Health & Environmental Effects of Air Pollution

There have been many reports of death, illness and environmental damage caused by air pollution. This chapter describes the more important effects of major air pollutants and explains how they interact with each other and the environment.

Effects of Air Pollution on Human Health

In general specific sections of the public are most affected by air pollution. Those individuals already suffering from diseases of the cardiorespiratory system such as asthmatics and smokers are far more likely to be affected than those individuals who are healthy.

Figure 3.1 – The effect of smoking and pollution levels on the occurrence of cardiorespiratory disease in humans

Pollutants generally act on the surfaces of the respiratory system, causing chronic respiratory and cardiovascular disease. They may also alter important body functions such as oxygen exchange in the lungs, or oxygen transport in the blood. Irritant pollutants may lead to irritation and long term damage to eyes, nose, throat and other wet surfaces of the body. Deposition of particulate matter in the alveoli of the lung is quite serious as
removal can be slow. Soluble particles will be transferred to the blood. One of the most important particles man encounters in the atmosphere is H₂SO₄, which irritates the mucous membranes and leads to bronchial constriction. Most of the gaseous effects are more acute than chronic (as opposed to the particulate pollutants). SO₂, O₃ and NO₂ are all pulmonary irritants, and may lead to congestion, oedema and even haemorrhage. NO, H₂S and CO are asphyxiant gases. These combine with haemoglobin molecules to prevent oxygen transfer around the body. These molecules bind far more successfully to haemoglobin than does O₂. Many of the organic gas pollutants such as acrolein (1-propenal) as well as those gaseous pollutants mentioned above produce eye irritation.

Other chronic diseases related to air pollution include lung cancer, emphysema, chronic bronchitis and asthma. In addition air pollution can have serious effects on acute diseases such as the common cold.

In the following sections each major pollutant’s specific effects on human health and is examined. Synergistic effects are also considered.

**Interactions Between Pollutants and Synergism**

Ambient air is a complex mixture of gases and particulate matter. In such a mixture it is likely that there will be interaction between the components to modify the physiological effects of others. This may occur in several ways. For example, one pollutant may affect the site of deposition of another as is the case with sulfur dioxide. Due to its great solubility in water, sulfur dioxide is normally be removed in the upper respiratory system – where it has a corrosive effect. When sulfur dioxide is adsorbed/absorbed onto particles, it can however, be transported deep into the pulmonary system, where irritation and corrosion may occur. Thus, the particles may amplify the effect of sulfur dioxide. Also, harmful aerosols such as sulfuric acid can be produced by the interaction of gaseous pollutants in the warm moist environment of the lung.

Interaction between pollutants may result in effects that may be additive, synergistic, or antagonistic. Additive effects are those which occur when the exposure to several pollutants produces an effect equal to the sum of the effects of the pollutants acting alone. Synergistic effects are those where the sum of the effects of two or more pollutants is greater than the combined effect. Antagonistic effects refer to the situation where one pollutant lessens the effect of another pollutant. The great majority of pollutant effects are additive.

**Susceptible parts of the Human Body - The Respiratory System**

All forms of air pollution (gases, liquids and particulates) can damage human health. There are three main routes by which they enter the body:

- absorption through the skin
- ingestion, and
- inhalation.
Of these the latter is by far the most important as we exchange large amounts of atmospheric gas every day through our lungs. All forms of air pollutants can enter the lungs, but at this point it is probably appropriate to describe the structure of the human respiratory system, which allows us to exchange air with the atmosphere.

Figure 3.2 shows the details of the respiratory system. During breathing air is passed through the nose, then down the nasopharynx (a space at the back of the nose) and into the Trachea (windpipe). From here it is passed into the lungs which consist of a series of pipes (the bronchi and bronchioles) which become thinner and thinner until they reach the small air sacs where gas exchange takes place. These are the alveoli, which appear like a bunch of microscopic grapes. They have a very fine membrane that separates the blood from the air, which is where gas exchange ($\text{O}_2$ uptake and expulsion of $\text{CO}_2$) occurs.

All of the air passages have a coating of mucus and are also lined with tiny branch like structures called cilia, which constantly move in an upward fashion to remove any solid material that enters the lungs.
Injury to the respiratory tract occurs when chemicals enter the lungs and cause direct damage to the lung tissue, or when the lungs trap material and allow it to be transferred to the bloodstream where it may affect any organ in the body.

**Carbon Monoxide**

High levels of carbon monoxide are a major concern, since the gas preferentially and irreversibly binds to haemoglobin in the blood forming carboxyhaemoglobin. The net effect is thus a reduction in the blood's capacity to carry oxygen. Carbon monoxide has a
higher affinity (200 times greater) for haemoglobin than O\textsubscript{2}, and also tends to remain more tightly bound.

The first discernible effect of CO at around 10ppm is a reduction in awareness, a probable cause of many car accidents. At around 100ppm, headaches and drowsiness set in. Loss of consciousness and death require levels of 250ppm and over. There is now also some medical evidence to suggest that continued exposure to low levels of CO may cause nervous disorders and be a factor in the cause of heart disease.

Carbon monoxide is highly toxic at concentrations in excess of 1000ppm. Death results from anoxia since body tissues, especially the brain, are deprived of an adequate O\textsubscript{2} supply. Because it is colourless, odourless and tasteless, individuals exposed to toxic concentrations are unaware of its presence. Since elevated CO levels are produced in a variety of circumstances or activities, CO deaths are not uncommon.

The concentrations of CO normally encountered in urban environments are usually only a fraction of those levels that cause asphyxiation.

When a human or animal is exposed to CO, the amount of carboxyhaemoglobin produced in the blood is directly related to the CO dose. For example, exposure to 35ppm CO for 1 hr, or 9ppm for 8 hr, will result in 1.3% carboxyhaemoglobin saturation. These exposure levels represent the air quality standards for CO. No adverse biological effects have been shown for such exposure levels, and as a consequence they are assumed to be safe.

The effects of CO exposures can be related to carboxyhaemoglobin levels. Table 3.1 shows the response to different levels of CO. Most low level effects are central nervous system (CNS) related, such as behavioural changes, decreased time interval discrimination, impairment of brightness discrimination, increased reaction time to visual stimuli, and lowered performance in driving simulations. It is thought that the latter may be the cause of many motor vehicle accidents in peak hour traffic of cities where peak hour CO levels may rise above 50ppm.
### Table 3.1 – The effects of CO on human health

<table>
<thead>
<tr>
<th>Concentration of CO (ppm)</th>
<th>Physiological Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>Lowered awareness and driving performance</td>
</tr>
<tr>
<td>50 - 100</td>
<td>Headaches and drowsiness, changes in driving performance and increased reaction time to visual stimulation</td>
</tr>
<tr>
<td>&gt;250</td>
<td>Death</td>
</tr>
</tbody>
</table>

In addition to CNS effects, CO exposures have the potential for contributing to or exacerbating cardiovascular disease. Animal studies indicate that CO exposures may be related to arteriosclerotic heart disease (AHD), or hardening of the arteries. Arteriosclerotic heart disease accounts for about 35% of all deaths and is a major cause of morbidity (illness) as well. It is characterised by a severe diffuse narrowing of the coronary arteries.

Ambient exposures to CO may be a direct cause of increased blood carboxyhaemoglobin levels. It may also elevate levels that are already high from other exposures (e.g. cigarette smoking, cooking and heating, seepage into motor vehicle interiors during normal driving, etc.). The health effects of ambient CO cannot be dissociated from the effects of other CO exposures. For cigarette smokers, CO exposures are far more significant (an average 3 – 8% carboxyhaemoglobin saturation) than those experienced under urban ambient conditions. Additive effects of ambient CO could further exacerbate this considerable health burden.

Reduction in the levels of CO in car emissions have been associated with changes in the design of the exhaust system, whereby oxidation catalysts, such as Pd and Ru, promote further reaction with excess air to carbon dioxide. Uncontrolled emissions average 3.5% CO: current design rules limit emission to 1.0%. Under specified testing procedures, this is equivalent to 24.2 g/km.

#### Sulfur Compounds

Sulfur compounds include the very corrosive sulfur dioxide and sulfur trioxide, and hydrogen sulfide.

Sulfur dioxide and sulfur trioxide produce very similar effects as both are very corrosive, whilst hydrogen sulfide produces similar effects to CO in that it can lead to anoxia when present in high concentrations – a situation which is very rare in ambient air.
**Sulfur Dioxide and Particulates**

Analysis of the effects of sulfur dioxide are complicated by the fact that it is often associated with particulate pollutants – so the direct effects of each individual pollutant are difficult to separate. They are often produced by a common source, such as the combustion of coal, hence high SO\(_2\) levels are often associated with high particulate matter levels often forming sulfate aerosols.

Sulfate aerosols present a more significant threat to health than do sulfur dioxide emissions alone. These are produced when acidic particles are formed by the reaction of water with ash and SO\(_2\), to give SO\(_4^{2-}\) and H\(^+\). Particles from aerosols are just the right size to be retained in the lungs so cause maximum physiological damage.

There is also some scientific evidence to suggest that particulate matter exacerbates the toxic response to SO\(_2\). Studies of acute air pollution episodes in the United States and Great Britain have indicated an association between increased mortality and elevated levels of combined SO\(_2\) and particulate matter. Epidemiological studies of longer-term, lower-level exposures have also shown an association with certain adverse health effects, such as significantly higher mortality from bronchitis and lung cancer.

**Sulfur Dioxide**

Sulfur dioxide in the atmosphere has its primary effect on the respiratory tract, producing irritation and difficulty in breathing. It affects most strongly those people who already have respiratory problems. Children are known to suffer increased frequency of infection upon prolonged exposure to sulfur dioxide – but true long term effects of exposure are not well understood. The acute effects of sulfur dioxide on humans are listed in Table 3.2.

Table 3.2 – Acute effects on humans of different atmospheric SO\(_2\) levels

<table>
<thead>
<tr>
<th>[SO(_2)] in ppm</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>500</td>
<td>Lowest level of human sensation</td>
</tr>
<tr>
<td>800</td>
<td>Threshold of taste</td>
</tr>
<tr>
<td>1400</td>
<td>Threshold of odour</td>
</tr>
<tr>
<td>4400</td>
<td>Threshold for reversible bronchial constriction</td>
</tr>
<tr>
<td>20000</td>
<td>Immediate throat irritation</td>
</tr>
<tr>
<td>30000</td>
<td>Immediate eye irritation</td>
</tr>
<tr>
<td>50000</td>
<td>Immediate coughing</td>
</tr>
</tbody>
</table>

To put this in perspective, the London smog of 1952 averaged about 4000-5000ppm. Street levels in Wollongong in the late 1970’s recorded values of 2250 ppm – probably due to smelting operations in the area.

Because of its solubility in water, SO\(_2\) is almost entirely removed in the mouth, throat, and nose through normal breathing. Less than 1% of inspired SO\(_2\) reaches the lung tissue (alveoli). Although exposure of the lower airways and alveoli to SO\(_2\) increases
considerably during exercise, the principal effect of SO\textsubscript{2} exposure is to alter the mechanical function of the upper airway.

Laboratory animal exposures at very high concentrations indicate that SO\textsubscript{2} has the potential for causing chronic bronchitis.

Studies on humans and animals report an exaggerated air flow resistance in response to short-term acute exposures in about 10\% of the apparently normal population under test. Human challenge studies have further demonstrated that SO\textsubscript{2} exposures at relatively low levels (0.25 and 0.5ppm) could produce acute bronchoconstriction on inhalation by strenuously exercising asthmatic subjects. The mechanism of these apparently non-allergenic hypersensitive responses is unknown.

The health consequences of ambient SO\textsubscript{2} exposures may only partly lie in the direct effects of this gas on the human respiratory system. It is more likely that health effects attributed to SO\textsubscript{2} are due to the highly irritant effects of sulfate aerosols, such as sulfuric acid, which are produced from SO\textsubscript{2}.

**Oxides of Nitrogen**

Nitric oxide is a relatively nonirritating gas and is thought to pose little threat to health at normal ambient levels. Its importance lies in the fact that it is rapidly oxidised to NO\textsubscript{2}, a gas of much greater toxicity.

Whilst SO\textsubscript{2} is rapidly absorbed in the fluids in the upper respiratory tract, NO\textsubscript{2} is less soluble and generally penetrates deep into the lung leading to tissue damage. Exposure to acute high levels leads to effects such as pulmonary oedema.

The lowest NO\textsubscript{2} exposure level shown to cause physiological change is 0.5ppm at which adverse effects included the destruction of cilia, alveolar tissue disruption, and obstruction the respiratory bronchioles. Exposures at higher levels can lead to severe tissue damage. Minor respiratory problems are caused by brief exposures at levels below 5ppm. At levels below 100ppm, exposure leads to prolonged, but non fatal inflammation of lung tissue. Higher levels are generally toxic. There is also evidence that NO\textsubscript{2} can damage respiratory defence mechanisms, allowing bacteria to proliferate and invade lung tissues.

Another health problem linked to nitrogen oxides is that they are precursors for pollutants that form photochemical smog.

**Hydrocarbons**

Most hydrocarbons are relatively nontoxic at the ambient levels found in normal atmospheres. They are however key reactants in photochemical smog formation. Where they undergo complicated reactions in the atmosphere with O\textsubscript{2}, O\textsubscript{3}, NO\textsubscript{x}, SO\textsubscript{x} and other components to form photochemical smog (see following section) which is very
deleterious to human health. They reduce visibility, have unpleasant odours and cause skin and eye irritation at higher levels. Some are carcinogenic, such as benzo[α]pyrene.

Hydrocarbon air quality standards are not based on the health effects of hydrocarbon chemicals, but rather an attempt to reduce photochemical smog formation.

Hydrocarbons that may be of concern to human health include the carcinogenic polycyclic aromatic hydrocarbons (PAH), such as benzo(α)pyrene (BaP), and the eye irritants, including HCHO (methanal), acrolein, and peroxyacetyl nitrate (PAN). The PAHs are produced as a result of the incomplete combustion of high-molecular-weight hydrocarbon species. Benzo(α)pyrene is the most abundant PAH in urban air. It is normally found adsorbed on aerosol particles. Controlled animal studies indicate that exposures in excess of ambient levels may be required to induce tumours.

The PAH levels of urban areas in the United States have been reduced by a factor of three since the mid-1950s. This decrease has been due to a change of fuel usage from coal to natural gas and oil for home heating. If the PAHs in urban areas were contributing to the urban excess of lung cancer, then the decrease in PAH should have resulted in a decrease in lung cancer attributed to the "urban factor." There is no evidence that this has occurred.

Hydrocarbon derivatives such as HCHO, acrolein, and PAN are of interest because they contribute to the eye irritation experienced in photochemical smog. The threshold for HCHO-induced eye irritation is in the range 0.1 to 1 ppm; for acrolein, moderate eye irritation can be detected at 0.25 ppm for PAN, 20 ppb. In general, eye irritation experienced in urban areas is likely to be produced by the combined effects of these substances and other hydrocarbon derivatives.

**Ozone & Photochemical Smog**

Ozone is one of the most toxic pollutants regulated under ambient air quality standards. It may cause significant physiological and pathological changes in both animals and humans at exposure concentrations that are within the range of those measured in polluted ambient environments. The ambient air quality standard for O₃ is 0.12 ppm (235 mg/m³) averaged over 1 hour. The short exposure duration for this standard is based on the historical regulatory assumption that O₃ levels have a tendency to peak at mid-day and that these peaks are primarily responsible for health risks associated with ambient O₃ exposures.

O₃ may cause significant lung function changes even with exposures in the 0.10-0.40 ppm range of for 1-2 hours. The changes are transient; and lung function appears to return to normal after the exposure is stopped. Lung function changes are concentration dependent and increase with increasing depth of breathing. Such changes have been reported for healthy adolescents and young adults. There is no evidence, however, that smokers, older adults (over 55), asthmatics, or individuals with chronic obstructive lung disease are more responsive to O₃ exposures.
Exposure to O₃ levels above 0.12ppm, may lead to a variety of symptoms including throat dryness, chest tightness, coughing, pain whilst deep breathing, shortness of breath, lassitude, malaise, headache, and nausea. O₃ exposures also cause decreases in athletic performance.

O₃ exposures may inhibit the immune system's ability to defend the body against infection. A single 3-hour exposure at concentrations of 0.08-0.10ppm has been reported to increase the susceptibility of mice to bacterial infection, and increased O₃-associated mortality occurred at the elevated levels commonly reported in Los Angeles.

Studies of prolonged exposures to O₃ indicate progressive changes in respiratory function, a marked increase in nonspecific airway reactivity, and progressive changes in symptoms. Repetitive daily exposures can also have significant effects. Repetitive exposures result in an enhanced response on the second day with diminishing responses on days 3 and 4 and virtually no response on day 5. This apparent adaptation persists about a week after exposure ceases. Animal exposure studies indicate that despite the fact that functional adaptation takes place, progressive and persistent damage to lung tissue continues to occur. Damage may include necrosis of cilia cells, cilia shortening and damage to the bronchioles.

Studies of chronic human exposures to ambient air indicate that there appears to be a functional adaptation that persists several months after the O₃ season, returning to normal by the following spring. Other studies indicate that chronic exposures to O₃ in community air may result in a premature aging of the lungs.

Studies on microorganisms, plant root tips, and tissue cultures indicate O₃ is mutagenic, that is, it can cause heritable changes in genes. Mutagenic substances have a high probability of being carcinogenic as well.

**Particulate Matter**

Particulate matter exhibits toxic effects due to the direct irritant action of particles (such as sulfuric acid) and substances that are readily adsorbed to the large surface area of small particles. The concentration of adsorbed substances may be considerably greater than in the ambient atmosphere. Adsorbed substances of particular concern include SOₓ, polycyclic aromatic hydrocarbons (PAH), and heavy metals such as lead, cadmium, zinc and mercury. The PAH and heavy metals tend to predominate in the small respirable particles.

**Respiratory Deposition and Retention of Particulate Matter**

The health consequences of atmospheric particulate matter depend on its ability to penetrate respiratory defence mechanisms. These are adequate to remove inhaled particles in excess of 10µm, but particles smaller than can enter and be deposited in the respiratory system. These are described as "inhalable particles". Particles less than 2.5µm are called "respirable"; they can enter pulmonary tissue and be deposited there. Particles
larger than 2.5µm are removed in the upper respiratory system. Deposition of particles as a function of particle size is illustrated in Figure 3.3.

![Deposition of particles in respiratory tract according to particle size](image)

**Figure 3.3 – Deposition of particles in respiratory tract according to particle size**.

Because of changes in flow patterns in the tracheobronchial zone, particles tend to be deposited at or near airway intersections. As nerve endings are concentrated at these sites, the mechanical stimuli of deposited particles often lead to reflex coughing and bronchoconstriction. This leads to an increased breathing rate.

The deposition of particles is not only influenced by particle size but also by mass concentration, molecular composition, pH and solubility. Deposition also varies among non-smokers, smokers, and individuals with lung disease. Tracheobronchial deposition is slightly higher in smokers and greatly increased in individuals with lung disease.

After particles have been deposited, their retention may be a function of the rate of clearance, which varies greatly among the different regions of the respiratory tract. In the ciliated airways of the nose and upper tracheobronchial zone, clearance in healthy individuals is achieved in less than a day. As the site of deposition becomes deeper in the lungs the time required for clearance greatly increases. Clearance of particles in the alveolar region may take weeks to months. Slow clearance of particles from the respiratory system of humans is generally considered to be detrimental since toxic substances are in contact with sensitive tissue for longer periods of time.
Alveolar deposition of particles is most efficient in the range of 0.1 to 2.5\(\mu\)m. The effective toxicity of these small particles may be greater than that of larger particles since the concentrations of toxic substances such as lead, zinc, chromium, mercury, sulfates, nitrates, etc., increase with decreasing particle size. Additionally, the enormous surface area of small particles allows for higher reaction and dissolution rates for toxic chemical species. Their relatively long retention in the alveolar region permits substances such as lead to be extracted and transported to other parts of the body\(^1\).

Particulate matter may contribute to the development of chronic bronchitis and may be a predisposing factor to acute bacterial and viral bronchitis, especially in smokers and children. It may also aggravate bronchial asthma and the late stages of chronic bronchitis and pulmonary emphysema. Health effects of particulate matter may also depend in large measure on synergistic effects with pollutants such as \(\text{SO}_2\)\(^1\).

Studies of the effects of particulate materials in the atmosphere are shown in Table 3.3.

### Table 3.3 – The effect of levels of TSP on human health

<table>
<thead>
<tr>
<th>Total Suspended Particulate Level ((\mu)g/m(^3))</th>
<th>Effect(^1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1000</td>
<td>Increased mortality</td>
</tr>
<tr>
<td>250 – 500</td>
<td>Aggravation of bronchitis</td>
</tr>
<tr>
<td>200</td>
<td>Small reversible changes in lung function of children</td>
</tr>
</tbody>
</table>

There is now strong evidence that acid aerosols composed of \(\text{H}_2\text{SO}_4\) and \(\text{HNO}_3\), have significant health effects. Exposures to these at concentrations slightly above ambient levels significantly decrease the ability of the upper respiratory tract to remove potentially harmful particles. Acid aerosols also appear to be able to induce asthmatic attacks in exercising asthmatic adolescents at near-ambient concentrations.

**Lead**

Depending on the level of exposure, individuals may develop symptoms of acute or chronic lead poisoning. The most affected organs are the blood, the brain, the kidney, the nervous system and the reproductive system.

Symptoms of acute lead poisoning include shock, anaemia, nervousness, and irreversible kidney and brain damage.

Episodes of acute lead poisoning are rarely associated with ambient atmospheric lead levels – even in lead smelters. Atmospheric lead is normally a chronic cumulative poison, mostly affecting the central nervous system. Lead is found at highest concentrations in particles of 0.2\(\mu\)m or less, which increases its access to animals by allowing access to the deep lung tissue where it is readily absorbed into the blood and transferred to other
organs. In growing children, lead affects intelligence, length of concentration and activity. Pregnant women are also especially susceptible to the effects of the metal. This is because lead crosses the placenta, resulting in high lead levels in the foetus, which may lead to mental retardation.

Lead has been shown to interfere with the maturation and development of red blood cells. It does so by inhibiting enzymes involved in haemoglobin synthesis. Blood lead is much higher in smokers than non-smokers. Atmospheric lead does not lead to acute intoxication, but rather is a chronic problem.

Asbestos
The name asbestos refers to number of minerals which are compressed together to form fibrous materials. Particulate asbestos is a considerable community health problem due to its past use as a construction and insulating material. Medical evidence suggests that inhalation of the fibres causes the lung disease asbestosis – which is characterised by scarring of the lower lungs and lowered breathing function. It is a common disease in those exposed to asbestos for long periods. Some forms of asbestos are responsible for causing lung cancer and mesothelioma (cancer of the lining of the body cavity). Asbestos fibres are chronic toxins with very long latency periods. Once the fibre is inhaled it has the potential to cause cancer for the rest of the individuals life. It is estimated that between 25-50% of all individuals have asbestos in their bodies.

Fluoride
Gaseous fluorides damage vegetation and if ingested in sufficient quantities the bones and teeth of animals eating these plants.

Environmental Effects of Air Pollutants
When developing standards for the assessment of air pollution the emphasis has always been on controlling the effects on human beings, with all other effects being treated as of much lesser importance. Air pollutants do have significant effects on plants, buildings and other animals and these should also be considered when developing standards to assess environmental harm from air pollution.

Effects on Plants
Plants are often the first to show damage associated with increased ambient levels of air pollution. The effects of SO$_2$, HCl and HF have been reported as early as the middle of the 19$^{th}$ century. The most severe damage seems to have been associated with high levels of SO$_2$ and heavy metal particulates associated with mining and smelting, but many other pollutants such as NO, Cl and NH$_3$ are now know to cause direct toxic effects on plant tissue.

The problems of acid rain and photochemical smog were first suggested as major environmental problems by their actions on plants.
Air pollutants may injure plants in several ways— with some effects being subtle, but others clearly visible. Visible effects normally involve identifiable changes in the leaf structure such as chlorophyll destruction (chlorosis), tissue death (necrosis) and pigment formation. Subtle effects include inhibition of growth and lowered rates of photosynthesis.

Plant tissue damage may be either acute or chronic. Acute exposures to high levels of pollutants generally lead to necrosis. Chronic injuries result from intermittent or long term exposures to lower levels of pollutants, with chlorophyll destruction or chlorosis being the most common symptoms of injury. Different pollutants produce differing symptomatic effects on plants and this can be used as an indicator as to the possible presence raised levels of certain pollutants. These are discussed in more detail in the paragraphs below.

**Sulfur Dioxide**
Atmospheric sulfur dioxide is harmful to certain plants at levels below 1ppm, causing tissue damage and destruction of chlorophyll. However, it is through chemical reactions in the atmosphere which produce sulfuric acid, that sulfur dioxide emissions have caused their most significant damage. In conjunction with the nitric acid produced from nitrogen oxide emissions, the phenomenon known as acid rain has transformed ecosystems throughout Europe and North America, destroying forests and increasing acidity levels in lakes by several pH units.

Sulfur dioxide causes damage to plant tissues by entering through stomates where it immediately comes into contact with the spongy mesophyll cells of the leaf. Initially the damage is restricted to the near vicinity of the pore, but as exposure continues, there is a progressive expansion of injury and tissue collapse (see Figure 3.4). As the injury develops, affected tissue may have a greyish green water-soaked appearance, which on drying becomes ivory-to-white, red, brown or even black, depending on the species. The injury extends from the bottom to the top of the leaf and is visible on both surfaces.

The severity of the injury, that is, the amount of leaf tissue affected, is dependent on the dose to which the plant has been exposed. The greater the dose or exposure, the more severely injured are individual leaves as well as the whole plant. The entire leaf, or in some extreme cases the entire plant, may be killed.

The severity of observed injury on individual leaves is also dependent on leaf maturity. Young, fully expanded leaves are most sensitive to SO₂. Older leaves and those not fully expanded are much less sensitive.

In dicots, acute SO₂ injury is usually manifested as an interveinal necrosis, although necrosis in some species may appear on the margins. In some instances, chlorosis may be associated with the necrotic areas. In monocots, such as grains, lilies, and gladioli, injury may appear as irregular necrotic streaks between the larger veins on both surfaces of the leaf. At low to moderate concentrations, SO₂ may cause leaf tip injury on monocots.
Sulfur dioxide injury on conifers is observed as reddish brown to brown tip necrosis that may have a banded appearance from repeated exposures.

Different species and varieties vary in their sensitivity to SO$_2$ exposure. Alfalfa is the most sensitive species, with acute injury observed at a dose of 1ppm for 1hr. Because of its hypersensitivity, alfalfa has been used as a bioindicator of SO$_2$ levels in the ambient atmosphere.

![Injured Tissue](image)

**Figure 3.4 – Tissue injury symptoms associated with exposure to high levels of SO$_2$**

**Ozone**
Ozone also enters the leaf through the stomates on the lower leaf surface. In dicots it bypasses the spongy cells and injures the palisade mesophyll cells preferentially (see Figure 3.5). Due to this the symptoms of acute injury are visible on the upper leaf surface. In monocots, conifers, and dicots without a palisade mesophyll, cells near the stomates are injured, with symptoms appearing on either or both surfaces. Developing leaves ranging from 65-90% of their full size are most sensitive to O$_3$ injury. As with SO$_2$ the maturity of the plant determines sensitivity, with younger plants being more sensitive and older plants more resistant.
The most common O$_3$-induced symptom patterns observed on dicots are upper surface flecks (see Figure 3.6). These flecks are produced when groups of palisade cells are killed or injured, resulting in chlorotic or necrotic lesions. The adjacent epidermal cells may in some cases also collapse. The flecks may be white, tan or yellow in color. If flecking is extensive the upper leaf surface may appear bronzed or chlorotic. A variation of this symptom is called stipple. In stipple, the injured palisade cells may become intensely pigmented, giving the leaf surface a red to purple to black to brown appearance. The collapse of the overlying epidermal cells is generally not observed with the stipple symptom. Flecking is generally a more severe reaction than stipple, but it is apparent that these symptoms are more related to plant species than to ozone dose. In both the fleck and stipple symptom patterns, injury is confined to the interveinal tissues, the veins being uninjured.
Ozone sensitivity varies from species to species and from variety to variety within species. Ozone is more toxic to plants than SO\textsubscript{2}. Symptoms may be observed on sensitive plants from exposures of as little as 0.10 - 0.30ppm for a few hours.

Ozone is considered by plant scientists to be the most important of all of the phytotoxic air pollutants. It has been estimated to be the cause of over 90% of all plant injury due to air pollution in North America.

**Peroxyacyl Nitrate (PAN)**

Peroxyacyl nitrate is the causal agent of "smog injury" observed on vegetable crops. PAN enters the leaf through the stomate, then reacts immediately with the spongy mesophyll around the stomate. This causes the stomate to collapse, forming large air pockets that lead to a glazing/browning appearance on the lower surface of the leaf. Patterns of banding may also be observed with PAN exposures. These bands occur in the same location on leaves of the same age. PAN injury often appears as bands at the apex of the youngest sensitive leaf, the middle of an intermediate-aged leaf and the base of the oldest sensitive leaf. Young, rapidly developing leaves on young rapidly growing plants are most sensitive to PAN.

In monocots, such as grasses, glazing and bronzing rarely occur. PAN injury usually appears as distinct bands several millimeters to 2 cm wide across the leaf blade. If the injury is slight the injured tissue may be chlorotic. Severe injury results in tissue collapse and bleaching of the necrotic tissue.
Peroxyacetyl nitrate is more toxic to plants than O\textsubscript{3}. Sensitive plants such as petunia, tomato, and lettuce may be injured by an exposure to as little as 15-20 ppb for a few hours\textsuperscript{1}.

**Fluorides**

Fluoride can have substantial effects on growing plant tissue. Injury may result from the uptake of gaseous hydrogen fluoride through the stomates or from soluble particulate fluorides absorbed through the leaves and/or roots. Regardless of the means of entry, fluorides enter the veins and are transported to leaf margins and/or the leaf tip, where they accumulate. Fluoride injury generally results from the accumulation of small quantities of fluoride over long periods. The severity of the injury is directly related to the dose absorbed / level of exposure.
Figure 3.8 – Tissues of a leaf affected by fluoride exposure

Fluoride injury normally appears as a marginal or tip necrosis (commonly called “tip burn”). The injury occurs in both the mesophyll and the epidermis. There is often a narrow reddish band between the living and dead tissue.

Typical fluoride-induced injury on selected species is illustrated in Figures 3.8 and 3.9. Some sensitive plant species (such as iris, wheat, tulip and gladiolus) can be injured by fluoride exposures of only a few ppb for a 1 week period.
Figure 3.9 – Symptoms of a typical leaf exposed to fluoride for an extended period
**Particulate Materials**
Particulate dusts may cause injury to vegetation both directly and indirectly.

Direct effects such as those for cement kiln dust include a variety of plant responses, such as
- reduction in yield and growth without visible injury,
- increase in disease incidence,
- severe injury to leaf cells,
- suppression of photosynthesis, and
- death of trees.

The injury may result from the deposition of a thick crust that suppresses photosynthesis and/or alkaline toxicity when alkaline solutions are produced when dusts are present in free moisture. Ecological studies of forest communities affected by the deposition of limestone dusts have shown significant changes in tree growth and species composition. Changes in dominant tree species and the decrease in herbaceous vegetation are particularly evident.

There are indirect beneficial effects from soil neutralisation by alkaline dusts. Atmospheric aerosols near urban areas may significantly elevate lead levels in both soil and vegetation. The uptake of lead by roadside plants results in no apparent visible injury. Heavy metal aerosols generated by nonferrous metal smelters may, however, have significant plant effects. The severe devastation of vegetation and denudation of the landscape around smelters is likely due to aerosol-derived heavy metal contamination of the soil and subsequent accumulation of phytotoxic levels by plants. There is evidence of this in the hills around Queenstown in Tasmania. This heavy metal problem is exacerbated by high concentrations of SO$_2$, which occur simultaneously with metal aerosol emissions. Growth of vegetation around smelters that have ceased operation is still suppressed for many years after their closure.

**Acid Deposition**
Acidic particles which fall onto plants or soil in which they grow can cause significant changes in plant wellbeing. These can result in necrotic lesions of the upper leaf surface, which is followed on subsequent exposures by tissue collapse on both surfaces.

An indirect effect is the deposition onto soil, which affects soil pH. The effects here may be positive or negative depending on the make up of the soil.

**Effects of Air Pollutants on Buildings and Materials**
Air pollution is renowned for soiling building surfaces, clothing and other articles. This is generally a result of smoke particles adhering to the surface in question, but their are many other more sinister effects.

Air pollutants significantly affect many non-biological structures and materials. These effects cause economic losses in billions of dollars each year. Most important are effects
on metals, carbonate building stones, paints, textiles, fabric dyes, rubber, leather, and paper. In Western Europe, which is a repository for many monuments of history and fine works of art, air pollutant-induced damage has been incalculable. Because these cultural treasures are irreplaceable, their preservation from the destructive effects of airborne contaminants poses a significant challenge to scientists.

Materials can be affected by both physical and chemical mechanisms. Physical damage may result from the abrasive effect of wind-driven particulate matter impinging on surfaces and the soiling effect of passive dust deposition. Chemical reactions may result when pollutants and materials come into direct contact. An example of this the reaction of lead in older paint materials with sulfurous materials (particularly H₂S) from air pollution to produce the unsightly lead sulfide black streaks on buildings.

**Metal Corrosion**
Metal corrosion in industrialised areas represents one of the most costly effects of atmospheric pollutants. Since corrosion is natural we tend not to recognise the role that pollutants play in accelerating this process. As the ferrous metals, iron and steel, account for about 90% of all metal usage, pollution-induced corrosion on these metals is of particular significance. The acceleration of corrosion in industrial environments is associated with high levels of atmospheric SO₂ and particulate matter pollution is very significant. Oxidants, such as ozone, inhibit the effects of SO₂ by producing a more corrosion-resistant product.

**Rubber and Fabrics**
Another important material damaged by pollution is rubber, which is attacked by ozone. This leads to cracking, which is an economically significant problem. Fabrics such as nylon are also affected by air pollution. These tend to disintegrate upon prolonged exposure to the air. Bleaching and discolouration may also occur.

**Accepted Levels of Major Air Pollutants**
These vary significantly from country to country, so the values listed in Table 3.4 should only be used as guidelines. For the most up to date information on accepted pollutant levels you should consult the NSW EPA.
Table 3.4 – Accepted levels of major air pollutants

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<th>Air Pollutant</th>
<th>Acceptable Level</th>
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| **CO**                        | 1 hour ave. 30ppm (60ppm detrimental)  
|                               | 8 hour ave. 9ppm (20 ppm detrimental)   
|                               | 1 hour alert level 150ppm                                                        |
| **NO₂**                       | 1 hour ave. 0.12ppm (0.25ppm detrimental)  
|                               | 8 hour ave. 0.06ppm (0.15 ppm detrimental)  
|                               | 1 hour alert level 0.50ppm                                                       |
|                               | 1 year 0.03ppm                                                                   |
| **NH₃**                       | Ground level conc. 0.83ppm (0.6 mg/m³)                                            |
| **HNO₃**                      | Ground level conc. 0.067ppm (0.17 mg/m³)                                          |
| **SO₂**                       | 1 hour ave. 0.20ppm (0.34ppm detrimental)  
|                               | 8 hour ave. 0.06ppm (0.11 ppm detrimental)  
|                               | 1 day ave. 0.08ppm                                                               |
|                               | 1 year ave. 0.02ppm                                                             |
|                               | 1 hour alert level 0.50ppm                                                       |
| **H₂S**                       | Ground level conc. 0.0001ppm (0.00014 mg/m³)                                     |
| **Photochemical oxidants**    | 1 hour ave. 0.10ppm (0.15ppm detrimental)  
| (as O₃)                       | 4 hour ave. 0.08ppm (0.15ppm detrimental)  
|                               | 8 hour ave. 0.05ppm (0.08 ppm detrimental)  
|                               | 1 hour alert level 0.25ppm                                                       |
| **Respirable particles**      | 24 hour ave. 120 mg/m³ (240mg/m³ detrimental)  
|                               | 1 year ave. 40 mg/m³ (80mg/m³ detrimental)                                        |
| **PM₁₀ Respirables**          | 1 day ave. 50µg/m³                                                               |
| **Atmospheric Lead**          | 3 month ave. 1.0 µg/m³                                                            |
|                               | 1 year ave. 0.50µg/m³                                                            |
| **Benzo[α]pyrene**            | 1 year ave. 5.0ng/m³                                                             |
| **Benzene**                   | 1 year ave. 10.0ng/m³                                                            |
| **Fluorine**                  | Ground level conc. 0.033ppm (0.067 mg/m³)                                        |

**References**