Air Pollution: A Fatal Attack on Cardio-Respiratory System

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Abstract

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. In spite of the great medical advances, cardio-respiratory disease remains one of the major causes of mortality worldwide, especially in industrialized countries. It develops as a result of countless complex interactions between genetic factors such as those related to age, sex, family history, weight, and post-menopausal status in women; and to environment-related factors such as cigarette smoking, alcohol use, eating habits, physical activity, and others. For more than a decade, several epidemiological studies have demonstrated the existence of a consistent association between air pollution and increased risk for cardio-respiratory events, that is, not only death, but also acute myocardial infarction and arrhythmias. However, some effects resulting from the exposure to different air pollutants have been evidenced and the most significant of which involve pulmonary and systemic inflammatory response, blood clotting disorders, promotion and potentiation of the atherosclerotic process, and cardiac autonomic dysfunction. Air pollution is a major environmental health problem affecting the developing and the developed countries alike. The effects of air pollution on health are very complex as there are many different sources and their individual effects vary from one to the other. It is not only the ambient air quality in the cities but also the indoor air quality in the rural and the urban areas that are causing concern. In fact in the developing world the highest air pollution exposures occur in the indoor environment. Air pollutants that are inhaled have serious impact on human health affecting the lungs and the respiratory system; they are also taken up by the blood and pumped all round the body. These pollutants are also deposited on soil, plants, and in the water, further contributing to human exposure and hence prove fatal to human health.

Keywords: Air pollution, fatal effects, cardio-respiratory disorders, human health.

INTRODUCTION

Since the first half of the 20th century, air pollution has been a serious problem in industrialized urban centers. Episodes of excessive air pollution caused increased number of deaths in some cities of Europe and the United States, which made the world’s scientific community turn their eyes to this issue, and brought about questions on the adverse effects on human health, thus leading to the search for answers. One of the first and well-documented episodes occurred in a small industrial valley of Meuse River, Belgium. From December 1 to 5, 1930, heavy smog caused more than 60 deaths, a mortality rate ten-fold higher than normal, and hundreds of people developed respiratory diseases frequently accompanied by worsening of heart failure (Pope, Dockery & Schwartz, 1995). In October 1948, there was the Donora event (Helfand, Lazarus, Theerman, 2001), also...
an industrial valley in Pennsylvania, where combustion by-products from local industries remained over the town due to the occurrence of temperature inversions that hindered their dispersion, and caused approximately 14,000 residents to experience some type of clinical sign; 400 hospitalizations and 20 deaths were recorded. However, the most dramatic event occurred in London, England. In the winter of 1952, the impossibility of dispersion of a stagnant air mass with high concentrations of sulfur dioxide and ambient particulate that lasted four to five days, resulted in approximately 4,000 deaths, and although respiratory causes had been accounted for the relative increase in mortality, the number of deaths from cardio-respiratory causes was quite significant, from then on, it was clear that both lung and heart are affected by pollution.

Aware of the London event, the US Congress, in 1955, released approximately five million dollars for studies on the health and economic impact of air pollution. In Europe, in 1956, the English Parliament assigned to the local authorities the task of controlling the areas at greater risk of accumulation of black smoke emitted by the houses, forcing the replacement of the charcoal system for electricity, gas, or diesel oil. Later, the “Clean Air Acts” was implemented, extending the air pollutant emission control to industries (Quality of Air Means Quality of Life, 2006).

Since then, the evidences of the human health impact of pollution have been obtained through a variety of epidemiological studies (cross-sectional, cohort, observational, case-control studies) conducted in different countries, proving a strong association with increased cardio-respiratory morbidity and mortality, especially in groups of susceptible individuals such as the elderly and children, particularly when associated with chronic exposure. More recently, persistence of these associations with low pollutant concentrations and short-term exposure has been demonstrated. This knowledge has influenced the improvement of control actions, especially in developed countries and developing countries are also following the same guidelines (Saldiva, Pope, Schwartz, Dockery, Lichtenfels & Salge, 1995; Samet, Dominici, Curriero, Coursac & Zeger, 2000; Braga, Saldiva, Pereira, Menezes, Conceição & Lin. 2001; Katsouyanni, Touloumi, Samoli, Gryparis, Le Tertre & Monopolis, 2001; Hoek, Bruneekreef, Goldbohm, Fischer & Brandt. 2002; Pope, Burnett, Thurston, Thun, Calle & Krewski, 2004; Dominici, McDermott, Daniels, Zeger & Samet. 2005; Pope, Muhlestein, May, Renlund & Anderson, 2006).

CLASSIFICATION OF AIR POLLUTANTS

Technological advances have allowed the recognition of urban and non-urban air pollution as a complex mixture of components with different physical and chemical characteristics, namely gases, liquids, and particulate material, derived from a variety of sources; and its classification according to the chemical composition, particle size, emitting source, and mode of release into the indoor and outdoor environments (Table-1).

Primary pollutants are those emitted directly into the atmosphere, whereas secondary pollutants are those resulting from chemical reactions with other pollutants or atmospheric gases. This distinction is important because reducing the precursor does not necessarily lead to a proportional reduction in the secondary pollutant. For instance, ozone levels may paradoxically increase when nitrogen oxide emission is reduced.

In the urban atmosphere, the particulate material (PM) is divided into three categories according to the particle size, and it differs as to the origin, chemical composition and physical characteristics. These categories are as follows:

• coarse particulate: particles with aerodynamic diameter from 2.5 to 10 μm. They are mechanically produced by the break-up of larger particles from industrial activity; in roads, they come from the soil (road dust, for instance, brake and tire dust); construction debris; biological material, such as pollen and bacteria; agricultural processes;
• fine particulate, with aerodynamic diameter lower than 2.5 μm. It is usually formed from the combustion process including motor vehicles, wood burning, plantation or forest burning, and some industrial processes. The major precursor gases are sulfur dioxide (SO2), nitrogen oxides (NOx), ammonia (NH3), and volatile organic compounds;

Table-1: Classification of Air Pollutants

<table>
<thead>
<tr>
<th>Classification of Air Pollutants</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A. Regarding the mode of emission into the atmosphere</strong></td>
<td></td>
</tr>
<tr>
<td>1. Primary pollutants: pollutants emitted directly into the atmosphere, for example: sulfur dioxide (SO2), some types of nitrogen oxide (NOx), carbon monoxide (CO), and particulate material</td>
<td></td>
</tr>
<tr>
<td>2. Secondary pollutants: pollutants that are formed in the atmosphere as a result of chemical reactions with other pollutants and gases, such as ozone (O3).</td>
<td></td>
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<tr>
<td><strong>B. Regarding the origin (indoor or outdoor environment)</strong></td>
<td></td>
</tr>
<tr>
<td>1. <strong>Indoor pollutants</strong></td>
<td></td>
</tr>
<tr>
<td>(a) Sources: combustion of products such as wood, in cooking activities; particle resuspension; building supplies; air conditioner; tobacco smoke; heating; and biological agents.</td>
<td></td>
</tr>
<tr>
<td>(b) Products: CO, carbon dioxide (CO2), aldehydes, alcohol, ketones, alkalis, microbial agents, and organic dusts.</td>
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<tr>
<td>2. <strong>Outdoor pollutants</strong></td>
<td></td>
</tr>
<tr>
<td>(a) Sources: industrial, commercial, urban, regional, natural, agricultural.</td>
<td></td>
</tr>
<tr>
<td>(b) Products: SO2, NOx, PM, benzene</td>
<td></td>
</tr>
<tr>
<td><strong>C. Regarding the type of pollutant</strong></td>
<td></td>
</tr>
<tr>
<td>1. Gaseous: SO2, NOx, CO, benzene, aldehydes, 1,3-butadiene</td>
<td></td>
</tr>
<tr>
<td>2. Particulate: Total Suspended Particulate (TSP), coarse particulate material (PM10), fine particulate material PM2.5, and ultrafine particles (UFP &lt; 0.1 μm)</td>
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</tr>
</tbody>
</table>

• ultrafine particles: with aerodynamic diameter lower than 0.1 μm. They are produced by metal condensation or high-temperature vaporization of organic compounds (fossil fuel combustion). They are usually composed of nitrate, sulfate, carbon, ammonia, and metal traces;

• total suspended particulate (TSP): represents the pool of several particulate material usually exceeding 10 μm in diameter, sometimes reaching up to 50 μm in diameter.

The particles in the inhaled air selectively deposit in the respiratory tract according to their size, so that the coarse particles (PM10) are able to penetrate the upper airways, whereas only the fine (PM2.5) and ultrafine particulate are able to penetrate up to the pulmonary alveoli. To make the idea of the size of different particulate matters clearer, Brook et al., 2004 compared these pollutants to infectious agents and cells, and showed that TSP has dimensions ranging from the thickness of a hair thread to those of a cell, that is, below vision limit (between 5 and 10 μm). PM2.5 is comparable to the size of bacteria (1 μm), whereas ultrafine particles have dimensions close to those of viruses (0.1 μm), or even molecules (0.01 μm).
As from the 1960’s, the United States of America established air quality standards specifying the pollutants that would be controlled. To make this control effective, the US Environmental Protection Agency – EPA was created, with powers to determine the technical criteria for the control of substances considered toxic, based on their health effects. The following substances were selected: ozone (O3), sulfur dioxide (SO2), nitrogen dioxide (NO2), carbon monoxide (CO), and inhalable particles – PM10. In 2006, the control of PM2,5 atmospheric levels was included (Table 2).

Table 2: Air quality standards for the major pollutants according to the US Environmental Protection Agency (EPA)

<table>
<thead>
<tr>
<th>Pollutants</th>
<th>Primary standards</th>
<th>Averaging time</th>
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<tbody>
<tr>
<td>PM10</td>
<td>50 μg/m³</td>
<td>Annual arithmetic mean</td>
</tr>
<tr>
<td></td>
<td>150 μg/m³</td>
<td>Limit level for 24 hours</td>
</tr>
<tr>
<td>PM2,5</td>
<td>35 μg/m³</td>
<td>Annual arithmetic mean</td>
</tr>
<tr>
<td></td>
<td>15 μg/m³</td>
<td>Limit level for 24 hours</td>
</tr>
<tr>
<td>O₃</td>
<td>0.12 ppm (225 μg/m³)</td>
<td>Maximum hourly average</td>
</tr>
<tr>
<td>SO₂</td>
<td>0.03 ppm (80 μg/m³)</td>
<td>Annual arithmetic mean</td>
</tr>
<tr>
<td></td>
<td>0.14 ppm (80 μg/m³)</td>
<td>Maximum level in 24 hours</td>
</tr>
<tr>
<td>CO</td>
<td>9 ppm (10 μg/m³)</td>
<td>Maximum 8-hour average</td>
</tr>
<tr>
<td></td>
<td>35 ppm (40 μg/m³)</td>
<td>Maximum level in 1 hour</td>
</tr>
<tr>
<td>NO₂</td>
<td>0.053 ppm (100 μg/m³)</td>
<td>Annual arithmetic mean</td>
</tr>
</tbody>
</table>

Cases of Ill Effects of Different Air Pollutants

Increased rates of hospital admissions and daily mortality for cardiovascular diseases related to the air pollutant mass have been demonstrated by several authors worldwide. In those with chronic lung and heart diseases, these rates are even higher (Pope, Burnett, Thurston, Thun, Calle & Krewski,. 2004; Dominici, McDermott, Daniels, Zeger & Samet. 2005; Goldberg, Burnett, Bailar, Tamblyn, Ernst, Flegel, et. al., 2001). In 1994, Schwartz showed that air pollution is primarily associated with non-hospital deaths represented by sudden deaths, many of which for arrhythmia and myocardial infarction (Schwartz, 1994) and verified an increase by 1.06 (95%CI = 1.03-1.10 in the relative risk for mortality for each 100 μg/m³ increase in TSP levels, and even higher relative risks for cardiovascular disease (1.08) and for the elderly population (1.09) (Schwartz & Morris, 1995).

The adverse effects of high levels of fine inhalable particles, the PM2,5, were the object of the Harvard Six Cities Study, which involved six US cities and showed a clear association with hospital admissions of cardiovascular causes and, to a lesser extent, with chronic obstructive pulmonary disease (COPD) (Laden, Neas, Dockery & Schwartz, 2000). These findings were also confirmed for the coarse particulate, the PM10, in the National Morbidity, Mortality and Air Pollution Study (NMMAPS), the largest longitudinal study ever conducted, involving a variety of environments in 90 different American cities, and which resulted in positively associated high PM10 levels with cardio-respiratory mortality and hospital admissions for pneumonia, worsening of cardiovascular diseases and of chronic obstructive pulmonary disease (COPD) in patients older than 65 years (Dominici, McDermott, Daniels, Zeger & Samet. 2005). Five thousand individuals living close to heavy-traffic roads in the Netherlands were followed up for eight years, and a higher association with
cardio-respiratory mortality was found for this group in comparison with individuals who live in regions with less environmental pollution (Hoek, Brunekreef, Goldbohm, Fischer & Brandt, 2002).

Cardiovascular ischemic complications such as the activation of the atherosclerotic process, exacerbation of cardiac symptoms due to arrhythmias, increased heart rate, decreased heart rate variability, significant conditions for the risk of sudden death, and worsening of heart failure were related to acute increases in pollutant concentrations (Routine & Ayres, 2005; Delfino, Sioutas & Malik, 2005; Bhatnagar, 2004). These findings suggest the possibility of the occurrence of the cardiac autonomic control, being affected due to the pollutant (fine particulate) toxicity directly in the heart (Nemmar, Hoet, Vanquickenborne, Dinsdale, Thomeer & Hoylaerts, 2002). More recently, increased risk for mortality and increased hospital admissions, both for stroke, were demonstrated and related to chronic and acute exposures to air pollution (Maheswaran, Haining, Brindley, Law, Pearson, Fryers, et al., 2005). Another key issue is the strong association between the particulate material and the levels of gaseous pollutants (CO, NO2, SO2, and O3). A study including 29 European cities (Katsouyanni, Touloumi, Samoli, Gryparis, Le Tertre & Monopolis, 2001) concluded that in the cities with the highest NO2 levels, the positive association for overall mortality and PM10 was significantly higher. The same has been demonstrated for cardiovascular mortality (Bhatnagar, 2004).

**Possible Biological Mechanisms Associating Air Pollution to Heart Disease**

Studies on controlled exposure involving humans and animals show that, when inhaled, the ambient PM may cause direct effects on the respiratory tract, the major of which is the production of an inflammatory response. Other effects include the exacerbation of preexisting respiratory disease (for instance, hyperactivity), imperfect pulmonary defense mechanisms, and increased production of antigen-specific immune-globulins, which may change the airway reactivity to antigens or affect the pulmonary ability to deal with bacterial agents, thus facilitating the occurrence of bacterial infections, and this would explain the findings of increased incidence of pneumonia in the individuals exposed (Saldiva PH, Clarke RW, Coull BA, Stearns RC, Lawrence J, Murthy GG, et al., 2003). Lung inflammation induced by concentrated ambient air particles is related to particle composition. Am J Respir Crit Care Med. 2002;165(12):1610-7. Samet J, Brauer M, Schlesinger R. WHO Air Quality Guidelines Global 24 update 2005. WHO; 2005). Also, as a result of these types of study, the production of systemic inflammation secondary to pulmonary inflammation mediated by increased levels of some interleukins, especially IL-6 and IL-8, with subsequent increase in plasma levels of acute phase proteins, especially C-reactive protein and fibrinogen (a key protein in blood clotting) could be observed both in humans or animals (Seaton A, Soutar A, Crawford V, Elton R, McNerlan S, Cherrie J, et al., 2004). Particulate air pollution and the blood. Thorax. 1999;54(11):1027-32.

Pekkanen J, Brunner EJ, Anderson HR, Tiittanen P, Atkinson RW. Daily 26. concentrations of air pollution and plasma fibrinogen in London. Occup Environ Med. 2000;57(12):818-22.). In observational studies in humans, there is also a clear relationship between the exposure to urban combustion particles and the elevation in polymorph nuclear neutrophil and platelet count in blood samples of healthy individuals, as well as bone marrow stimulation. Although with a low relative risk, increased levels of fibrinogen and of neutrophil count have been associated with coronary disease, sudden death and acute myocardial infarction. Observational studies in humans showed increased blood pressure levels, decreased heart rate variability and death for arrhythmia. Bradycardia and decreased heart rate variability were also observed in exposed animals. Due to the variability of the chemical profiles of the ambient particles, the most important components have not been positively determined, although the most common constituents are known. Experimental studies with rats suggest that adverse biological responses to PM are more related to the emitting-source-dependent particulate composition and to weather conditions, than to the particle mass (Magari, Schwartz, Williams, Hauser, Smith & Christiani, 2002; Rivero, Sassaki, Filho Lorenzi, Saldiva. 2005; Schaumann, Borm, Herbrich, Knoch, Pitz M, Schins, 2004).
Many transition metals (iron, copper, vanadium, zinc, chrome, cobalt, cadmium and nickel) have been suggested as potential agents inducing the inflammatory response to the particulate matter, since they more constantly associate with smaller particles and exhibit a great oxidative potential. The free radicals generated in the oxidative process play a key role in the pathogenesis of many pulmonary diseases and, more recently, the cardiac oxidative effect has been considered by many authors as a risk factor for the occurrence of dysfunction of the cardiac autonomic regulation and, consequently, for the occurrence of arrhythmias, changes in the heart rate variability, vasoconstriction, arterial thrombosis and heart failure. Studies with rat models have demonstrated increased oxidative stress in the heart after particle inhalation (Samet, Brauer & Schlesinger, 2005). Although a considerable amount of data relates the coarse and fine particulate to adverse health effects, very little is known about the ultrafine particles (< 0.1 μm), which are more abundant, potentially more toxic, and, to date, with no standardization for the control of their levels in the ambient air. More recently, in an attempt to explain the rapid adverse effects seen in the cardiovascular system, such as acute myocardial infarction, a few hours following exposure to pollution, many authors have postulated the possibility of ultrafine particles being able to rapidly cross the pulmonary epithelial barrier toward the circulation, thus leading to direct effects on the cardiovascular system and blood with higher toxicity than PM2.5 and PM10 because they contain higher concentrations of transition metals and reluctant chemicals (Nemmar, Hoet, Vanquickenborne, Dinsdale, Thomeer & Hoylaerts, 2002).

However, despite all the evidences shown in countless studies, the exact biological mechanism or mechanisms responsible for the adverse health effects have not been fully identified. Factors depending on the recipient, such as genetic and comorbidity factors, as well as the composition and aerodynamic characteristics of the particles, play an important role in the pathogenesis of particle-induced inflammation. The possible biological mechanisms associating air pollution with heart disease are listed in Table 3, and involve the direct effect on the cardiovascular system and blood, and/or indirect effects mediated by pulmonary oxidative stress as well as by pulmonary and systemic inflammatory response.

Table-3: Possible Biological Mechanisms Associating Air Pollutants to Heart Diseases

- Pulmonary inflammation induced by particulate matters and/or gaseous pollutants, and related to chemical composition of the particles.
- Systemic inflammation secondary to pulmonary inflammation (via 2. interleukins).
- Local (pulmonary) and systemic generation of free and oxidative-stress radicals related to transition metals present especially in particles from fossil fuel combustion.
- Indirect induction of a pro-coagulation state, that is, by increasing the synthesis of acute phase proteins such as fibrinogen, and/or direct induction after access to the circulatory system (blood).
- Stimulation of the autonomic nervous system activity, which regulates airway reactivity and heart rate variability.

Impact of Air Pollution on Health

The magnitude of the London fog of 1952, which affected such a large number of people, was the first incident that made people aware of the damage done to the atmosphere due to industrialization. The SPM levels increased manifold and resulted in over 4000 deaths. Indoor air pollution can be particularly hazardous to health as it is released in close proximity to people. It is stated that a pollutant released indoors is many times more likely to reach the lung than that released outdoors. In the developing countries a fairly large portion of the population is dependent on biomass for their energy requirements. These include wood, charcoal, agricultural residue, and animal waste. Open fires used for cooking and heating are commonly found in the household both in the rural and the urban areas. The stove is often at floor level, adding to the risk of accident and the hygiene factor. In addition, they are often not fitted with a chimney to remove the pollutants. In such households the children and women are most likely to be affected, as they are the group...
that spends more time indoors. The main pollutant in this environment is the SPM. In fact, death due to indoor air pollution, (see figure1) mainly particulate matters, in the rural areas of India are one of the highest in the world. Many of the deaths are due to acute respiratory infections in children; others are due to cardiovascular diseases, lung cancer, and chronic respiratory diseases in adults. If emissions are high and ventilation is poor, household use of coal and biomass can severely affect the indoor air quality. Pollutant emissions per meal are also very high compared to those of other fuels. Household use of fossil fuel is also fairly common in the developing countries, particularly coal—both bituminous and lignite. These are particularly damaging as they burn inefficiently and emit considerable quantities of air pollutants. If emissions are high and ventilation poor, then the exposure levels to the gases emitted are far higher. The most harmful of the gases and agents that are emitted are particulate matter, carbon dioxide, polycyclic organic matter, and formaldehyde. The indoor concentrations of these are far higher than the acceptable levels and is a cause for concern in rural areas (Braga, Saldiva, Pereira, Menezes, Conceição & Lin, 2001; Pope, Muhlestein, May, Renlund & Anderson, 2006).

**Figure 1: Indoor Air Pollution**
Health impacts of Specific Air Pollutants: Some of these gases can seriously and adversely affect the health of the population and should be given due attention by the concerned authority. The gases mentioned below are mainly outdoor air pollutants but some of them can and do occur indoor depending on the source and the circumstances (Sunyer, 2008).

- **Tobacco smoke.** Tobacco smoke generates a wide range of harmful chemicals and is a major cause of ill health, as it is known to cause cancer, not only to the smoker but affecting passive smokers too. It is well-known that smoking affects the passive smoker (the person who is in the vicinity of a smoker and is not himself/herself a smoker) ranging from burning sensation in the eyes or nose, and throat irritation, to cancer, bronchitis, severe asthma, and a decrease in lung function.

- **Biological pollutants.** These are mostly allergens that can cause asthma, hay fever, and other allergic diseases.

- **Volatile organic compounds.** Volatile compounds can cause irritation of the eye, nose and throat. In severe cases there may be headaches, nausea, and loss of coordination. In the longer run, some of them are suspected to cause damage to the liver and other parts of the body.

- **Aldehyde.** Exposure causes irritation to the eyes, nose and may cause allergies in some people.

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<table>
<thead>
<tr>
<th>Category</th>
<th>Source</th>
<th>Emitting pollutants</th>
</tr>
</thead>
<tbody>
<tr>
<td>Agriculture</td>
<td>Open burning</td>
<td>Suspended particulate matter, carbon monoxide, volatile organic compounds</td>
</tr>
<tr>
<td>Mining and quarrying</td>
<td>Coal mining; crude oil and gas production; stone quarrying</td>
<td>Suspended particulate matter, sulphur dioxide, oxides of nitrogen, volatile organic compounds</td>
</tr>
<tr>
<td>Power generation</td>
<td>Electricity; gas; steam</td>
<td>Suspended particulate matter, sulphur dioxide, oxides of nitrogen, carbon monoxide, volatile organic compounds, sulphur trioxide, lead</td>
</tr>
<tr>
<td>Transport</td>
<td>Combustion engines</td>
<td>Suspended particulate matter, sulphur dioxide, oxides of nitrogen, carbon monoxide, volatile organic compounds, lead</td>
</tr>
<tr>
<td>Community service</td>
<td>Municipal incinerators</td>
<td>Suspended particulate matter, sulphur dioxide, oxides of nitrogen, carbon monoxide, volatile organic compounds, lead</td>
</tr>
</tbody>
</table>
Lead. Prolonged exposure can cause damage to the nervous system, digestive problems, and in some cases cause cancer. It is especially hazardous to small children.

Radon. A radioactive gas that can accumulate inside the house, it originates from the rocks and soil under the house and its level is dominated by the outdoor air and also to some extent the other gases being emitted indoors. Exposure to this gas increases the risk of lung cancer.

Ozone. Exposure to this gas makes our eyes itch, burn, and water and it has also been associated with increase in respiratory disorders such as asthma. It lowers our resistance leading to colds and pneumonia.

Oxides of nitrogen. This gas can make children susceptible to respiratory diseases in the winters.

Carbon monoxide. CO (carbon monoxide) combines with haemoglobin to lessen the amount of oxygen that enters our blood through our lungs. The binding with other haem proteins causes changes in the function of the affected organs such as the brain and the cardiovascular system, and also the developing foetus. It can impair our concentration, slow our reflexes, and make us confused and sleepy.

Sulphur dioxide. \( \text{SO}_2 \) (sulphur dioxide) in the air is caused due to the rise in combustion of fossil fuels. It can oxidize and form sulphuric acid mist. \( \text{SO}_2 \) in the air leads to diseases of the lung and other lung disorders such as wheezing and shortness of breath. Long-term effects are more difficult to ascertain as \( \text{SO}_2 \) exposure is often combined with that of SPM.

SPM (suspended particulate matter). Suspended matter consists of dust, fumes, mist and smoke. The main chemical component of SPM that is of major concern is lead, others being nickel, arsenic, and those present in diesel exhaust. These particles when breathed in, lodge in our lung tissues and cause lung damage and respiratory problems. The importance of SPM as a major pollutant needs special emphasis as a) it affects more people globally than any other pollutant on a continuing basis; b) there is more monitoring data available on this than any other pollutant; and c) more epidemiological evidence has been collected on the exposure to this than to any other pollutant.

CONCLUSION

To conclude, though to date, the literature on the analysis of the biological effects of the exposure to air pollution has strongly suggested the existence of a cardiac toxicity mechanism, surprisingly, little attention has been given to environmental factors that could influence inflammation levels, oxidative stress, and cardiac autonomic regulation, thus increasing the cardiovascular risk. Therefore, the identification of the toxicity of several air pollutant constituents (both chemical and biological), and of the basic mechanisms (local and systemic mediators, cellular and molecular signaling, pathways, and the toxin-specific metabolism) affecting atherogenesis, vascular function, heart rate variability induction of thrombosis is worth being further investigated through basic research. Also important is that the initiative of PM2.5 level reduction promoted by the US Environmental Protection Agency in 2006 be widely adopted: that primary standards for the control of ultrafine particles be established, both in the 24 hours and annual means, since the latter are considered the most hazardous for the cardiovascular system and are, up to the moment, free of any type of control action; that pollutant levels considered “safe” to be established for individuals belonging to cardiovascular event risk groups; and, mainly, that governmental authorities be aware of the need for improving urban vehicular traffic conditions to save the precious life of human beings and to make this earth a best place to live in.
REFERENCES


