcinogenic
A carcinogen is a substance which can cause cancer. Carcinogenic means able to cause cancer. Carcinogenicity is the ability of a substance to cause cancer.

Distal Lung
There are four distal lung regions. These include the small airway inner and outer wall, alveolar attachments, and peripheral alveolar tissue.

Dry Deposition
The process by which atmospheric gases and particles are transferred to the earth's surface as a result of random turbulent air motions or any means that do not involve precipitation. This includes absorption, impaction, sedimentation and chemical reaction. Dry deposition rates are often drastically different than wet deposition rates.

Emission
Pollution discharged into the atmosphere from a range of stationary and mobile sources. These include smokestacks, vents and surface areas of commercial or industrial facilities; residential sources; motor vehicles and other transport related sources.

Environment
The surroundings within which humans exist and that are made up of (i) the land, water and atmosphere of the earth; (ii) micro-organisms, plant and animal life; (iii) any part or combination of (i) and (ii) and the interrelationships among and between them; and (iv) the physical, chemical, aesthetic and cultural properties and conditions of the foregoing that influence human health and well-being (definition from the National Environmental Management Act - NEMA)

Epidemiology
The scientific study of epidemics and epidemic diseases, especially the factors that influence the incidence, distribution, and control of infectious diseases; the study of disease occurrence in human populations.

Epithelial Cells
Epithelium is a tissue composed of a layer of cells. Epithelium lines both the outside (skin) and the inside (e.g. intestine) of organisms. The outermost layer of our skin is composed of dead squamous epithelial cells, as are the mucous membranes lining the inside of mouths and body cavities. Other epithelial cells line the insides of the lungs, the gastrointestinal tract, the reproductive and urinary tracts, and make up the exocrine and endocrine glands. Functions of epithelial cells include secretion, absorption, protection, transcellular transport, sensation detection, and selective permeability.

Gastrointestinal Tract
The part of the digestive tract where the body processes food and eliminates waste. It includes the oesophagus, stomach, liver, intestines, and rectum.

Greenhouse Gas
Any gas that absorbs infra-red radiation in the atmosphere. Greenhouse gases include water vapour, carbon dioxide (CO_2), methane (CH_4), nitrous oxide (N_2O), halogenated fluorocarbons (HCFCs), ozone (O_3), perfluorinated carbons (PFCs), and hydrofluorocarbons (HFCs).

Haemoglobin
A protein found in the red blood cells that is responsible for carrying oxygen around the body. Haemoglobin picks up the oxygen in the lungs, and then releases it in the muscles and other tissues where it is needed. When it has absorbed oxygen in the lungs, it is bright red and called oxy-haemoglobin. After it has given up its oxygen in the tissues, it is purple in colour and is called reduced haemoglobin. Haemoglobin also contains iron which is critical for it to work properly. The haemaglobin content of the blood can be measured and is used to assess anaemia in kidney failure.

Haematological
Haematology is the branch of medicine that deals with diseases of the blood and blood-forming organs. Haematological is of or relating to or involved in haematology

Hypoxia
Hypoxia is a pathological condition in which the body as a whole (generalized hypoxia) or region of the body (tissue hypoxia) is deprived of adequate oxygen supply.

Immunological
Immunology is a broad branch of biomedical science that covers study of all aspects of the immune system in all organisms. Immunological is anything that pertains to the body's natural defences or immunity against disease. For example, immunological factors for pregnancy loss include antiphospholipid antibodies, lupus anticoagulant, antinuclear antibodies, and antithyroid antibodies.

Lipid Peroxidases
An enzyme that catalyses reduction of hydrogen peroxides by a substrate that loses two hydrogen atoms.

Macrophages
Macrophages are differentiated from monocytes, which are eater cells derived from the bone marrow. When a monocyte enters the attacked tissue through the endothelium of a blood vessel, it undergoes a series of changes and becomes a macrophage. The attraction of wandering macrophages to a damaged site occurs through chemotaxis, triggered by a number of things, depending on circumstances;

primarily, damaged cells and pathogens release chemical substances to which macrophages are attracted, mast cells and basophils release histamine and macrophages already at the site release cytokines to attract more of its kind. Unlike neutrophils, the eater cells arriving earlier to sites of infection. The life span of a macrophage ranges from months to years, as opposed to the few days a neutrophil lives. Macrophages are however unable to divide and must mature from monocytes produced in the bone marrow.

Natural sources
Pollution sources that are related to natural processes as opposed to those which are due to human activities.

Neurological
Relating to the body's nerves or nervous system, which oversees and controls all body functions?

Neutrophils
A neutrophil is a type of white blood cell. White blood cell disorders are those diseases affecting the cells in blood that fight infection. White blood cells are one of three blood cells produced by bone marrow. Disorders involving white blood cells include leukaemia, a cancer of the blood, and neutropenia, a rare disorder that causes children to have lower than normal levels of neutrophils. A high count of Neutrophils points to possible infection, some cancers, arthritis and sometimes stress.

Placental membrane
The placenta is a fetomaternal organ. The placental membrane separates maternal blood from fetal blood. Although the placental membrane is often referred to as the placental barrier, many substances, both helpful and harmful, can cross it to affect the developing embryo.

Pulmonary Oedema
Pulmonary edema is swelling and/or fluid accumulation in the lungs. It leads to impaired gas exchange and may cause respiratory failure.

Wet Deposition
The removal of atmospheric particles to the earth's surface by rain or snow

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1 Impacts of Air Pollution

Air pollution is the contamination of the air by harmful gases and particulates at concentrations that are higher than natural background levels. Since the onset of the industrial revolution, there has been a steady change in the composition of the atmosphere mainly due to the combustion of fossil fuels used for the generation of energy and transportation.

Air Pollution has negative impacts on the environment in which we live. It causes health problems in humans and animals, damages plants, pollutes water, threatens aquatic life, deteriorates infrastructure, and reduces visibility. It can also lead to acid rain, global warming, and smog.

This booklet will provide an overview of the effects of air pollution on human health and ecological systems.

2 Effects of Air Pollution on Human Health

Early concerns about health-related effects of air pollution originated from catastrophes like the Fog Disaster in the Meuse Valley, Belgium in 1930; The Donora Fluoride Fog, Pennsylvania, in 1948; and the Great London Smog, in 1952 which caused acute illnesses and premature deaths. Although the effects of these severe pollution episodes remain a topic of debate, well-documented, episode-related increases in morbidity and mortality from cardiopulmonary causes provided clear evidence that extremely high concentrations of air pollution can have adverse effects on health (Pope *et al*., 2002).

Air pollution can affect human health in a number of ways with both acute (short-term) and chronic (long-term) effects. Different groups of individuals are affected by air pollution in different ways depending on their level of sensitivity. Young children and elderly people often suffer more from the effects of air pollution. People with health problems such as asthma, heart and lung disease may also suffer more when exposed to polluted air. Continual exposure to air pollution affects the lungs of growing children and may aggravate or complicate medical conditions in the elderly. The extent to which an individual is harmed by air pollution usually depends on the total exposure to the damaging chemicals. In some cases, even healthy individuals may be at risk to health effects.

Examples of short-term effects include irritation to the eyes, nose and throat, and upper respiratory infections. Other symptoms may include headaches, nausea, and allergic reactions. Short-term exposure to air pollution can aggravate the medical conditions of individuals with asthma and emphysema. Long-term health effects can include chronic respiratory disease, lung cancer, heart disease, and even damage to the brain, nerves, liver, or kidneys.
Air pollution has increasingly become a significant cause of health problems affecting the developed and developing nations around the world. Air pollutants that are inhaled can have serious impacts on human health and mainly target the respiratory and cardiovascular system. Because human lungs have a large surface area and because people inhale large volumes of air, lungs are the most significant site of interaction between air pollutants and the physiological system. Pollutants are also absorbed by the blood and circulated throughout the body, and may cause tissue and organ damage. Humans are exposed to air pollutants in the ambient and indoor environment.

2.1 Ambient Air Pollution

Ambient air pollution refers to pollution that occurs outdoors. Pollutants found in the ambient environment are categorised into criteria and other air pollutants. The potential sources of ambient air pollution are discussed in more detail in the Publication Series 3, Book 3.

2.1.1 Criteria Pollutants

Criteria pollutants are regarded as the common air pollutants for which national ambient air quality standards or guidelines have been set. The six criteria pollutants in South Africa are carbon monoxide (CO), nitrogen dioxide (NO_2), sulphur dioxide (SO_2), ozone (O_3), particulate matter and lead (Pb). Each pollutant in this section is discussed in terms of exposure, population at risk and associated health effects. The nature and characteristics of these pollutants are discussed in detail in Series 3, Book 3. National air quality standards and guidelines exist for criteria pollutants in order to protect human health. The South African guidelines under the Atmospheric Pollution Prevention Act (APPA) are compared with the limit values under the National Environmental Management: Air Quality Act (AQA) and with World Health Organisation (WHO) and the United States Environmental Protection Agency (USEPA) guidelines in Table 1.

Table 1: Comparison of guidelines/standards

	APPA before Jan 02	**AQA, 2004**	**SANS 1929**	**WHO**	**US-EPA**
Ozone					
Instant		500 µg.m^{-3}			
1-hour		230 µg.m^{-3}	200 µg.m^{-3}		
8-hour			120 µg.m^{-3}	120 µg.m^{-3}	155 µg.m^{-3}
NO_2					
1-hour		400 µg.m^{-3}	200 µg.m^{-3}	200 µg.m-3	
24-hour		200 µg.m^{-3}			
1-month		160 µg.m^{-3}			
Annual		100 µg.m^{-3}	40 µg.m^{-3}	40 µg.m-3	100 µg.m-3
SO_2					
Instant peak	600 ppb 1 600 µg.m-3				
10-minute		500 µg.m^{-3}	500 µg.m^{-3}	500 µg.m-3	
1-hour	300 ppb 800 µg.m-3		350 µg.m^{-3}		
3-hours					1300 µg.m-3
24-hour	100 ppb 260 µg.m-3	125 µg.m^{-3}	125 µg.m^{-3}	125 µg.m-3	365 µg.m-3
1-month	50 ppb 130 µg.m-3				
Annual	30 ppb 78 µg.m-3	50 µg.m^{-3}	50 µg.m^{-3}	50 µg.m-3	80 µg.m-3
Pb					
1-month		2.5 µg.m^{-3}			
Quarterly					1.5 µg.m-3
Annual			0.5 µg.m^{-3}	0.5 µg.m-3	
PM10					
24-hour		180 µg.m^{-3}	75 µg.m^{-3}		150 µg.m-3
Annual		60 µg.m^{-3}	40 µg.m^{-3}		50 µg.m-3
CO					
1-hour			30 mg.m^{-3}	30 mg.m^{-3}	40 mg.m-3
8-hour			10 mg.m3	10 mg.m-3	10 mg.m-3

Sulphur Dioxide (SO_2)

Sulphur dioxide causes a wide variety of health effects because of the way it reacts with other substances in the air. Sulphur dioxide is a gas; therefore, the route of exposure will be inhalation. Forty to ninety percent of inhaled SO_2 is absorbed in the moist upper respiratory tract (CCINFO, 1998). Exposure to SO_2 may also occur through skin contact. Exposure may also occur where coal is burnt for domestic purposes, particularly in unvented indoor environments (ATSDR, 1998). Sensitive individuals include asthmatics, the elderly, children and individuals with impediments to nasal breathing (e.g. deviated nasal septum) (Folinsbee, 1992).

Sulphur dioxide is a moderate to strong irritant. Most SO_2 only penetrates as far as the nose and throat, with minimal amounts reaching the lungs, unless the person is breathing heavily, breathing only through the mouth, or if the concentration of SO_2 is high (CCINFO, 1998). The acute response to SO_2 is rapid (Folinsbee, 1992), about 5-10 minutes in asthmatics with significant increase in airway resistance within 2 minutes. Acute responses occur within the first few minutes after commencement of inhalation. Further exposure does not increase effects (WHO, 1999). A reduction in lung function, an increase in airway resistance, wheezing and shortness of breath, are enhanced by exercise that increases the volume of air inspired, as it allows SO_2 to penetrate further into the respiratory tract (WHO, 1999).

An exposure of 1-6 hours to 1 ppm (1 000 ppb or 2 618 µg/m^3) produced a reversible decrease in lung function in sensitive people (CCINFO, 1998). A 10-30 minute exposure to concentrations as low as 5 ppm, (5 000 ppb or 13 089 µg/m^3) has produced broncho constriction in human volunteers (CCINFO, 1998). The acute health effects experienced in sensitive and healthy individuals after acute exposure to SO_2 are listed in Table 2. Epidemiological studies have found associations between hospital admissions for chronic obstructive pulmonary disease and low levels (less than 200 µg/m^3 (or 76 ppb) of SO_2 (Brunekreef *et al*., 1995).

Table 2: Acute health effects experienced in sensitive and healthy individuals after acute exposure to sulphur dioxide

Normal healthy subjects	Reference	Sensitive subjects	Reference
1 ppm (2 600 µg/m³) Pulmonary function changes (during exercise)	Costa & Amdur, 1996	1 ppm (2 600 µg/m^3) Rapid broncho-constriction	Costa and Amdur, 1996
		0.5 ppm (1 300 µg/m^3) Wheezing and shortness of breath (during exercise) Max mid exp flowrate (MMFR) significantly decreased while resting	Folinsbee, 1992 Harrison, 1998 WHO, 1987
		0,25-1 ppm (650-2 600 µg/m^3) Broncho-constriction in mild asthmatics during exercise	Costa & Amdur, 1996

Evidence for effects of SO_2 other than short-term bronchoconstriction is less direct (Lippmann, 1992). Workers exposed to a daily average of 5 000 ppb (13 089 µg/m^3) showed a higher incidence of chronic bronchitis. Human studies showed that repeated exposure to SO_2 levels below 5 000 ppb (13 089 µg/m^3) resulted in pulmonary impairment (probably due to repeated episodes of broncho constriction) (CCINFO, 1998).

Most studies on humans and animals have indicated that 49-90% or more of inhaled SO_2 is absorbed in the moist upper respiratory tract where it is quickly converted to sulphuric acid (H_2SO_4) (CCINFO, 1998). Sulphuric acid imparts irritation to respiratory tissues through its acidity. Inhaled SO_2 is slowly removed from the respiratory tract. After absorption in the blood stream the H_2SO_4 is widely distributed throughout the body, quickly converted to sulphite and bisulphite, which in turn is oxidised to sulphate in the liver and excreted in the urine (CCINFO, 1998).

A summary of health effects of sulphur dioxide is provided in Table 3.

Table 3: Summary of sulphur dioxide health effects (adapted and modified from WHO, 1979 and Turco, 2002)

Concentrations (ppm)	Effects
0.3	Taste threshold
0.5	Odour threshold
1.5	Bronchiolar constriction Respiratory infection
2	ACGIH recommended TLV-TWA
3	Easily detected odour
6-12	May cause nasal and throat irritation
10	Upper respiratory irritation, nose bleeding
20	Eye irritation, development of chronic respiratory symptoms, respiratory protection is advised
50-100	Maximum tolerable exposures for 30-60 minutes
≥ 100	NIOSH recommended immediate danger to life

Particulate Matter (PM)

Particulate matter may contain both organic and inorganic pollutants. It is regarded as one of the most critical of all pollutants. The extent to which particulates are considered harmful depends on their chemical composition and size. For example particulates such as emissions from diesel vehicle exhausts mainly contain unburned fuel oil and hydrocarbons that are known to be carcinogenic. Very fine particulates pose the greatest health effects as they can penetrate deep into the lung and cause more damage, as opposed to larger particles that may be filtered out through the airways' natural mechanisms.

Exposure to particulate matter occurs as a result of inhaling air that contains the pollutant or ingesting (air cannot truly be "ingested"; the mechanism is through the ingestion of inhaled particulates that are expelled by the normal actions of the ciliated epithelium of the lung. The expelled particulates are deposited in the back of the throat, from where it is swallowed and ingested). Dermal contact may also occur although this is more significant to occupational exposure. People with chronic obstructive pulmonary and/or cardiovascular disease, asthmatics, the elderly and children, are more at risk to the inhalation of particulates than normal healthy people (Pope, 2000; Zanobetti *et al.*, 2000).

The exact mechanism of action for the observed health effects from particulate matter is not known (Martin *et al.*, 1997; Monn 2001). Human studies show pro-inflammatory effects that involve both epithelial cells and macrophages, particularly metal and organic compounds (Brunekreef and Holgate, 2002). Some recent research suggests that particles (particularly the ultra fine ones) or particle components may physically move out of airways into the bloodstream to trigger effects at distant sites (HEI, 2002).

The health effects of particulates can range from negligible to potentially fatal depending on particle size, chemical composition, mass inhaled, route of exposure (WHO, 1999) and the number of particles per unit volume of air (<$PM_{2.5}$) (Lippmann, 2003). Health effects associated with particulate matter include both acute and chronic effects (Brunekreef and Holgate, 2002). Acute effects include an increase in daily mortality with an increase in particulate matter concentrations, increased hospital admissions for exacerbations of respiratory illnesses such as asthma while chronic effects may include lung cancer (WHO, 1999). Fine particulate matter ($PM_{2.5}$) is regarded as a better indicator for health effects than coarse particles (>$PM_{2.5}$) (Lippmann, 1998). Fine particles penetrate deeply into the lungs, and are more likely than coarse particles to contribute to the health effects (e.g. premature mortality and hospital admissions).

Both PM_{10} and TSP are more likely to be trapped by the nasal hairs leading to a limited effect on the respiratory system (Lippmann, 1998). However there are studies showing that coarse particulate matter induces adverse effects. Wilson and Spengler (1996) reported that the estimated effect of PM_{10} across daily time-series mortality studies shows a statistically significant effect. These results are similar to those reported by Levy and co-workers which indicated that for every 10 µg/m³ increase in PM_{10} over 24 hours an increase in acute morbidity, such as asthma attacks and bronchitis, were reported (Levy *et al.*, 2000).

Long-term repeated exposure to particulates may increase the risk of chronic respiratory disease, and the risk of cardio-respiratory mortality (Wilson and Spengler, 1996). A critical review of about 200 scientific publications by The Air and Waste Management Association on ambient particulates and health, found no evidence that long-term low-level exposure to particles, apart from repeated short-term increases in particle concentrations, is associated with adverse health effects (Vedal, 1997). There is thus controversy among scientists in the field.

One group acknowledged that it is impossible to set a standard that will be totally protective against all adverse effects and therefore recommended a concentration at which health effects on individuals are likely to be small and the very large majority of individuals will be unaffected. A differing opinion is that a threshold for adversity can be identified and a margin of safety can be applied (Lippmann, 1998). Reanalyses of three of the largest US cohort studies confirmed the original findings, namely robust associations between mortality rates and levels of particulate matter (Brunekreef and Holgate, 2002).

Ozone (O_3)

Ozone is a gas and exposure occurs through inhalation. Ozone even at low concentrations is very reactive and can have adverse effects on human health. People with existing respiratory illnesses and those who are involved in outdoor activities are most at risk to ozone exposure.

Ozone mainly affects the respiratory system. Both short-term and long-term effects of ozone have been reported in human subjects (Table 4). Short-term ozone exposure leads to the development of airways irritation and inflammation leading to a decrease in lung function. These are marked by a two-fold increase in neutrophils in the bronchoalveolar lavage (Lippmann, 1992). Associated symptoms include wheezing, coughing, pain when taking a deep breath and breathing difficulties during exercise or outdoor activities (WHO, 1999). There are also reports of increased hospital admissions for respiratory problems related to ozone exposure (WHO, 2003).

Prolonged exposure to ozone leads to a reduction in lung function in children (WHO, 2003). It also leads to morphological changes in the lung and permanent lung damage (WHO, 1999). There is limited evidence for an independent long-term ozone effect on lung cancer or total mortality (WHO, 2003). Even at very low levels, increased susceptibility to respiratory illnesses like pneumonia and bronchitis are possible (WHO, 1999).

Table 4: Summary of ozone health effects (adapted form Turco, 2002)

Concentrations (ppm)	Effects
0.02	Odour threshold
0.1	Nose and throat irritation in sensitive people
0.12	Decreased lung function
0.3	General nose and throat irritation
1.0	Airway resistance and headaches Premature ageing of lung tissue

Lead (Pb)

There are two forms of lead, organic and inorganic. Inorganic lead is the predominant form of lead in the environment. Organic lead can be metabolised to inorganic lead in the body. Lead is absorbed, metabolised and stored in the body. The half-life of inorganic lead in the bone is 27 years. Information on the distribution of organic lead in the body is extremely limited.

Lead occurs in particulate form in the environment. Upon inhalation, the particles are deposited in the respiratory system where they become absorbed into the bloodstream. Deposition and absorption of lead particles in the respiratory system depends on particle size and ventilation rate.

Lead can also enter the body through ingestion. Ingested lead is deposited into the gastrointestinal tract where absorption takes place. The rate of absorption is influenced by various factors including person-based factors such as age, and nutritional iron and calcium status. Children who have high iron levels in their blood appear to have lower blood lead levels suggesting that iron mitigates against high lead absorption. Lead absorption increases when calcium levels are low (ATSDR, 1999).

Lead absorption is also affected by physical and chemical properties of the lead particles (particle size, mineralogy, and lead species) (ATSDR, 1999). In the gastrointestinal tract, absorption of lead appears to be higher in children than in adults (Alexander *et al*., 1974; Ziegler *et al*., 1978).

The absorbed lead is transported to various body organs and tissues through blood. In the blood tissue, lead is found in red blood cells where it binds with haemoglobin. Lead in the blood is transported to soft tissues like the brain, liver and kidneys and is eventually stored in the bone (ATSDR, 1999).

Children are more vulnerable to lead poisoning than adults. Expectant mothers pass lead onto their unborn children if the mother has a high blood lead content. Infants may also be exposed to lead through breast feeding, eating foods and/or drinking water contaminated with lead. Children can also ingest lead from playing with toys that have dust lead particles, or which are painted with paint containing lead.

According to the ATSDR (1999), the main health effect of lead is impairment in the functioning of the nervous system. Other effects include weaknesses in fingers, wrists or ankles. Dietrich *et al*. (1993) reported that children in the 6.5 year age group experienced difficulty with IQ tests while Wasserman *et al*. (1998) observed abnormal behaviour in children of 3 years of age.

Lead can affect the blood tissue causing high blood pressure, anaemia, and a decrease in blood cells. At high concentrations, lead may lead to brain and kidney damage. Lead may also affect the liver and hormonal functions in the body (ATSDR, 1999) (ATSDR, 1999).

Lead can be a cause of decreased fertility (Lin *et al*., 1996). Lead exposure has been associated with miscarriages and stillbirths in pregnant women (Hu, 1991; Baghurst *et al*., 1987). It can also affect sperm production in males at high exposures (Alexander *et al*., 1996). . A summary of health effects of lead is given in Table 5.

Table 5: Summary of lead health effect (adapted from ATSDR, 1999.)

Concentration (μg/dL) Blood lead levels	Effects
30 - ≥ 70 40-80	Neurological effects (decreased nerve conduction velocity in adults, Disturbances in ocular motor function, reaction time, visual motor performance, hand dexterity, IQ test and cognitive performance, mood, coping ability, memory
≥ 40	Haematological effects
21-90	Immunological effects (Depression of cellular immune function but no effect on humoral immune function)
400-200	Gastrointestinal effects (abdominal pain, constipation, cramps, nausea, vomiting, anorexia weight loss)

Carbon Monoxide (CO)

People with pre-existing heart and respiratory conditions, blood disorders and anaemia are sensitive to the effects of CO. Health effects of CO are mainly experienced in the neurological system and the cardiovascular system (WHO, 1999).

Upon inhalation, CO enters the blood stream by crossing the alveolar, capillary and placental membranes. In the bloodstream approximately 80-90% of absorbed CO binds with haemoglobin to form carboxyhaemoglobin. The haemoglobin affinity for CO is approximately 200-250 times higher than that of oxygen. Carboxyhaemoglobin reduces the oxygen carrying capacity of the blood and reduces the release of oxygen from haemoglobin, which leads to tissue hypoxia (WHO, 1999).

Tissue hypoxia may lead to reversible, short lived neurological effects and sometimes delayed severe neurological effects (WHO, 1999). These effects may include impaired coordination, vision problems, reduced vigilance and cognitive ability, reduced manual dexterity, and difficulty in performing complex tasks. At high concentrations poisoning and death may occur (WHO, 1999).

Tissue hypoxia also affects the cardiovascular system. People with existing heart conditions such as angina, clogged arteries, or congestive heart failure are particularly sensitive. In these cases, CO may induce chest pain and lead to the development of other cardiovascular effects such as myocardial infarction, and cardiovascular mortality (WHO, 1999). A summary of health effects of CO is given in Table 6.

Table 6: Summary of CO health effects (adapted from WHO, 1999 and Turco, 2002)

Concentrations (ppm)	Effects
10-30	Time distortion (typical urban)
~100	Throbbing headache (freeways, 100 ppmm)
300	Vomiting, collapse (tobacco smoke, 400 ppmm)
600	Death

Nitrogen Dioxide (NO_2)

Health risks from nitrogen oxides may potentially result from NO_2 itself or its reaction with other chemicals to form ozone and secondary particles (WHO, 2003). It is therefore important to understand the chemistry of NO_2 when assessing its health impacts.

Nitrogen dioxide is a gas and the route of exposure is therefore through inhalation. About 80-90% of inhaled NO_2 is absorbed through the lungs (CCINFO, 1998). People will be exposed to inhalation of NO_2 by inhaling contaminated ambient air or air from unvented indoor domestic fuel burning or tobacco smoke. The widespread use of combustion appliances in homes is responsible for indoor NO_2 concentrations that exceed those found outdoors (WHO, 1987).

People with chronic respiratory problems and people who work or exercise outside will be more at risk to NO_2 exposure (EAE, 2006). People with a vitamin C deficiency may be more at risk, as vitamin C inhibits the oxidation reactions of NO_2 in the body (WHO, 1997).

The seriousness of effects of exposure to NO_2 depends more on the concentration than the length of exposure. The onset of some symptoms can be delayed for up to 36 hours (CCINFO, 1998). The site of deposition for NO_2 is the distal lung (Costa and Amdur, 1996). In the lung NO_2 reacts with moisture in the fluids of the respiratory tract to form nitrous and nitric acids (WHO, 1997).

Nitrogen dioxide causes decrements in lung function, particularly increased airway resistance in resting healthy individuals at 2-hour concentrations as low as 4700 µg/m³ (WHO, 1997). Increased airway responsiveness to broncho constrictive agents in exercising healthy, non-smoking subjects was the result when they were exposed to 2800 µg/m³ NO_2 for 1-hour (WHO, 1997).

Some studies reported that exposure of asthmatics to NO_2 may cause increased airway responsiveness to provocative mediators (such as SO_2), which may already begin at 380 µg/m³ (WHO, 1997). Nose, eye and throat irritation can occur at concentrations between 20000 to 33000 µg/m³. Other symptoms may include coughing, dyspnoea (a feeling of inability to breath), headache and nausea. At concentrations of 33000-130000 µg/m³, irreversible inflammation of the lungs may develop. Concentrations of ~200000 µg/m³ lead to potentially fatal pulmonary oedema and progressive blockage of the small airways (CCINFO, 1998). Romieu and Hernandez-Avila (2003) also reported that exposure to NO_2 leads to immune system effects.

According to the World Health Organisation (WHO), there is no evidence of a clearly defined concentration-response for NO_2 despite the large number of acute controlled exposure studies in humans. A lowest observed adverse effect level (LOAEL), based on slight changes in lung function in asthmatics, is given as a range between 365-565 µg/m³ (200-300 ppb) (WHO, 2000).

Reports of lung effects from long-term exposure to low levels of NO_2 are inconsistent (CCINFO, 1998). Chronic exposure to NO_2 increases the risk to respiratory tract infection in young children (WHO, 1997).

The recommended long-term guidance value, based on epidemiological studies of increased risk of respiratory illness in children, is 40 µg/m³ as an annual average (WHO, 1997). The association between outdoor NO_2 and respiratory health is not clear. There is some evidence that the duration of respiratory illness may be increased at higher ambient NO_2 levels. The difficulty is to distinguish effects of NO_2 from other associated pollutants (WHO, 1997).

Nitrogen dioxide (present in the blood as the nitrite (NO_2-ion)) oxidises unsaturated membrane lipids and proteins, which then results in the loss of control of cell permeability. The dominant product of oxidation of unsaturated lipids is peroxide. Acute exposure to 750 µg/m³ NO_2 can result in lipid peroxidases (WHO, 1997). Nitrogen dioxide may also alter cellular chemical balance

leading to cellular alterations (WHO, 1999). Table 7 provides a summary of nitrogen dioxide health effects.

Table 7: Summary of nitrogen dioxide health effects (adapted from WHO, 1999 and Turco, 2002)

Concentration	Effects
0.06-0.1	Respiratory impact (long-term exposure promotes disease)
0.5	Increased susceptibility to bacterial and viral infections
1.5-5.0	Breathing difficulty
25-100	Acute bronchitis
150	Death (may be delayed)

2.1.2 Other Air Pollutants

A number of other air pollutants also induce health effects which can be carcinogenic or non-carcinogenic. The group of pollutants discussed in this section include volatile organic compounds, metals and persistent organic pollutants.

Volatile Organic Compounds (VOCs)

VOCs are organic chemicals that easily vaporize at room temperature and are called organic because they contain the element carbon in their molecular structures. VOCs include a very wide range of individual substances, such as hydrocarbons (for example benzene and toluene), halocarbons and oxygenates. The VOCs discussed in this section include benzene, toluene, xylene and polycyclic aromatic hydrocarbons (PAHs).

Benzene

Benzene is also known as benzol, coal naphtha, mineral naphtha, phenylhydride, pyrobenzol and pyrobenzole, and is a volatile, colourless, highly flammable liquid, with an odour threshold of 1.5 ppm (5 mg/m^3) (ATSDR, 1997).

Benzene, a non-methane hydrocarbon, is a colourless, clear liquid. It is fairly stable but highly volatile, readily evaporating at room temperature. Since 80% of man-made emissions come from petrol-fuelled vehicles, levels of benzene are higher in urban than rural areas. The main exposure route of benzene is through inhalation. Most people are exposed to a small amount of benzene on a daily basis. The general public may become exposed to benzene from motor vehicle exhaust fumes, unvented wood fires, cigarette smoke, service stations, exhaust from motor vehicles, and industrial emissions. Vapours (or gases) from products that contain benzene, such as glues, paints, furniture wax, and detergents can also be a source of exposure. Benzene levels in the home are usually higher than outdoor levels. People living around hazardous waste sites, petroleum refining operations, petrochemical manufacturing sites, or gas stations may be exposed to higher levels of benzene in air.

Literature shows that alcohol enhances benzene metabolism and toxicity. Therefore people who drink alcohol and are exposed to benzene may be more at risk to the effects of benzene,

particularly blood disorders. Benzene can also increase ethanol-induced effects on the brain (ATSDR, 1997).

People with blood disorders, as well as children and unborn babies (their blood cell populations are growing), are also more at risk to the toxic effects of benzene (ATSDR, 1997). Health effects are reflected in Table 8.

Table 8: Summary of benzene health effects (adapted from CCINFO, 1998)

Concentrations	Effects
50 to 150 ppm	Neurological effects (headaches, tiredness)
	Blood (excessive bleeding) and immune system (changes in antibody levels in the blood and loss of white blood cells) disorders , cancer

Toluene

Toluene is also known as methyl-benzene, phenylmethane, or toluol. It is a colourless, flammable liquid, with an odour threshold that varies between 0.16 and 37 ppm (CCINFO, 1998).

The public may be exposed to toluene by inhaling contaminated ambient air, motor vehicle exhaust fumes, or by using consumer products that contain toluene, such as nail polish and stain removers. Smoking tobacco and solvent abuse also exposes people to toluene.

The population at risk includes people drinking ethanol (alcohol), as this may slow down the clearance of toluene from the body (CCINFO, 1998). Combinations of toluene and common medicine, like aspirin, may increase the effects of toluene on hearing (ATSDR, 1997). People with neurological disorders may also be at risk when exposed to toluene, as the central nervous system is the main target organ of toluene. Long-term malnutrition may increase susceptibility of the developmental effects of toluene (ATSDR, 2000). Table 9 shows health effects associated with toluene exposure.

Table 9: Summary of Toluene health effects (adapted from CCINFO, 1998)

Concentration ppm	Effects
50	Slight drowsiness and headaches
50-100	Upper respiratory irritation
100	Fatigue, dizziness
≥ 200	Mild nausea symptoms similar to drunkenness
≥ 500	Mental confusion
4 000 to 12 000	Persistent neurological damage (chronic exposure)

Xylene

Xylene (non-specific) is also known as dimethylbenzene, xylol or methyltoluene. There are three isomers of xylene in which the methyl groups vary with regard to their position on the benzene ring: meta- ortho- and para-xylene (m-, o- and p-xylene). Xylene is a colourless flammable liquid with an odour threshold of 0, 08 ppm (ATSDR, 1997).

Xylene exposure occurs when people inhale air containing xylene, fumes from motor vehicle exhausts, using consumer products containing xylene, and tobacco smoking.

Foetuses and very young children have immature enzyme detoxification systems, and are therefore more at risk to xylene exposure. People with epilepsy are more at risk to xylene, because of its excitatory effects on the central nervous system. People taking alcohol are more at risk to xylene exposure, as alcohol consumption promotes xylene toxicity and people with asthma, liver or kidney disorders will also be more sensitive to xylene exposure (ATSDR, 1997). A summary of health effects is provided in Table 10.

Table 10: Summary of xylene health effects (adapted form ACGIH, 1996 and CCINFO, 1998)

Concentration ppm	Effects
200 230	Eye, nose and throat irritation. Slight lightheadedness
700	nausea and vomiting
10 000	Death (6 hrs)

Polycylic Aromatic Hydrocarbons

Polycyclic aromatic hydrocarbons (PAHs) are a group of more than 100 different chemicals, including carcinogens, found in a mixture in the environment (WHO, 1998). Exposure to PAHs occurs through inhalation of contaminated air and ingestion of grilled or charred meat and processed or pickled foods (WHO, 1998).

Both non-cancerous and cancerous health effects have been observed in exposed populations. However non-cancerous effects have largely been observed in animals, not humans. Through inhalation, PAHs may induce lung tumours in the exposed. They also lead to the development of tumours in the urinary bladder, renal pelvis, mouth, pharynx, larynx, and oesophagus. Skin contact may induce skin reactions (WHO, 1998).

Metals

Mercury (Hg)

Mercury is a naturally occurring element that is found in air, water and soil. It exists in several forms: elemental or metallic mercury, inorganic mercury compounds, and organic mercury compounds.

Mercury is released into the atmosphere in three forms. These include elemental (or metallic) mercury, particle-bound mercury and oxidized mercury (sometimes called ionic or reactive gaseous mercury). Atmospheric mercury can be transported over a range of distances before it is deposited, potentially resulting in deposition on local, regional, continental and/or global scales.

The most common way people are exposed to mercury is by eating fish containing methylmercury. Other exposures may result from using or breaking products containing mercury (EPA, 2006a).

Methylmercury exposure

Microscopic organisms convert inorganic mercury into methylmercury, which accumulates up the food chain in fish, fish-eating animals, and people. Adults, children, and developing foetuses are at risk from dietary exposure to methylmercury. The developing nervous system of the fetus may be more vulnerable to methylmercury than is the adult nervous system. Mothers who are exposed to methylmercury and breast-feed their babies may also expose their infant children through their milk (EPA, 2006a).

Elemental mercury exposure

Elemental or metallic mercury is the liquid metal used in thermometers, barometers, and thermostats and electrical switches. If broken, they release metallic mercury to the environment. Children often break fever thermometers in their mouths. Mercury that is swallowed in this way poses low risk in comparison with the risk of breathing mercury vapour. When elemental mercury is spilled or a device containing mercury breaks, the exposed elemental mercury can evaporate and become an invisible, odourless toxic vapour. This often occurs in warm or poorly-ventilated rooms or spaces (EPA, 2006a).

Exposure to other mercury compounds (inorganic and organic)

Inorganic mercury compounds take the form of mercury salts. They are generally white powders or crystals, with the exception of mercuric sulphide (cinnabar) which is red. Inorganic compounds and organic compounds (such as phenylmercury acetate and ethylmercury), have been commonly used as fungicides, antiseptics or disinfectants. They have also been used in a variety of products (EPA, 2006a).

Excessive exposure to inorganic and organic mercury compounds can result from misuse or overuse of mercury-containing products, especially outdated products containing more mercury. Exposure to mercury compounds occurs mainly through ingestion. Ingested organic mercury compounds are more readily absorbed through the gastrointestinal tract than are inorganic compounds (EPA, 2006a).

Effects of methylmercury exposure on wildlife can include mortality (death), reduced fertility, slower growth and development and abnormal behaviour. In addition, the endocrine system of fish, which plays an important role in fish development and reproduction, may be altered by the levels of methylmercury found in the environment (EPA, 2006a).

Chromium (Cr)

In the natural environment, chromium is present as fine particles and usually occurs in two main oxidation states, trivalent (Cr^{3+}) and hexavalent (Cr^{6+}). Each of the oxidation states has very different biological and chemical properties and its behaviour in the environment depends on its oxidation state. Hexavalent chromium is generally more toxic, while Cr^{3+} is more stable and presents lower toxicity (IPCS, 1988).

The main routes of human exposure to chromium are inhalation and ingestion. Inhalation intake in occupationally exposed people and cigarette smokers may far exceed the inhalation intake of the general population (Spectrum, 1999). Autopsies showed that inhaled chromium builds up in the lungs (Kimbrough *et. al*., 1999). Trivalent chromium is an essential nutrient required to promote the action of insulin so that sugar, protein and fat can be used by the human body. Inorganic chromium compounds do not appear to have insulin potentiating activity (IRIS, 1999). Respiratory cancer is the health effect of most concern, and is the basis for the regulation of Cr^{6+}. Hexavalent chromium appears to be a contact carcinogen to the respiratory system. It has not been reported to be a carcinogen for organs for which the only exposure to Cr^{6+} is via the blood (Kimbrough *et. al*., 1999).

People at risk to chromium include people living near landfill sites with chromium-containing waste and people working in or living near facilities that manufacture or use chromium and chromium-containing compounds.

The US Food and Nutrition Board have recommended a safe and adequate dietary intake of 50-200 µg total chromium per day for adults (±0, 7-2, 9 µg/kg/day) (Spectrum, 1999). The oral reference dose (RfD) for Cr^{6+} soluble salts (including potassium dichromate, sodium dichromate, potassium chromate and sodium chromate) is 3 µg/kg/day. The RfD for Cr^{3+} (insoluble salts) is 1, 5 mg/kg/day (1500 µg/kg/day). The RfD is an estimate of a daily exposure to the human population that is likely to be without an appreciable risk of deleterious effects during a lifetime (IRIS, 1999).

Once absorbed into the bloodstream, Cr^{6+} readily enters red blood cells. Trivalent chromium cannot be readily absorbed therefore enters the cells through a different pathway, but with very low efficiency (IRIS, 1999).

A significant amount of chromium is taken up in bone. It is also concentrated in tissues of the liver, kidney and spleen. The latter could be because the liver and kidney are the main routes of excretion of chromium (Kimbrough *et. al.*, 1999). Once in the cell, Cr^{6+} may be reduced to Cr^{3+}, which may subsequently interact with cellular macromolecules, including DNA, or it may be slowly released from the cell. The bio-accessibility of chromium to absorption processes may be the single most important factor determining the toxicity of a specific chromium source (IRIS, 1999).

The main reason Cr^{3+} does not cause toxic effects is because it has a poor ability to enter cells but if the Cr^{6+} acts as a "Trojan horse" delivering Cr^{3+} into the cell, then toxicity is possible. Hexavalent chromium $(Cr^{6})^{+}$ is considerably more toxic than Cr^{3+}. A concentration of 0,5 mg/kg/day of Cr^{6+} in drinking water caused oral ulcers, diarrhoea, abdominal pain, indigestion, vomiting and disorders of white blood cells. Data of effects for lower exposure doses were not available (IRIS, 1999).

The conclusion of almost all epidemiology studies were that not all forms of Cr^{6+} were carcinogenic, but rather the water-insoluble species (Kimbrough *et. al.*, 1999). The water-soluble species are regarded as causing an allergic reaction to the skin (IRIS, 1999). It is therefore believed that the use of total Cr^{6+} overestimates most cancer risk assessments, because it fails to distinguish water-soluble from insoluble Cr^{6+} in non-aqueous matrices (Kimbrough *et al.*, 1999).

The US Department of Health and Human Services classified calcium chromate, chromium trioxide, lead chromate, sodium dichromate (limited evidence), strontium chromate and zinc chromate as known carcinogens, while the EPA has classified Cr^{6+} as a human carcinogen of high carcinogenic hazard (ATSDR, 1997). The carcinogenicity of Cr^{6+} through ingestion could not be determined (IRIS, 1999). EPA considers Cr^{3+} as not classifiable as to carcinogenicity in humans (EPA-TTN, 1998).

Other Metals

The health effects of other heavy metals is summarised in Table 11.

Table 11: Health effects associated with metals (after Turco, 2002)

Metals	Concentration (ppm)	Effects
Arsenic	0.5	Cancer of the lungs, liver, and skin Teratogenic Poisonous in large doses
Cadmium (Cd)	0.2	Accumulation in the kidneys, lungs and heart Symptoms like Wilson's diseases Carcinogenic

	50	Fatal within 1 hour
Manganese	5.0	Aching limbs and back, drowsiness, loss of bladder control, nasal bleeding
Nickel (Ni)	1.0	Skin rashes, cancer of the sinus and lungs (after continued exposure), 0.001 ppm of nickel carbonyl leads to nausea, vomiting, and possible death
Vanadium (V)	0.5	Acute spasm of the bronchii, emphysema
Zinc (Zn)	5.0	Fever, muscular pain, nauseas and vomiting

2.2 Indoor Air Pollution

Indoor air pollution is of importance since people spend close to 90% of their time indoors. In the developing world the highest air pollution exposures occur in the indoor environment.

2.2.1 Concerns about the State of Indoor Air Pollution in South Africa

Despite widespread electrification, a large part of the South African population depends on biomass fuels such as coal, wood, paraffin, dung and agricultural residues, for cooking and heating. The use of such solid fuels on open fires or stoves without chimneys leads to indoor air pollution. Indoor smoke contains a range of harmful pollutants including tiny soot or dust particles and carbon monoxide.

Exposure is particularly high among women and children, who spend the most time near the fire. In most societies, women are in charge of cooking and therefore spend most time near the domestic hearth, preparing food. Young children are often carried on their mother's back or kept close to the warm hearth. In this way, infants spend many hours breathing indoor smoke during their first year of life when their developing airways make them particularly vulnerable to hazardous pollutants (WHO, 2006).

Indoor air pollution from solid fuel use is responsible for 1.6 million deaths due to chronic respiratory disease and lung cancer, globally. There is consistent evidence that exposure to indoor air pollution increases the risk of pneumonia among children under five years, and chronic respiratory disease and lung cancer (in relation to coal use) among adults over 30 years old (WHO, 2006). According to Scorgie *et al*. (2004), domestic fuel burning was estimated to result in the greatest non-carcinogenic health risks across all conurbations considered, accounting for approximately 70% (or 83 230) of all respiratory hospital admissions in South Africa. Acute Lower Respiratory Infections which commonly lead to pneumonia (WHO, 2006) accounts for approximately 14% of deaths amongst children less than five in South Africa (MRC, 2006).

Measures to reduce indoor air pollution and associated health effects include switching to cleaner alternatives, such as gas, electricity or solar energy, improved stoves or hoods that vent health-damaging pollutants to the outside and behavioural changes (WHO, 2006).

2.2.2 Other Important Sources of Indoor Pollutants

Important sources of indoor pollutants include ambient air, emissions from building materials, furnishings and appliances such as stoves and use of consumer products. Most pollutants present in the outdoor air are also found indoors. However, indoor sources may lead to an accumulation of

certain compounds that are not often found in ambient air. The main indoor pollutants include particulate matter, SO_2, nitrogen oxides (NO_x) CO, volatile organic compounds (VOCs).
Microbial contamination is mostly related to the presence of humidity. Air conditioning systems can also act as a pollutant source, especially when not properly maintained. For example, old filters can eventually re-emit particulate contaminants and be a breeding source of micro-organisms. Concentrations of combustion products in indoor air can be substantially higher than those outdoors when heating and cooking appliances are used. Tobacco smoke contains a complex mixture of several thousand chemicals, including known carcinogens such as nitrosamines and benzene. The exposure of children to tobacco smoke is therefore a major problem particularly for indoor air quality and environmental health.

2.2.3 Health Effects and Symptoms

Health effects of indoor air pollution depend on the specific pollutants, the intensity and duration of exposure, and also on the health status of the exposed population. The population which may be at greater risk include babies, children, pregnant women and the unborn babies, the elderly, those suffering from respiratory or allergic diseases and people exercising. Common indoor air pollutants are:

Tobacco smoke

Inhalation of tobacco smoke can lead to reduced pulmonary function, an increased incidence of respiratory symptoms and infections, and to an increased incidence of lung cancer.

Infectious microorganisms

The primary mechanism of most acute respiratory infections is the inhalation of micro organisms that may cause infection. These micro organisms are discharged by people and animals. Reduced ventilation and recirculated air may cause the concentration of micro organisms to increase in indoor environments.

Outdoor allergens, house dust mites, and moulds

Outdoor allergens, house dust mites, and moulds in indoor environments especially in high humidity can cause allergic asthma (reversible narrowing of lower airways), allergic rhinoconjunctivitis in children and young adults.

Volatile Organic Compounds (VOCs)

Health effects reported for VOCs range from sensory irritation to behavioural, neurotoxic, hepatoxic and genotoxic effects.

Asbestos and other mineral fibres

Asbestos and other mineral fibres may be a cause of an increased incidence of lung cancer. Acute exposure to asbestos and glass fibres can cause severe skin irritation.

3 Effects of Air Pollution on Ecological Systems

When pollutants are released into the atmosphere, they react with other airborne chemicals to form acid aerosols which can be transported over long distances, following the pattern of prevailing winds. They eventually settle on ground or water through dry deposition or wet deposition. The deposition of atmospheric pollutants and high concentrations of pollutants in the environment represent a significant threat to sensitive ecosystems. The effects of this settling include the

acidification of lakes and streams; modification of the nutrient balance in coastal waters and large river basins, depleting the soil nutrients, damaging sensitive forests and farm crops, and affecting the ecosystem diversity. Prolonged exposure can lead to bioaccumulation of certain pollutants and changes in the natural variety of plants and animals in an ecosystem. In addition, acid rain and certain pollutants accelerate the decay of building materials and paints, including monuments, statues, and sculptures. Microscopic acid aerosols impair visibility and contribute to climate change.

3.1 Pollutant Impacts

Air pollution effects of particular concern on ecological systems are NO_x, SO_2, O_3, Pb and particulate matter, fluoride, mercury and benzene. Carbon dioxide and chlorofluorocarbons have global scale implications.

Sulphur Dioxide (SO_2)

Sulphur dioxide is one of the main precursors of acid rain. In the atmosphere, SO_2 oxidises and combines with water vapour forming sulphuric acid (H_2SO_4), which is the main component of acid rain. Sulphur dioxide also accelerates the decay of building materials, paints, historical monuments; corrodes metal; and deteriorates electrical installations, paper and textiles.
Sulphur dioxide damages trees and crops by causing injuries to leaves and reducing photosynthesis. Sulphur dioxide enters leaves through the stomata, but is also deposited on wet leaf surfaces, where it may form sulphite or bisulphite and react with cuticular waxes (Wellburn, 1994). Internally SO_2 can damage plants with a low buffering capability. Visible SO_2 damage is characterised by chlorosis of leaf tissue (whitened areas of dying tissue). Sulphur dioxide can cause a reduction in growth and yield (Emberson, 2003).

Nitrogen Oxides (NO_x)

Nitrogen oxides cause a wide variety of environmental impacts because of various compounds and derivatives in the family of nitrogen oxides. Nitrogen oxides react with ammonia, moisture, and other compounds to form nitric acid (HNO_3) and related particles which contribute to acid rain. Increased nitrogen loading in water bodies, particularly coastal estuaries, upsets the chemical balance of nutrients used by aquatic plants and animals. Additional nitrogen accelerates "eutrophication," which leads to oxygen depletion and reduces fish and shellfish populations.

Nitrogen oxides contribute to ground-level ozone (smog) when it reacts with volatile organic compounds (VOCs) in the presence of sunlight. Nitrogen oxides also react with common organic chemicals and even ozone, to form a wide variety of toxic products, some of which may cause biological mutations (EPA, 2006b). Examples of these chemicals include the nitrate radical, nitroarenes, and nitrosamines.

The main pathway for NO_x entry to plant leaves is through the stomata, although cuticular resistance to NO_x is lower than for NO_x and O_3. Nitrogen oxides can reduce plant growth at high concentrations. However, in situations of low soil nitrogen growth stimulation can be caused by low NO_x concentrations (CLAG, 1996). Prolonged exposure to NO_x suppresses plant growth through inhibiting photosynthesis. The combination of NO_x with other pollutants such as SO_2 and O_3 results in synergistic negative effects on plants (Emberson, 2003).

Ozone (O_3)

Ozone can have long term impacts on forests and ecosystems - including disruption of ecological functions (such as water movement and mineral nutrient cycling) and adverse impacts on the natural habitat of plants and animals.

Ground-level ozone interferes with the ability of plants to produce and store food, which makes them more at risk to disease, insects, other pollutants, and harsh weather.

Ozone uptake is almost entirely through the stomata. In the sub-stomatal cavity O_3 reacts with constituents of the aqueous matrix with the cell wall to form other derivatives which result in the oxidation of sensitive components. The inability to repair or compensate for altered membrane permeability can be manifest as symptoms of visible injury. This is often associated with short term exposure to high O_3 concentrations (Emberson, 2003). The symptoms on broad leaf plants include chlorosis, bleaching, bronzing, flecking, stippling and necrosis. Tip necrosis, mottling and banding are common on conifers (Kley *et al*, 1999). Reductions in growth can result in crop yield losses and reduction in annual biomass increments for forest trees (Fuhrer *et al*., 1997)

Suspended Particulate Matter (SPM)

Particles such as soot are mainly responsible for staining buildings and objects made from stone.

The effect of SPM on vegetation depends in most cases on the chemical composition of particles. Heavy metals and toxic particles can cause damage and death to some species as a result of phytotoxicity and abrasive action during turbulent deposition. Heavy deposition loads can result in reduced light transmission to chloroplasts and the occlusion of stomata, decreasing efficiency of gas exchange (Emberson, 2003). Physiological processes such as bud-break, pollination and light absorption can also be disrupted (Beckett *et a*l., 1998).

Fluoride (F)

Particulate fluoride can enter the leaf through the cuticle if the cuticle is old or weathered (Emberson, 2003); while gaseous fluoride enters through the stomata and can accumulate in toxic levels in the tips and margins of leaves (Jacobsen *et al*., 1996). Plants show a wide range of susceptibility to fluoride with sensitive plants showing damage in the form of chlorosis and necrosis of leaf tips and margins (Wellburn, 1994).

Exposure to fluoride can result in reductions in photosynthesis, respiration and the metabolism of amino acids and proteins (Emberson, 2003). Accumulation of fluoride in plants has been associated with fluorosis in grazing animals (Patra *et al*., 2000).

Lead (Pb)

Animals and fish are mainly exposed to lead by breathing and ingesting it in food, water, soil, or dust. Lead accumulates in the blood, bones, muscles, and fat. Wild and domestic animals can ingest lead while grazing. They experience the same kind of effects as people who are exposed to lead. Lead can enter water systems through runoff and from sewage and industrial waste streams. Elevated levels of lead in the water can cause reproductive damage in some aquatic life and cause blood and neurological changes in fish and other animals. Low concentrations of lead can slow down vegetation growth (EPA, 2006c).

Mercury (Hg)

Mercury that reaches the ground through dry deposition is eventually washed into water bodies by rain. Bacteria in soils and sediments convert mercury to methylmercury. In this form, it is taken up by tiny aquatic plants and animals. Fish that eat these organisms build up methylmercury in their bodies. As bigger fish eat smaller ones, the methylmercury is concentrated further up the food chain. This process is called "biomagnification. Birds and mammals that eat fish are more exposed to methylmercury than other animals in water ecosystems. Similarly, predators that eat fish-eating animals are at risk (EPA, 2006a).

Effects of methylmercury exposure on wildlife can include mortality (death), reduced fertility, slower growth and development and abnormal behaviour. In addition, the endocrine system of fish, which plays an important role in fish development and reproduction, may be altered by the levels of methylmercury found in the environment (EPA, 2006a.

Benzene (C_6H_6)

Benzene can evaporate from contaminated water and soil surfaces. Once in the air, benzene reacts with other chemicals and breaks down within a few days. Benzene in the air can attach to rain or snow and be carried back down to the ground. Benzene in water and soil breaks down more slowly. Benzene is slightly soluble in water and can pass through the soil into underground water. Benzene in the environment does not build up in plants or animals.

In animals, exposure to food, water or air contaminated with benzene can damage the blood and the immune system and cause neurological effects, behavioural disturbances and cancer.
Studies with pregnant animals show that breathing benzene has harmful effects on the developing fetus. These effects include low birth weight, delayed bone formation, and bone marrow damage. Concentrations that may not be toxic to the mother may be toxic to the embryo and foetus (EBI, 2006).

Benzene can cause death in plants and roots and membrane damage in leaves of various agricultural crops (EBI, 2006).

Carbon Dioxide (CO_2)

Greenhouse gases allow sunlight, which is radiated in the visible and ultraviolet spectra, to enter the atmosphere unimpeded. When it strikes the Earth's surface it is absorbed and reradiated as infrared radiation. Some is reflected as infrared radiation (heat). Greenhouse gases tend to absorb this reradiated and reflected infrared radiation, trapping the heat in the atmosphere. Carbon dioxide (CO_2) is therefore referred to as a greenhouse gas because of its global warming potential.

Carbon dioxide occurs naturally in the atmosphere and is also produced in all combustion processes. It is uniformly distributed over the earth's surface. Carbon dioxide is released into the atmosphere when carbon-containing fossil fuels such as oil, natural gas and coal are burned in air, but also through the burning of forests and savannah. As a result of the tremendous worldwide consumption of such fossil fuels and deforestation, the amount of CO_2 in the atmosphere has increased over the past century (Figure. 1), presently rising at a rate of about 1 ppm per year. Major changes in global climate are related to the continued increase in CO_2 concentration, as it is one of the most important greenhouse gases.

Concentrations of CO_2 in the atmosphere are regulated by numerous processes, collectively known as the carbon cycle (Figure 2). Natural processes, such as plant photosynthesis and respiration, dominate the movement of CO_2 between the atmosphere and carbon sinks on land and in the oceans. While these natural processes can remove some of the net 6.6 billion metric

tons of anthropogenic CO_2 emissions produced each year, an estimated 3.3 billion metric tons of this carbon is added to the atmosphere annually in the form of CO_2.

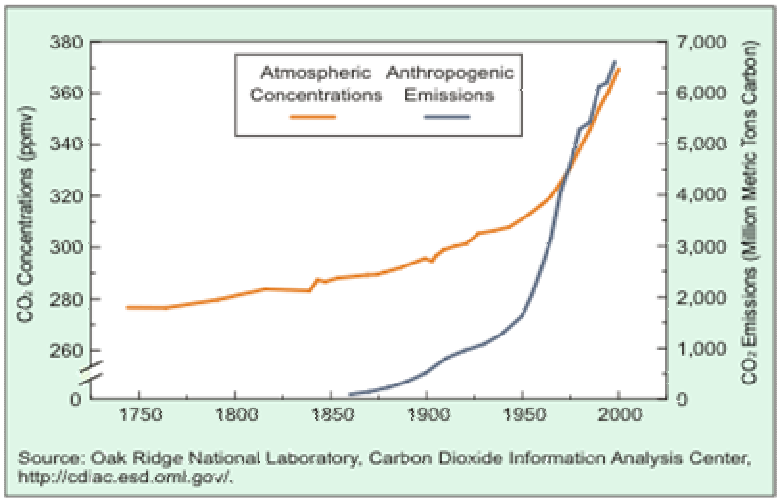

Figure 1: Trends in atmospheric concentrations and anthropogenic emissions of CO_2 (Source: EIA, 2006)

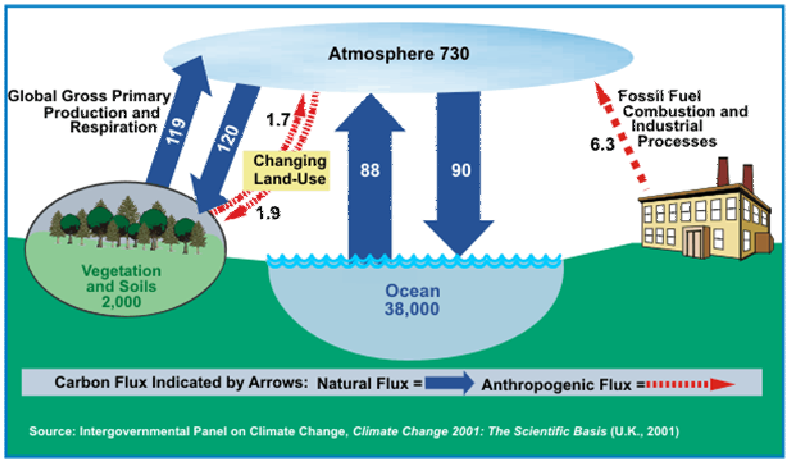

Figure 2: Global carbon cycle (billion metric tons) (Source: EIA, 2006)

Chlorofluorocarbons (CFCs)

The ozone layer in the stratosphere absorbs most of the sun's UV-A and UV-B radiation thus preventing harmful effects of the sun's ultraviolet radiation from passing through the Earth's atmosphere. The depletion of the ozone layer is caused by chlorine molecules in ozone depleting substances (ODS) that have been transported to the stratosphere which then react catalytically with ozone, thus destroying it (Ramacher *et al.*, 1997; Jobson *et al.*, 1994; Martinez *et al.*, 1999).

There has been a steady decline of the ozone layer at mid-latitudes and much larger losses over the poles. In both cases, the cause is catalytic destruction of ozone by atomic chlorine and bromine (McElroy *et al.*, 1986). The source of these atoms is the photodissociation of CFCs and Halon molecules transported into the stratosphere after being emitted at the surface. Chlorofluorocarbons (CFC) are haloalkanes with both chlorine and fluorine. They were formerly used widely in industry,

for example as refrigerants, propellants, and cleaning solvents. The CFCs and Halons are predominantly anthropogenic in nature and do not break down in the lower atmosphere. In this way, they can be transported into the stratosphere within 5 years. They are then decomposed into Cl/Br atoms by UV light. The environmental concern for CFCs follows from their long atmospheric lifetime (55 years for CFC-11 and 140 years for CFC-12, CCl_2F_2) (Elkins *et al.*, 1993) which limits our ability to reduce their abundance in the atmosphere and associated future ozone loss.

A variety of consequences which include increases in skin cancer, damage to plants, and reduction of plankton populations in the ocean's photic zone, may result from the increased UV exposure due to ozone depletion. Besides participating in the destruction of stratospheric ozone, the release of CFCs may also contribute to global warming, which means that CFCs influence the reflection of infrared radiation from the surface of the earth and thus cause global climate change (Dekant, 1996).

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