

# Identifying the Sources of Primary Air Pollutants and their Impact on Environmental Health: A Review

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**Abstract**— Air quality in most of the mega-cities has been resulted from natural as well as anthropogenic environmental conditions. As compared to developed countries, the issue of air pollution is more serious and complicated in developing countries. Air pollution can be governed by two ways such as emission and transboundary movement of air pollutants. Each factor has different paths and sources through which they enter into atmosphere and affect the surroundings. The problem which needs to be addressed is to find out the paths and causes of emissions. Though there are several air pollutants which comes into focus while taking air pollution issue into consideration, but to explore and solve the behaviour, pattern and chemistry of primary air pollutants (NO<sub>x</sub>, SO<sub>x</sub>, CO, VOCs and PM) is a big task. Identification of sources of primary air pollutants is important because they are the only one which is responsible for the production of secondary air pollutants. In this review the focus is given on discussion about general concepts of air pollution by highlighting more specifically primary air pollutants, nature of primary air pollutants, emission budgets and their impact on environmental health by taking plant and human health into consideration. The review ends up with some of the control policies and regulations to curb the concentration of primary air pollutants.

**Index Terms**— Air quality, primary air pollutants, plant health, human health, sources.

## I. INTRODUCTION

Air pollution is a global issue which can be defined as “the alteration in the earth’s atmosphere due to the presence of contaminants which cause physical, chemical or biological short – or long-term change which causes deleterious effects to human and plant health” [1]. The pollutants can be formed from different processes like (1) combustion or industrial process sources present in a particular area, (2) from same sources present along the periphery of that area or, (3) from natural or anthropogenic processes present in the area of earth’s atmosphere. The pollutants may transport from many distant sources, or their concentrations can be affected by change in meteorological conditions. The phenomena of air pollution can be caused by anthropogenic and natural activities. Anthropogenic sources such as vehicular, industrial, agricultural, mining or domestic activities contribute a large part of causing air pollution [2]. Simultaneously, natural sources like volcanic activities, dust storms, forest fires which also plays a significant role in

causing the air quality poorer and worse. The amount of air pollution is dependent upon two factors: the nature of location

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and duration. The concentration of air pollutants varies from location to location and different durations like a number of weeks, months or years till the air pollutants reside in the environment. Air pollutants can affect human as well as plant health by causing respiratory diseases, cardiovascular, lung disorders etc. and decline in photosynthetic activity, sunken stomata, chlorosis, necrosis in leaves etc. respectively [3].

In recent years, the problem of air pollution has been increased by rapid urbanization, industrialization, large number of vehicular fleet and increase in high energy consumption. The pollutants enter into the atmosphere by primarily these main routes and categorized as primary and secondary pollutants. The pollutants are those which are emitted directly into the air are called as primary pollutants while those which are formed in the atmosphere by consequent reaction of primary pollutants are called as secondary pollutants [4]. The residence time of a pollutant is a major factor in atmospheric sciences studies. Every pollutant has different residence time and as per their stability and nature of pollutant, it can reside in the atmosphere. As long as any pollutant stays in the atmosphere, it can contribute largely in terms of pollution of that particular area. There are some climate forcing pollutants which stays in the atmosphere for many centuries like CO<sub>2</sub> that tend to accumulate in the atmosphere, so their total warming impact continues over time. The other climate forcers, such as ozone and black carbon, remain in the atmosphere for very short duration. Therefore, their emissions were reduced and may have beneficial impacts on climate. These pollutants can be transported through long distances and may produce an irreparable loss to the environment [5]. By taking these issues into consideration, the present review focuses on discussion about general concepts of air pollution by highlighting more specifically primary air pollutants, nature of primary air pollutants, emission budgets and their impact on environmental health by taking the plant and human health into consideration. The review ends up with some of the control policies and regulations to curb the concentration of primary air pollutants.

## II. AN OVERVIEW OF AIR POLLUTION

Most of megacities worldwide suffer from severe air pollution related hazards, and they have been increasing since few decades. As already discussed before, the primary factors responsible for this is the population explosion, a drastic increase in vehicular growth, rapid industrialization and changing lifestyles. The urban population growth has an exponential increase due to increase in birth rate over death rate especially in developing countries and migration in the cities. On the other hand, air pollution can cause serious health related issues such as i) increase in cardiovascular and respiratory diseases ii) decrease in lung function capacity iii)

increase in respiratory symptoms such as difficulty in breathing and coughing iv) decrease in immunity levels v) affects central nervous system which causes loss of IQ and impacts on learning, memory and behaviour vi) cancer and vii) premature death [6]. Some sensitive populations are at higher risk for air pollution related health problems like pre-existing heart and lung diseases, diabetes in older adults and even in children. According to EPA [7], approximately 127 million people lived in counties exceeded national air quality standards. Moreover, William [8] states that these conditions are worse in China, US and other big nations. In US, about 1.3 billion people are exposed to hazardous air pollutants every year. There are various pollutants which cause damage to human health. Among primary and secondary air pollutants, primary air pollutants like  $\text{NO}_x$ ,  $\text{SO}_x$ ,  $\text{CO}_2$ ,  $\text{CO}$ , VOCs and PM possess more deleterious effects. For example  $\text{SO}_2$  and  $\text{NO}_x$  cause eye irritation and respiratory ailments and will cause vital damage to tissues and organs in the body. On the other hand, carbon monoxide decreases the oxygen carrying capacity of red blood cells around the body when combines with it and forms carboxyhemoglobin [9] which causes choking and ultimately death of an individual. There are various major air pollution episodes which create history in the field of air pollution and health. For example, London Smog of 1952 which was held on 5th – 9th December, 1952. At that time, the air was stagnant and solar radiation could not be able to penetrate the cloud cover, humidity recorded was 80% and temperature was dropped to approximately  $10^\circ\text{C}$  [10]. There was severe cold that includes whole London and the smoke comes from burning of coal mixed up with dense layer of fog forms “SMOG” and Spread throughout the city for five days. About 4000 people were dead, another 8000 people dead after an interval of a week from the exposure of hazardous air pollutants which is referred to as “Great Smog of 1952” making the death toll to be approximately to 12000. This episode gives rise to implementation of Clean Air Act which was passed by Parliament in 1956 and 1968. There are some other examples which experience air pollution disasters like Belgium 1930, Mexico 1950, New York, 1953, 1963 and 1966, India 1984 and they are called as “Air Pollution Episodes” [10]. Clean Air Act focuses on reducing the emissions of carbon monoxide (CO). CO has a major role in the photochemistry of atmosphere which leads to high levels of tropospheric ozone [11].

Most of air pollutants enter the atmosphere through transport sector. The fuel present in the automobiles is responsible for increasing the amount of air pollutants in the environment and affecting the air quality. Gasoline is the principal fuel present in the automobiles. There are different types of gasoline fuels such as aviation gasoline, diesel, jet fuel and fuel oils that emit several air pollutants due to incomplete combustion results mostly in the emission of unburnt fuel, carbon monoxide, and lubricating oil. Gasoline engines consists of nearly stoichiometric ratio that is, air:fuel ratio is approximately 14:6:1 [12]. Gaseous and particulate phase chemical compounds are also emitted while incomplete combustion of gasoline. All the major pollutants viz. oxides of sulphur ( $\text{SO}_x$ ), oxides of nitrogen ( $\text{NO}_x$ ), volatile organic compounds (VOCs), carbon monoxide, and particulate matter are emitted from gasoline exhaust. Hence, in case of primary pollutants, vehicular pollution is one of the sources which is primarily responsible for their emissions into the atmosphere. Apart

from transport sector, there are other sources like burning of fossil fuels, industries, domestic and incineration of garbage. Huge amount of combustion produces mostly particulate matter which is highly responsible for haze formation and becomes the cause of smog problem in most of the megacities [13].

Air pollution can also cause damage to plants. Air pollution can affect plant health by altering their plant physiology, biochemistry and morphology. Various atmospheric gasses produce different types of visible symptoms which give rise to reduced plant growth and productivity. On global scale, plant – environment interactions has also been studied when air pollution and plant health issues taken into consideration [14]. The symptoms or effects in physiology and biochemistry indicates the state of environment. In air pollution studies, biomonitoring is also an interesting concept. There are some plants which act as indicators when exposed to air pollutants, as a consequence of which different plants produce different symptoms especially morphologically which are different for different air pollutant. Hence, plants can act as a valuable tool in the methods for controlling of air pollutants. Therefore, such type of plants come into “sensitive category”. For biomonitoring purpose, Air Pollution Tolerance Index (APTI) is used to demarcate the plants into different categories like tolerant, moderate, sensitive and very delicate. Tolerant plants act as “sinks” and delicate plants act as “indicators” [15]. Plants have taken air pollutants from the surroundings and they have adsorbed, absorbed, accumulated or imbibed in the plant body. If the air pollutant is toxic, it may cause injury and produce symptoms especially morphologically. The tolerant plant species help in scavenging which help in reducing overall pollution load. Air pollutants can also cause damage to leaf cuticles and affect stomatal conductance. They can also affect photosynthetic systems, respiration rate and patterns of carbon allocation within plants. The pattern of air pollutants impact on plant health can be taken into two ways viz. directly through visible injury (chlorosis and necrosis) or indirectly through growth and reproduction [16].

Primary air pollutants also involved in the formation of various types of phenomena in atmospheric sciences like acid rain, greenhouse effect, global warming, photochemical smog etc. The presence of some of the major gasses in the Earth’s atmosphere such as carbon dioxide, methane. When their levels become high due to any anthropogenic activity consequently raises the global mean surface temperature by 30 K. Thus, these major gasses act as pollutants (categorize under primary air pollutants) and trap the longwave radiation, thus another cause in increase in temperature [17]. This phenomena is known as “greenhouse effect”. It was estimated that concentration of these gases were increased since 1860 may be responsible for raising surface temperatures  $0.5^\circ\text{C}$  or so and in the next 40-50 years about  $1.5^\circ\text{C}$  has increased [17]. The increase in the concentration of  $\text{CO}_2$  specifically from fossil fuel combustion and smaller industrial sources were slowed down in 2012. Whereas, the global annual average growth rate of atmospheric  $\text{CO}_2$  concentrations was found high (2.4 ppm in 2012). In 2011, global emissions increased by 1.4%, reached up to a total of 34.5 billion tonnes in 2012. In 2012, the emissions were reduced to only 1.1% as compared to 2.9% increase in 2000 [18]. The growth in global  $\text{CO}_2$  emissions increased by only 0.5% in 2014 as compared to 2013 which was 1%. In the year 2014, 35.7 billion tonnes

CO<sub>2</sub> came from emissions from fossil fuel combustion and from industrial processes. Hence, CO<sub>2</sub> emissions were increased on higher rate and subsequently greenhouse effect and global warming are increasing [19].

Another phenomena is acid rain. It is mainly mixture of sulphuric and nitric acids depending upon the emissions of nitrogen and sulphur oxides. When these acids come into contact with other constituents of atmosphere, protons are released which cause increase in soil acidity. With the lowering of soil pH nutrient cations were mobilized and leached away and subsequently increases availability of toxic heavy metals and this will reduce the soil fertility which ultimately affects the crop productivity. The acidification causes large scale impact on aquatic organisms and have indirect effect on human health [20].

The problem of atmospheric acid deposition in the form of rain, fog or snow is most common in countries such as Europe, North America, and East Asia [21] comprising Canada, Sweden, England, Norway, Denmark, Scotland, West Germany, Netherland, Austria, Switzerland, Russia, Poland and Czechoslovakia, Southwest China and Japan. The first incidence of acid rain was observed in England in 1958 and then considered as regional problem in Scandinavia in late 1960's. In 1965, the pH of rainwater in Sweden was reported to be 4 or less and this result was published in 13th UN Conference on Human Environment held in Stockholm in 1972. It was reported that rain and snow in most of the industrial areas of the world are among five and thirty times as acidic as would be possible in an unpolluted atmosphere [22]. In 1974, in the northeastern part of US, the pH of rain and snow was found to be 4.0. Till the mid-1970s, the problem of acid rain was mainly restricted to North America and Scandinavia, but thereafter pH of precipitation was below 4.5 in most of the northern and central Europe (Table I).

**Table I: Rainwater pH values in different regions of world (modified from Singh and Agrawal, [20])**

COUNTRIES	PH RANGE
JAPAN	4.7
EUROPE	4.1 – 5.4
CHINA	4.1 – 6.7
US	4.1 – 5.5

The pH of rainwater was sometimes as low as 3.5 at Banchory in the northeast part of Scotland. There are some regions which have been affected by acidic deposition include Europe, eastern North America, and Southeast Asia [23] (Table II).

**Table II: Rainwater pH in different areas of India (modified from Singh and Agrawal, [20])**

REGIONS	CITIES	PH
COASTAL	TRIVANDRUM	5.3
INDUSTRIAL	CHEMBUR	4.8
POWER PLANT	INDRAPRASTHA	5.0
URBAN AREA	DELHI	6.1
NON URBAN AREA	MUKTSAR	7.3

In the case of sulphur emissions, there have been substantial reductions in Europe and North America from past two decades; 65% reduction in Europe and 40% in the United

States from 1982 to 1999. In comparison of sulphur emissions, NO<sub>x</sub> and ammonia emissions are harder to control [24]. Due to precipitation process created by neutralization of ammonia emissions, it may cause soil acidification by process of nitrification, and the emissions have increased largely over a period of decades especially in some Asian countries because of large increase use of fertilizers and animal waste [25]. In the case of India, a rainfall of pH 3.5 was reported in Mumbai and air pollution levels are gradually rising in the metropolis like Kolkata and Delhi. In the case of capital city Delhi, the mean pH value of rainwater was found to be 9.1 during 1963 and 6.2 during 1984 [26]. WMO has predicted a substantial increase in acidity in cities like Hyderabad, Chennai, Pune and Kanpur. Due to acid rain problem in Bihar, West Bengal, Orissa, and southern coastal India soil have become infertile [27].

The following phenomena is photochemical smog. "Photochemical smog," is a reddish brown haze seen in many urban areas, is a mixture of reactants and products that result from the sunlight-promoted interaction between organics and oxides of nitrogen in the lower atmosphere and has a devastating effect on the environment. The primary pollutants which are involved in the smog are nitric oxide and hydrocarbons. These, in the presence of sunlight and high temperature, convert rapidly to secondary pollutants such as ozone, hydrocarbons, and photochemical aerosols, which ultimately constitute photochemical pollution. Both anthropogenic and biogenic sources have been found to be responsible for this type of pollution. Anthropogenic sources such as vehicular emissions, industrial activities, and petroleum fuels are key factors for the production of precursors like nitrogen oxides (NO<sub>x</sub>) and volatile organic compounds (VOCs) which lead to the production of secondary pollutant, ozone (O<sub>3</sub>) [28]. Photochemical smog, first identified in Los Angeles in 1940s, is a popular phenomenon in the main cities of the world. Ozone, the main oxidant, is known to have adverse effects on human health, vegetation and materials. In 1948 (30 – 31 October) at Donora, Pennsylvania smog incident occurred, twenty people died, 600 hospitalized and thousands stricken in this nationally publicized environmental disaster; in the year 1953 another smog episode kills between 170 and 260 in the month of November in New York. In another year, 1954, heavy smog conditions shut down industry and schools in Los Angeles for most of October months [29]. In the year 1959, the health of Mexico City has become worse and air has transformed from world's cleanest to dirtiest in the span of a generation. Pollutants concentrations of Mexico City are often above air quality standards. Fossil fuels are the main energy source for the Mexico's city. After their combustion, particularly in vehicles, emits incompletely burned compounds and oxidized species called as primary pollutants. These are responsible for their adverse effect onto health as well as global warming both. Several primary pollutants undergo for further transformation reaction under the influence of sunlight. The product forms due to these photochemical reactions are called as secondary pollutants. Primary and secondary pollutants, along with aerosols and water droplets are responsible for the brown haze formation in the atmosphere. In 1963, another smog incident occurred at New York and blamed for 200 deaths and in 1966, blamed for 169 deaths. A large scale air quality disaster occurred in 1997

and popularly known as “The Southeast Asian Haze” and its after-effects causing widespread atmospheric visibility and health problems within Southeast Asia. During the peak event, satellite imagery (TOMS/NASA aerosol index maps) showed a haze layer which extended over an area of more than 3 million km<sup>2</sup>, covering huge parts of Sumatra and Kalimantan. Its northward addition partially reached Brunei, Singapore, Thailand, and Malaysia. During this time, aerosol level repeatedly exceeded national ambient air quality standards (www.science24.org). Monthly mean horizontal visibility at different locations in Kalimantan and Sumatra in a month of September was below 1 km and daily maximum visibility was frequently below 100 meters [30]. In the year 2005, schools and public offices had to close in Iran, and Tehran 1600 people were taken to the hospital, in severe smog blamed largely on unfiltered car exhaust. Hospitals have mentioned increased cases of cardiopulmonary and respiratory difficulties, while several peoples are suffering from fatigue and headaches. Public offices and schools have been closed to control the number of vehicle on the roads to clean the city's blanket of smog. Several agencies have blamed the severe smog on emissions from Tehran’s (Tehran: capital of Iran) three million vehicles, many of them were without modern exhaust filters, it is estimated that up to 5,000 people die every year [20].

A smog-like haze was observed in Malaysia in 2005, which was coming from the central part of Peninsular Malaysia to a standstill. It caused a public inconvenience. Recently, in the year 2010 (Jan-Apr), South Asian haze occurred in Mekong Sub-Region including Thailand, Singapore. It mainly occurs due to vehicular pollution and forest fires. The other weather phenomenon responsible was that of La Nina.

The stringent requirement to check air pollution arises due to health problems in the daily routines of, especially megacities. Various outdoor and indoor air pollutants have produced various human health disorders like asthma, lung cancer, cardiovascular that can be considered fatal [31]. On an average, an individual inhales more than 10,000 – 20,000 liters of air per day. According to WHO [31], more than 2 million premature deaths per year may be the reason regarding effects of urban outdoor and indoor air pollution. In specific, every year deaths of 1.6 million people and 800 deaths from lung cancer, cardiovascular and respiratory diseases worldwide are caused by indoor and outdoor air pollution respectively [32].

Hence, it is mandatory for an environmentalist to pay attention towards the burning issue, air pollution. Therefore, the chapter focus is given on discussion about general concepts of air pollution by highlighting more specifically primary air pollutants, nature of primary air pollutants, emission budgets and their impact on environmental health by taking the plant and human health into consideration. The review ends up with some of the control policies and regulations to curb the concentration of primary air pollutants.

### III. TYPES OF AIR POLLUTANTS

Air pollutants can be vastly divided into their physical and chemical composition, their toxicity, and reactivity. The emission estimate of different air pollutants on global and South Asia scenario in various timelines is shown in Table 3.

The standard classification of pollutants can be of two types:

**Primary Pollutant:** Such type of air pollutants which are emitted directly from a source. For example, oxides of nitrogen (NO<sub>x</sub>), oxides of sulphur (SO<sub>x</sub>), Volatile organic compounds (VOCs), Particulate Matter (PM) and oxides of carbon (CO<sub>2</sub> + CO) [33].

**Secondary Pollutant:** Those pollutants that are derived from various reactions between different primary pollutants or with natural constituents in the atmosphere. Examples are ozone (O<sub>3</sub>) and PAN [33].

**Table III: Emission of air pollutants in World (1000 tonnes or Gg yr<sup>-1</sup>)**

Pollutants	Global		
	2003*	2008	2011
SO <sub>2</sub> /SO <sub>x</sub>	10.350	24.000	13.901
NO <sub>x</sub>	14.440	155.000	125.532
NM VOCs	93.50	100.000	81.432
PM <sub>10</sub>	PM		29.280
PM <sub>2.5</sub>	(3800)	21.000	23.196
NH <sub>3</sub>	4000	78.000	74.164

**Data source:** For 2003; [34], For 2008; Kurokawa et al., [35], For 2011; [36]. SO<sub>x</sub> is considered in case of year 2003 only.

\*approximate values

#### A. Primary Pollutant in the Atmosphere i) Oxides of Nitrogen

There are two oxides of nitrogen mainly nitric oxide (NO) and nitrogen dioxide (NO<sub>2</sub>). They are two important forms which are found in the troposphere. Another important gas is the nitrous oxide (N<sub>2</sub>O) which is greenhouse gas. It is colorless, tasteless and main form of nitrogen oxide. In nature, it is very difficult to measure nitric oxide due to its less residence time and hence it is readily converted into nitrogen dioxide which is harmful in nature by chemical reaction with ozone. NO<sub>2</sub> is yellowish-orange to reddish-brown gas with a strong, irritating odour and having strong oxidizing power. Most of the portion of NO<sub>2</sub> is converted to NO and ammonium salts. Wet or dry deposition is the same process that is required for the process of removal for nitrate aerosol and sulphate aerosol. The other and primarily very important oxides are NO<sub>2</sub> and N<sub>2</sub>O<sub>5</sub> but they are found in lower concentrations. In case of NO<sub>x</sub> emissions, only about 10% is contributed from manmade sources while remaining is produced by anaerobic processes in soil and water through natural processes like lightning and volcanic activity, and also by the photochemical breakdown of nitrogen compounds in the upper atmosphere. Overall, approximately 50 million metric tons of global nitrogen oxides emissions has been estimated from manmade sources [37]. In the US, about 20 million metric tons of nitrogen oxides are generated every year and about 40% is emitted from mobile sources. About 30% and 70% were resulted from fuel combustion in large industrial furnaces and electric furnaces respectively out of 11 to 12 million metric tons of nitrogen oxides. In the history of 1966-1980, largest emission rate was recorded in northern mid-latitudes while largest increase in the period was depicted in tropics [38].

With the help of advanced technologies like satellite observations from Global Ozone Monitoring Experiment (GOME) instrument which has European Space Agency's ERS-2 satellite found that the major source of NO<sub>x</sub> emissions is fuel combustion in northern mid-latitudes and fires were reported in tropics. At mid-latitudes, the emissions were found to be significant during summer and rainy season in the tropics. It was also found that soil and biomass burning emissions reported being about 22% and 14% of global surface NO<sub>x</sub> emissions respectively. According to an estimate, the contribution of fuel combustion of NO<sub>x</sub> emissions from Asia (including 8.8 TgN yr<sup>-1</sup> East).

**Table IV: Global NO<sub>x</sub> emission and projections in different years from various sectors, Mg**

Sector	2010	2015
Power sector	16146	9341
Heating plants	2766	2713
Petroleum refining	1544	1404
Oil/ Gas extraction	6490	5454
Commercial and Institutional plants	878	699
Residential plants	6617	5519
Plant related to agro-forestry and aquaculture	705	747
Combustion based Industrial plants	5941	4843
<b>Total</b>	<b>41087</b>	<b>30719</b>

Data Source: [36]

Asia, South East Asia or India and Japan) was found to be 28% larger than bottom-up inventory i.e. 6.9 TgN yr<sup>-1</sup> [39]. The annual mean concentrations which were found in urban areas all over the world are found in the range of 20-90 µg/m<sup>3</sup>. Maximum values of nitrogen dioxide can reach 850µg/m<sup>3</sup> and 400 µg/m<sup>3</sup> for the maximum half-hour and maximum 24-hour values respectively. According to World Bank [40], hourly averages near very busy roads often exceed 1,000 µg/m<sup>3</sup>. The pattern of NO<sub>x</sub> emissions from different sectors is shown in Table IV.

Atmospheric NO<sub>2</sub> in urban areas are dependent on the day, season and meteorological conditions. High peaks are observed during morning and afternoon peak hours. As compared to other seasons like summer and monsoon, elevated levels were found during winter due to increased use of heating fuels. Solar intensity act as a conversion factor for NO<sub>2</sub> into NO high during warm, sunny days. NO<sub>x</sub> decay rapidly because polluted air moves away from the source. In rural areas, concentrations of NO<sub>x</sub> without primary sources are typically close to background levels [41]. Nitrogen dioxide also acts as a precursor for ozone; therefore high ozone concentrations are expected in those areas where high NO<sub>2</sub> are found. In indoor, nitrogen dioxide levels are often found to be higher as compared to outdoor which can affect human health adversely. Indoor sources include cigarette smoke, gas-fired appliances, and space heaters. Kitchens with unvented gas appliances have observed high nitrogen dioxide concentrations of about 200µg/m<sup>3</sup> over a period of several days. Their one-hour concentrations may reach up to 500–1,900 µg/m<sup>3</sup> during cooking and 1,000–2,000 µg/m<sup>3</sup> in the additional use of gas-fired water heater. Though the

smoke from a cigarette may contain 150,000–225,000 µg/m<sup>3</sup> of nitric oxide, it usually has somewhat less nitrogen dioxide [41].

### Effect on Plant Health

NO<sub>x</sub> can affect vegetation both directly and indirectly. In direct process, it can enter through stomata present in the leaves and disrupt the photosynthesis and other physiological processes while indirectly by chemical reactions [42]. The effect of NO<sub>2</sub> on plants is determined by its adsorption and rate of uptake (which is called as flux) and plants. Foliar uptake in plants is highest in NO followed by NO<sub>2</sub> and than NH<sub>3</sub>, while in a case of wet deposition, the pathway by soil and roots are the primary routes for nitrogen-containing pollutants [43]. The degree at which the flux of pollutants from the atmosphere entering into the plant is a complicated procedure because this feature is highly dependent on the properties of the plant, pollutant, and surrounding conditions and it is the reason of variability of deposition velocities in different plant species (Table V).

In the case of NO, the amount and variability of deposition velocity depend on the sum of two different fluxes i.e. from the atmosphere into vegetation and from the soil into the air. Interestingly, in some cases where deposition velocity is near to zero, even then the uptake by the plant will occur [45]. According to Wellburn [43], NO and HNO<sub>3</sub> have higher deposition velocity than NH<sub>3</sub>, but this was not quantified. The flux from the atmosphere to leaf surface are dependent on two factors i.e. aerodynamic and boundary layer resistances, which are governed by meteorological conditions and also on

**Table V: Variability of deposition velocity of nitrogen-containing gases and aerosols**

Compound	Deposition Velocity (mm/s)	Sources
NO <sub>2</sub>	1-8	Department of Environment, London, 1993
NO	0-1	Prinz, 1982
NH <sub>3</sub>	12 (-5 to +30)	Fangmeijer et al. 1994
NH <sub>4</sub> <sup>+</sup>	1.4 (0.03 to 15)	Holtan-Hartwig and Bockman, 1994

Data Source: [44]

plant and leaf shape and size. The flux from the leaf surface to the cell is determined by stomatal, cuticular and mesophyll resistance. The factor responsible for the reaction of nitrogen to the plant inside depends on intrinsic plant properties and their nutritional status and also on environmental conditions. The flux of atmospheric nitrogen through the soil is influenced by properties of soil and vegetation and also by meteorological conditions. The chemical composition of soil water, nitrification rate, the preference of the plant for either NH<sub>4</sub><sup>+</sup> or NO<sub>3</sub><sup>-</sup>, the physical properties of root and metabolic activity of plants play main roles in this uptake process. Uptake of ammonia and NO<sub>x</sub> is controlled by the concentration gradient between the atmosphere and

mesophyll and mostly by stomatal conductance and also on those factors which affect stomatal aperture. In higher plants like trees, plants uptake takes place through stomata and through cuticle the chances are less [43]. Stomata is responsible for foliar uptake of atmospheric nitrogen compounds, and most of them affect stomatal pore size and the extent of further absorption. The nitrogen status of plants is also known to alter stomatal behaviour towards other environmental conditions like drought. Plants absorb gaseous NO<sub>2</sub> more rapidly than nitrogen oxide (NO). It rapidly reacts with water and gets converted to HNO<sub>2</sub> and HNO<sub>3</sub> before further utilization in plant metabolism. Nitrous and nitric acid dissociates to form nitrate, nitrite, and protons. Nitrite is more toxic than NO<sub>3</sub><sup>-</sup>. NO<sub>2</sub> exposure reduces chlorophyll content in plants. High concentration (100 ppb NO<sub>2</sub>) reduce stomatal conductance. The acidic conditions produced by NO<sub>2</sub> influence electron-flow and photophosphorylation. The damage to the photosynthetic apparatus occurs by swelling of chloroplast membranes. Biochemical and membrane injury created from NO<sub>2</sub> inhibit photosynthesis by uncoupling electron transport and inducing structural alterations. NO<sub>x</sub> could also cause photosynthetic inhibition because of competition for NADPH for the processes of nitrite reduction and carbon assimilation in chloroplasts. Exposure to low levels of NO<sub>2</sub>, increase RuBP carboxylase activity. Short exposures to a relatively high concentration stimulate RuBP carboxylase as well as glycolate oxidase activity. Polyamines such as spermine and spermidine prevent NO<sub>2</sub> induced decline in leaf damage [46].

**Effect on Human Health**

NO<sub>2</sub> is a reactive gas and causes bronchitis and pneumonia and also increasing susceptibility to respiratory infections. It affects both cellular and humoral immune system and impairs immune responses. According to Hasselbald et al. [47], children are more prone to respiratory illness and another study by Saldiva et al. [48], it has also been associated with daily mortality in children < 5 years old. Persons exposed to NO<sub>2</sub> causes chronic bronchitis and those who have already chronic respiratory disease, and emphysema is sensitive to NO<sub>2</sub>. Table VI showing possible health impact due to NO<sub>2</sub> exposure.

**Table VI: Health effects due to NO<sub>2</sub> exposure in Epidemiological Studies**

<b>Health effects</b>	<b>Mechanism</b>
Increased risk of respiratory infections	Reduced efficacy of lung defences
Increased susceptibility of respiratory infections	Reduced efficacy of lung defences
Respiratory symptoms	Airways injury
Reduced lung function	Airways and alveolar injury
Worsening clinical status of persons with asthma, chronic obstructive pulmonary disease or other chronic respiratory conditions	Airways injury

**Data Source: [44]**

In general, concentrations of NO<sub>2</sub> more than 1880 µg/m<sup>3</sup> (1ppm) are required during acute controlled exposures to produce changes in the pulmonary system in healthy adults. But such high concentrations almost not occur in ambient air; therefore concern has been shifted to those people with pre-existing lung disease. It has been observed in numerous studies that people with asthma, chronic obstructive pulmonary disease, or chronic bronchitis showed that there is a small decline in forced vital capacity and forced expiratory volume in 1 second (FEV) or increases the airway resistance. At 560µg/m<sup>3</sup> (0.3 ppm) for just 30 minutes of NO<sub>2</sub> exposure (which is considered under low level of nitrogen dioxide exposure) hardly showed significant results, generally, at this level, a direct effect on pulmonary function in asthmatics is not significant [49].

The results of effects of nitric oxide on human pulmonary function at concentrations higher than those given for nitrogen dioxide. One study indicated a likely increase in one pulmonary function variable in some healthy persons tested after 2 hours of exposure to NO concentrations of about 1200 µg/m<sup>3</sup> (1ppm) with light exercise, but the effects may occur by chance [50]. But interestingly, when Kagawa [51] evaluated the effects of a 2-hour exposure to a mixture of NO (740 µg/m<sup>3</sup> i.e. 0.6 ppm) and nitrogen dioxide (560 µg/m<sup>3</sup> i.e. 0.3 ppm) with mild exercise, no changes were found in function or nonspecific bronchial responsiveness. In addition to that, Frostell et al. [52] found no bronchoconstriction with inhalation of NO at 18 mg/m<sup>3</sup> (10 ppm) and at higher concentrations of 6 – 100 mg/m<sup>3</sup> may cause bronchodilation. For acute exposures, very elevated levels of 1990 µg/m<sup>3</sup> > 1000 ppb may affect healthy people. The patients of asthma and chronic obstructive pulmonary disease are more susceptible to acute changes in lung function, airway responsiveness, and respiratory symptoms. With small changes in lung function i.e. <5% drop in FEV between air and nitrogen dioxide exposure and also changes in airway responsiveness reported in many studies i.e. 375 – 565 µg/m<sup>3</sup> (0.20-0.30 ppm) is observed to be lowest effect level. Moreover, a 50% marginal safety is suggested due to reported statistically significant increase in response to bronchoconstrictor with exposure to 190 µg/m<sup>3</sup>. By human clinical experiments, a 1-hour guideline of 200 µg/m<sup>3</sup> is proposed. At this recommended guideline (400 µg/m<sup>3</sup>), there is possible proof to suggest possible small effects on the pulmonary function of asthmatics [53].

**ii) Oxides of Sulphur**

Oxides of sulphur (SO<sub>x</sub>) consists of sulphur and oxygen molecules. Sulphur dioxide is the primary form present in the lower atmosphere. It is colorless and found in the range of about 1,000 to 3,000 µg/m<sup>3</sup>. At high concentrations i.e. 10,000 µg/m<sup>3</sup> it produces pungent and unpleasant odour. The most common product is sulphurous acid (H<sub>2</sub>SO<sub>3</sub>) when it reacts with water. About 30% of SO<sub>2</sub> in the atmosphere is converted to sulphate aerosol, which is very quickly removed from the atmosphere through wet or dry deposition processes [53]. SO<sub>3</sub> i.e. sulphur trioxide is emitted directly into the atmosphere or produced from SO<sub>2</sub> which is easily converted to sulphuric acid (H<sub>2</sub>SO<sub>4</sub>). Most of the SO<sub>2</sub> is produced by

burning fuels which contain sulphur or by roasting or by roasting metal sulphide ores.

**Table VII: Global SO<sub>2</sub> emission in different years from various sectors, Mg**

Sector	2010	2015
Power sector	2622	1971
Heating plants	997	1383
Petroleum refining	231	321
Oil/ Gas extraction	11	7
Commercial and Institutional plants	119	105
Residential plants	1158	1380
Plant related to agro-forestry and aquaculture	1087	1202
Combustion based Industrial plants	3071	3441
<b>Total</b>	<b>9296</b>	<b>9809</b>

**Data Source: [36]**

The contribution of natural sources in the form of volcanic eruptions is about 35-65% of total sulphur dioxide emissions. Thermal power plants include high-sulphur coal or heating oil are also some of the leading man-made SO<sub>2</sub> emissions worldwide, followed by industrial boilers and nonferrous metal smelters. Emissions from motor vehicles and domestic coal burning can also contribute to high local ambient concentrations of SO<sub>2</sub> [54]. The image of SO<sub>x</sub> emissions would be clearer in Table VII.

Over a long period, high concentrations of SO<sub>2</sub> are believed to cause health and plant damage. SO<sub>2</sub> concentrations are dependent on meteorological conditions like wind, temperature, humidity and topography and can concentrate close to ground level. For example, during London Smog episode of 1952, levels have reached up to 3500 µg/m<sup>3</sup> (averaged over 48 hours) and would remain high over a period of 5 days. High concentrations have also been reported during temperature inversions in Central and Eastern Europe, in China and other localities [54].

SO<sub>2</sub> emissions in the world during 1850-2005 were determined by bottom-up mass balance method, by taking into account the country-level inventory data with special reference to coal combustion, petroleum combustion, natural gas processing and combustion, petroleum, processing, biomass burning, shipping fuels, metallurgical operations, pulp and paper processing, other industrial processes, and agricultural waste burning (AWB). In the year 1970, SO<sub>2</sub> emissions increased worldwide and decreased until 2000, but the sudden increase has been reported in last years due to high emission rate in China, international shipping, and developing countries. Mostly American and European countries were responsible for producing more than 50% of total SO<sub>2</sub> emissions in 1990. The contribution was drop down to less than 25% and emissions from Asian countries represent more than 50% of the total during 2010 [55]. It was reported by Hand et al. [56] that total SO<sub>2</sub> emissions have decreased considerably to be about 31 million tons in 1970 to 8 million

tons in 2010 in the United States. With the aid of regulation of power plant emissions have led to a decline in total emissions. Emissions in India were found to be on a sharp increase in the 1980s. During the year 2005, China, USA, Europe, South and East Asia, India and Russia tops regarding total SO<sub>2</sub> emissions. Out of the entire leading countries in the world, China emissions are highest among them. These emissions are further responsible for increasing approximately total coal combustion from 1995 to 2006 which are marked by the high growth of energy intensive industries [57]. In China, SO<sub>2</sub> emissions have increased by 53% during 2000-2006 and the main source behind them is power plants. However, the emission growth rate slowed around 2005 and consequently decreased emissions were observed after 2006 basically because of policy interventions to curb power plant emissions. It was also highlighted that the transport of SO<sub>2</sub> from the Asian continent to south-western areas of Japan despite the relatively short atmospheric lifetime of SO<sub>2</sub> [58].

### *Effect on Plant Health*

The pathway through which SO<sub>2</sub> enter into the stomata is entirely dependent on leaf age, concentration of pollutants. According to a study of Black and Unsworth [59], low concentration of SO<sub>2</sub> stimulates the stomatal conductance in *Vicia faba* L. Within 15 mins of exposure which persists for several days. This has been recognized by the destruction of epidermal cells adjacent to stomata and accumulation of sulphur within the guard cells. Larger stomatal apertures not only allow entry of pollutant but also enhance water loss. Once SO<sub>2</sub> enters by stomata, the path nearby epidermal cell is very short, and that's why the epidermal cells are more susceptible. The negative effects of SO<sub>4</sub><sup>2-</sup> occurred due to reactions under liquid phases after the process of uptake in plants. Reactive Oxygen Species (ROS) are produced by SO<sub>2</sub> absorption within cellular space. SO<sub>2</sub> easily dissolves in apoplasmic water to produce mainly sulphite (SO<sub>3</sub><sup>2-</sup>), bisulphite (HSO<sub>3</sub><sup>-</sup>) and H<sup>+</sup> ions which would reduce the pH of the medium. SO<sub>3</sub><sup>2-</sup> and HSO<sub>3</sub><sup>-</sup> ions are responsible for SO<sub>2</sub> phytotoxicity [60]. The plants which are tolerant to SO<sub>2</sub> have the ability in their leaves to detoxify sulphite and bisulphite when the concentrations are not high enough, by oxidizing them to less toxic sulphate ion. Many studies have depicted that effects caused due to SO<sub>2</sub> disrupt biochemical functions and cell structure [61] before to produce visible symptoms or reduced growth. Physiology of plant is affected by SO<sub>2</sub> and thereby creating change in photosynthesis, respiration, stomatal activity, transpiration and translocation. Plasma membrane also becomes permeable due to change in enzymatic activities and nutrient uptake and water relations [61]. Photosynthetic pigments and many enzymes are linked with the membranes of chloroplasts. Aqueous SO<sub>2</sub> is a prime factor which can produce damage to plant metabolism by acting as electron transport system. Agrawal and Deepak [62] observed the inhibition of photosynthetic CO<sub>2</sub> fixation by SO<sub>3</sub><sup>-</sup> and this effect was created due to competition between CO<sub>2</sub> and SO<sub>3</sub><sup>-</sup> for active sites of RUBISCO. When SO<sub>2</sub> alters the photosynthetic process, and it would lead down to degradation of chlorophyll. A significant decrease in chlorophyll content, root and shoot lengths, leaf area, ears, seeds and the photosynthetic rate at 60 ppb SO<sub>2</sub> have been studied [62].

The chlorophyll degradation in SO<sub>2</sub> exposed plants results from its strong redox properties. Chlorophyll destruction is also caused by free radicals produced during the oxidation of HSO<sub>3</sub><sup>-</sup>. The chlorophyll degradation produces phaeophytin molecule and Mg<sup>2+</sup>. Magnesium ions in the chlorophyll molecule are replaced by two atoms of hydrogen, thereby changing its light use efficiency. The chlorophyll b breakdown results from splitting of the phytol chain by chlorophyllase. A reduction in the Chl a/b ratio indicates that the ratio of the reaction center pigments to light-harvesting pigments in the photosystem on the thylakoid membrane in chloroplasts is noted in SO<sub>2</sub> treated plants. The reaction center is vulnerable simulated SO<sub>2</sub> treatment than the light-harvesting antenna system. Decreases in Fv/Fm and ΦPSII indicate that part of the PSII photochemistry and photochemical energy conversion in PSII were inactivated by NaHSO<sub>3</sub>. The reduction in electron transport from high concentrations of SO<sub>2</sub> is attributed to the destruction of epidermal cells adjacent to stomata and accumulation of sulphur within guard cells [63]. The inhibition of electron transfer occurs at the site close to the reaction center of photosystem II. Studies conducted using isolated chloroplasts indicate that oxidizing side of Photosystem II is more affected by SO<sub>2</sub>. The denaturation of the protein component of the pigment-protein complex caused by the destruction of disulphide bonds also accounts for the reduction in photosynthetic efficiency. Sulphurous acid (H<sub>2</sub>SO<sub>3</sub>) formed by as the byproduct of SO<sub>2</sub> degradation in plant irreversibly inhibits both cyclic and non-cyclic photophosphorylation. Short-term SO<sub>2</sub> fumigation causes a transient decrease in photosynthetic CO<sub>2</sub> uptake but the increase in non-assimilatory electron transport. Inhibition of the activity of enzymes of the Calvin cycle including 3-phosphoglycerate and hexosephosphate relate to declining in the rate of photosynthesis. The competition between CO<sub>2</sub> and SO<sub>3</sub><sup>-</sup> for active sites of Rubisco [62] is mainly responsible for the decline in carbon fixation by Rubisco. Sulphur dioxide showed inhibition of CO<sub>2</sub> assimilation accompanied by increased reduction of the quinone acceptor, QA of photosystem II and increased oxidation of the electron donor pigment P700 of photosystem I is reported. Plants exposed to SO<sub>2</sub> exhibit increase in the soluble sugars. This results from the breakdown of polysaccharides. Polyhydric sugars act as scavengers of free radicals and help to cope with increasing SO<sub>2</sub> pollution. The decrease in the content of non-structural carbohydrates and starch of damaged leaves probably correspond with the photosynthetic inhibition or increase in respiration rate [63].

### ***Effect on Human Health***

When inhalation of more than 10,000 µg/m<sup>3</sup> SO<sub>2</sub> was inhaled then, it would give rise to bronchoconstriction, chemical bronchitis. Concentrations of 2,600 – 2700 µg/m<sup>3</sup> give rise to immediate clinical symptoms with bronchospasm in asthmatics. There are some epidemiological studies which indicate the effects of short-term SO<sub>2</sub> exposures viz. small reversible decline in children's lung function (250-450 µg/m<sup>3</sup>); irritation in the bronchial system (about 500 µg/m<sup>3</sup>); increased mortality (500-1000 µg/m<sup>3</sup>); (World Bank, 1997). High concentrations of SO<sub>2</sub> (>1000 µg/m<sup>3</sup>) together with suspended particles are supposed to have been responsible for high death rate during London smog, 1952 with around 4,000

excess deaths. A large number of single and multi-pollutant time series studies have been observed relations with daily mortality and morbidity. SO<sub>2</sub> is also responsible for allergic reactions like the disease called as rhinitis. The symptoms of rhinitis is stuffing nose and sneezing, and its long-term exposure leads to other serious symptoms like atopy. A high amount of inhalation of SO<sub>2</sub> present in ambient air is shown to be linked with increased release of WBC and their precursors from the bone marrow and an elevated number of band cells in peripheral blood [65]. It may also cause damage to developing fetuses and to the reproductive system. The testes, in particular, are also affected. It may also cause increased morbidity and mortality and low birthweights. At a molecular level, SO<sub>2</sub> decreases immunity levels, increases membrane permeability, causes chromosome breakage and is mutagenic. High doses of inhaled SO<sub>2</sub> have associated with certain acute neurotoxic effects. These effects include peripheral neuritis, convulsions, agitation, tremor, vertigo and fever. Liquid SO<sub>2</sub> when found in high pressure or low-temperature conditions, may cause severe corneal damage. The corneal epithelium turns gray and after a period of hours, the eyelids swell. As a result of that, the conjunctiva may become white and opaque. Thrombosis of ocular blood vessels can also occur [66]. SO<sub>2</sub> exposure may also cause skin damage by coming directly into contact with it and produce dermatological reactions such as urticaria. At high doses, SO<sub>2</sub> severely irritates the skin which causes pain, redness, and blisters. SO<sub>2</sub> has also been responsible for inhibiting DNA synthesis and causes chromosomal damage. Such types of effects are found in sulphuric acid factory workers, whose lymphocytes have high frequencies of micronuclei. SO<sub>2</sub> exposure is associated with a decline in mitotic index and an increase in micronuclei frequency [67].

### ***iii) Volatile Organic Compounds***

VOCs are those group of organic compounds which usually present in the vapor phase at room temperature i.e. vapor pressure greater than 0.1 mmHg (0.0133kPa) at 25°C and are classified in various classes like aliphatic, aromatic and chlorinated hydrocarbons, aldehydes, ketones, esters, acids, and alcohols. VOCs have different pathways like air or through contaminated groundwater and soils. More than 1 lakh VOCs have been classified as per United States Environmental Protection Agency study [68].

VOCs have mainly two sources of emission viz. anthropogenic and biogenic. Vehicular emissions, industrial emissions (paints, varnishes, etc.) or domestic emissions are the examples of some anthropogenic source of VOCs [68]. Such type of emissions deposits gradually in the atmosphere, and their fate is determined by various chemical processes. On the other hand, biogenic emissions of these gasses (isoprenoid group - isoprene and monoterpenes) are not deposited at a significant rate. Biogenic Emission Inventory listed isoprene and monoterpenes as the most predominant biogenic VOC's groups. Isoprenes and monoterpenes are not only responsible for the tropospheric chemistry of greenhouse gas production but also take part in the aerosol formation and resulting increased acidity of the ecosystem arising from its deposition [69].

Isoprene (2-methyl-1, 3-butadiene, C<sub>5</sub>H<sub>8</sub>) is quantitatively the most important compound of NMBVOCs (Non- Methane Biogenic Volatile Organic Compounds), with an estimated

global annual emission of about 400–600 TgC/yr. It is believed that tropical forests are mainly responsible for maintaining global isoprene budget, transitional emissions from temperate forests and low emissions from agricultural areas and boreal forests, even though latter is a major source of monoterpenes [70]. Isoprene is reactive in nature due to the presence of carbon-carbon double bonds which makes it most sensitive to the attack by nitrate ( $\text{NO}_3^-$ ), hydroxyl radicals ( $\text{OH}^\cdot$ ), and ozone ( $\text{O}_3$ ), having lifetimes of 0.8 h, 1.7 h, and 1.3 days respectively [71]. Isoprene oxidation involves in the formation of tropospheric ozone and methane concentrations. It's a great matter of discussion that isoprene reactions produce ozone at two conditions i.e. whether  $\text{NO}$  is low or  $\text{NO}$  is high. But it is the isoprene oxidation at high  $\text{NO}$  concentrations that results in the  $\text{NO}_2$  formation eventually increasing the levels of  $\text{O}_3$  through its photolysis reaction. When there is a condition of high polluted urban environment, the oxidation of NMBVOCs with the help of  $\text{OH}$  leads to the production of hydroperoxy radical ( $\text{HO}_2^\cdot$ ), which when reacts with  $\text{NO}$  to recycle  $\text{OH}$  and forms  $\text{NO}_2$  and finally leads to the formation of tropospheric  $\text{O}_3$ . The total result is the maintenance of high  $\text{OH}$  concentrations, removal of NMBVOCs and total production of  $\text{O}_3$ . When there is a unpolluted condition with low  $\text{NO}_x$ , then isoprene can react directly with  $\text{O}_3$  and thus lower atmospheric concentrations of  $\text{O}_3$ . With the peaks of  $\text{OH}$  and isoprene concentration showing overlaps during the day along with their high reaction rates, the  $\text{OH}$ -pathway is coming to be popularly known as "isoprene oxidation pathway" [72]. Hence, removal of  $\text{OH}$  will reduce the rate of conversion of  $\text{NO}_2$  to nitric acid ( $\text{HNO}_3$ ), thereby, contributing directly to the efficacy of  $\text{O}_3$  production.

The most carcinogenic group of VOCs is BTEX (Benzene, Toluene, Ethylbenzene, and Xylene). These are mainly found in petroleum products, such as gasoline. Besides gasoline, BTEX can be found in many of the everyday household products like paints, varnishes, mosquito repellants, etc. [73].

**Benzene** is an aromatic hydrocarbon. It is a colorless and highly flammable liquid with a sweet smell. Benzene is a naturally produced from crude oil, and the most basic petrochemical. Vehicular exhaust and industrial emissions are known for contributing 20% of the total exposure to benzene. Benzene is found in cigarette smoke. Approximately 50% of the entire exposure to benzene results from smoking tobacco. Among BTEX, benzene is reported as the most carcinogenic compound and identified as human carcinogen by USEPA, 1999. It is used as an additive in gasoline, industrial solvent and precursor to basic industrial chemicals including drugs, plastics, synthetic rubber and nylon, insecticides, paints, dyes, resins-glues, furniture wax, detergents and cosmetics [74].

**Toluene**, formerly called as toluol, is a clear, water-insoluble liquid with the typical smell of paint thinners. Its chemical structure is a mono-substituted benzene derivative. It is produced by the process of making gasoline via a catalytic reformer and usually occurs naturally at low levels in crude oil. Toluene has good solubility with a number of inorganic chemicals like sulphur. It is used as paint thinner, silicone sealants, rubbers, printing ink, adhesives (glues), lacquers, leather tanners, as a disinfectant and as a fullerene indicator in the production of polyurethane foam and Trinitrotoluene. Industrial uses of toluene include dealkylation of benzene, octane booster in exhaust fuels used in internal combustion engines, coolant for its good heat transfer capabilities in

sodium cold traps and also in the process of removing the cocaine from coca leaves in the production of Coca-Cola syrup [75].

**Ethylbenzene** is an aromatic organic compound. It is present in small amounts in crude oil and is required as an intermediate in the petrochemical industry for the production of polystyrene, a common plastic material. It has been used as a solvent for aluminum bromide in the anhydrous electrodeposition of aluminum, additive in gasoline and aviation fuel, as an important ingredient in some paints, inks, plastics or pesticide products and solvent grade xylene (xylol) which is always contaminated with a few percent of ethylbenzene [76].

**Xylene** or xylol is a mixture of three structural isomers (ortho-, meta-, and para- Xylene) of the aromatic hydrocarbon dimethylbenzene.

**Ortho-xylene** is naturally-occurring form of xylene while the other two forms are man-made. Xylene is a clear, colorless, sweet-smelling liquid which is very flammable. It is usually refined from crude oil by a process called **alkylation**. It is also produced during the dehydrocyclodimerization and methylating of toluene and benzene, crude benzole extraction from gas or as a by product of the coal carbonisation being derived from the coke ovens. It is also used as a solvent in the printing (Xylene is commonly found in ink), rubber, and leather industries [77].

The anthropogenic sources emit about 142 teragrams of carbon per year in the form of VOCs and Muller reported that the global anthropogenic non-methane VOC emissions are about 150 Tg yr. However, the emissions can vary region to region depending on the surrounding environmental conditions. The regional breakdown of total global anthropogenic NMHCs is shown in Figure 1. In Asia, the emission from anthropogenic sources are the largest from the residential combustion of coal and biofuels (~34%) and from transportation (~27%) [78].

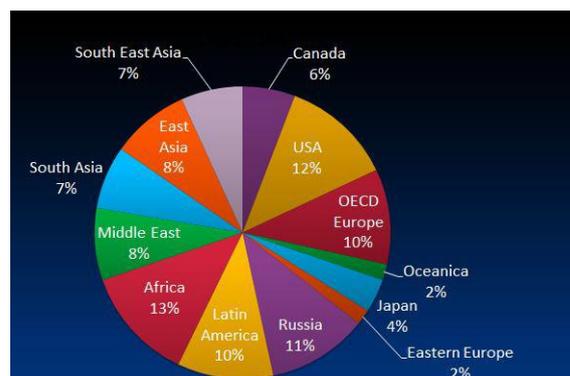


Fig. 1 Regional breakdown of total global anthropogenic non-methane VOC emission (Source: Ozone Lesson 5)

### Effect on Plant Health

Many experimental studies have already investigated the effects of VOCs on plants and all have involved short-term exposure to very high concentrations of VOCs i.e. acute exposure study. Almost all experiments were conducted on a wide variety of plants using various doses of VOCs, but with unrealistic concentrations (factors of 100 or greater than those observed value) for short exposure times and have been conducted for exposure times typically of hours. If any effects are depicted, it is considered the more sensitive species when

plant parts when exposed to high concentrations of VOCs (e.g. in chemical spillage or accident). However, there is no guarantee that those species or plant parts would be prone to much longer exposures at much smaller concentrations. The converse is also right - the lack of response to a short-term exposure to high concentrations does not mean that the species or plant part has shown similar effect if there is long-term exposure at low concentrations [79].

Alkanes and monoaromatic compounds (benzene, toluene, xylene, ethylbenzene etc.) has shown to have no direct effects on plants at ambient concentrations, even in grossly polluted air, but several papers have reported the damaging effects of vehicular exhausts on plants [80,81] or by exposure of plants beside motor ways (e.g. Sauter *et al.*, 1987). Similarly, though there is consensus that ethylene has no effect on photosynthetic process of higher plants, direct effects of ethylene applied externally have been observed at concentrations below 100 nl l<sup>-1</sup>. Dose response study using a wide range of ethylene concentrations showed large stimulation of oat and rice mesocotyl growth at 100 nl l<sup>-1</sup> (the lowest concentration tested) for some less days. The potential uptake of chlorinated VOCs to cause deleterious effects was confirmed by several short-term laboratory experiments, using large air concentrations. Experiments with halones 1211 (CBrClF<sub>2</sub>) and 1301 (CBrF<sub>3</sub>) at concentrations as low as 1 nl l<sup>-1</sup> over 45 days have shown prominent changes in photosynthetic pigments and in protein content, as well as increased activity of the glutathione S-transferases. Trichloroacetic acid (TCAA) was widely used as herbicide in the 1950s and 1960s, and its foliar accumulation potential was consequently identified as a likely causal factor for plant damage. In in-vitro study, lower concentrations of formaldehyde in solution (0.3 mM) were shown to affect Douglas fir pollen, and exposure to gaseous formaldehyde has been observed to affect the growth of bean plants [82].

In the atmosphere there are various oxidizing and non-oxidizing pollutants present, among them ozone, NO<sub>x</sub>, SO<sub>x</sub>, particulates and VOCs is by far the most significant for plant, because of its toxicity, wide spreads occurrence and increasing incidents. There is vast range of VOCs of anthropogenic and natural origin, the later including emission from plant, animal and microbial sources [83]. So far as effects on plants are concerned, overwhelming interest is in the concentration of those VOCs which have high potential to generate phytotoxic levels of photochemical oxidants. Thus direct impact of VOCs on plant life has received relatively little attention. Let's discuss some of the important class of VOCs like **ethylene**, **Trichloroacetic acid (TCA)**, **Nitrophenols** (automobile exhaust) and **Benzene**.

### Ethylene

Ethylene is an extremely unusual pollutant in that it is a natural plant hormone involved in wide range of processes, such as growth regulation, flower development, fruit ripening, senescence and abscission of organs. Interestingly this compound is popularly known as **fruit ripening hormone** but on the contrast there is small but significant literature concerned with ethylene as a **pollutant**. The major source of ethylene in the atmosphere is mainly by automobile exhaust as it has been estimated that 80 ppb may be present on busy roads in Texas (US) [84], even though catalytic converters

reduce emissions by 80%. The above reported data predicts that if this is this condition of emission of ethylene in the atmosphere by vehicular exhaust in such a developed country than anybody can expect the condition of the same compound in the developing country like India. Surprisingly diurnal as well nocturnal pattern of its concentration closely follows high peak patterns in the morning rush hours in the Texas (US) [84]. There are various reports suggested by various authors that damage to plant cover which occurs in 1864, with description of defoliation of street trees in Germany, due to escape of illuminating gas from underground pipes [85]. On the other hand problem were also arising in the glass-houses where gas was used as fuel for light, damaging agent was identified as **ethylene** by a Russian plant physiologist in 1901 [85]. According to Abeles [84], who noted that dose/response relationship for different effects and species tended to follow the same curve that is, 1-10 ppb: No Effects; 10-100 ppb: Discernible effects; 100-1000 ppb: half maximal effect and 1000-10000 ppb: saturation. The common symptoms which occur after exposure of ethylene to plants are inhibition of bud growth, abscission of various organs, reduced flower and fruit production, yellowing of conifer needles, premature opening of broad leaf tree flowers, inward rolling or twisting of leaf, ion monocotyledons, chlorosis or red or purple discoloration leaf, induction of roots from stem, bushy growth habit and premature fruit ripening in broad leafed plants. A vary common response to ethylene is **epinasty**, which can occur at very low ethylene concentrations, although this may not necessarily have an adverse effect on the plants. The failure of flowers on plants, can represent a serious economic loss, with **carnation** representing a particular problem; there have been report of their flowers remaining in bud (sleepy) in florist shops near traffic intersection in a narrow Dutch city [84]. This report serves as a special attraction for experts for using carnation as "**Biomonitor**", indicator plant species. However, with growing motor vehicles emission in the developing world it would seem likely that ethylene may well be having major impacts on vegetation in and near their urban areas, but this has not been seriously investigated.

### Trichloroacetic acid (TCA)

TCA is formed in the atmosphere as a product of the breakdown of C1 and C2 halocarbon. Symptoms of TCA toxicity have been observed: curling of the needles, increase in the number plastoglobules, reduced number of thylacoids and inhibition of wax layer development [86]. The principle pathways for the formation of TCA are believed to be the reaction of 1, 1, 1-trichloroethane and tetrachloroethene with hydroxyl radical in the atmosphere. In addition to direct deposition, TCA may enter the transpiration stream from soil following washout, as results of its high water solubility. TCA is also formed within plant cells as a result of the detoxification of the dry deposition of C<sub>2</sub> volatile chlorocarbons by Monoxygenase P450 [86].

Reported concentration of TCA was found in between 10-130 ngg<sup>-1</sup> in Scots pine needles in Finland [87]. Norokorpi and Frank [86] also reported a significant increase in loss and chlorosis of pine needles in Finnish forest in relation to TCA, with two distinct populations: a resistant group with a gradient of 0.32% defoliation per unit (ngg<sup>-1</sup>) TCA and 0.78% per unit for the sensitive population. One possible reason for these population differences may be the enzyme activities of glutathione transferase, monoxygenase P450.

### Nitrophenols

Automobile exhaust is primary source of nitrophenols, and they are also formed in the atmosphere by the reaction of benzene and alkylated aromatics with hydroxyl radicals and NO<sub>x</sub> [88]. Luttke et al. [89] proposed that mononitrophenols are principally emitted from car exhaust, while the dinitrophenols are formed in the atmosphere in the liquid phase. Gaseous concentrations of all the compounds are always higher than in the liquid phase, with 4 nitrophenol the most abundant compound with a concentration range of 14-70 ng m<sup>-3</sup>, while the other compounds are in the range of 1.2-30 ng m<sup>-3</sup>. These values were at a background (away from emission sources) site, but at a site, influenced by urban discharge the cumulative mononitrophenols are 0.35 nmol m<sup>-3</sup> and the cumulative dinitrophenols are 0.09 nmol m<sup>-3</sup> [88]. Values of 2, 4 dinitrophenol in rain at an Austrian background site was 1.61 µg l<sup>-1</sup>, but 2, 4 dinitrophenol was often only 20% of the total nitrophenol burden. The nitrophenols are suggested to be involved in forest decline by Rippen et al. [90], while Shea et al. [91] observed phytotoxic effects of 2, 4 dinitrophenol at 20 to 200 µg l<sup>-1</sup>. Nitrophenols are readily taken up by plants from both the gaseous and liquid phase. Weiss et al., [92] recorded a mean concentration of 60 µg kg<sup>-1</sup> for total nitrophenols in pine needles at background sites in Austria. Natangelo et al. [93] found higher total nitrophenol concentrations in leaves of a more damaged forest site (313 ng g<sup>-1</sup>) compared with 148 ng g<sup>-1</sup> at non-damaged site.

### Tolerance Mechanisms with Respect to VOCs on Plants

There are various pathways of tolerance mechanisms when plants are exposed to VOCs. One particular path of enquiry has been the action of glutathione-S-transferases (GST) which conjugates xenobiotics with glutathione: these conjugates are non toxic and are subsequently metabolize. This process has been widely studied in relation to herbicide type of compounds like TCA. Wolf [94] found that damaged trees on the Wank Mountain in Bavaria had different GST isozyme to that in the healthy trees and proposed that the damaged trees may lack the "right" isozyme to detoxify the xenobiotics they were exposed to. Glucosyl transferases have been shown to conjugate chloro and nitrophenols to glucosides. The antioxidant enzyme SOD (superoxidase dismutase) and AP (ascorbate peroxidase) are elevated following exposure to formic and acetic acid. It could therefore be hypothesized that these VOCs pollutants put the plant in the stress as a result to the redirection of their normal metabolism to increase synthesis of detoxification enzymes.

### Effect on Human Health

Many VOCs can affect human health by virtue of their carcinogenic, mutagenic or reproductive effects. In addition they may be toxic by inhalation or ingestion. The principal source of data relating to hazards to human health is the CHIP (Chemicals Hazard Information and Packaging) approved Supply List (ASL).

The health hazard categories in CHIP that are relevant to human health and the environment are very toxic, toxic and harmful, in association with a risk phrase to denote the pathway of harm, such as **inhalation** or **ingestion**. In addition the substances can be classed as **Carcinogenic**, **Mutagenic** or a **Reproductive Toxicity** (abbreviated to

reprotoxin) and graded from 1 to 3, 1 being the most harmful. In the previous categorization the CHIP regulations included teratogenic properties rather than reproductive toxicity. Whilst not directly synonymous, the former being a subset of the latter, reproductive toxicity data have become the standard measure of this form of toxicity. One additional selection criterion was used to support the CHIP data, namely the International Agency for Research on Cancer (IARC) classification of carcinogenicity to humans. There are three categories in the IARC classification, 1 (carcinogenic to humans), 2A (perhaps carcinogenic to humans) and 2B (possibly carcinogenic to humans). Category 1 and 2A substances were considered high risk and category 2B substances were considered medium risk.

The exhaust of gasoline and diesel from vehicles emit significant concentrations of about two dozen VOCs, the most important of which are benzene, 1, 3-butadiene, m- and p-xylenes (typically measured together), o-xylene, ethylbenzene, toluene, and formaldehyde. The EPA has designated many VOCs, including those typically found in automobile exhaust (pollution), as air toxics or hazardous air pollutants (HAPs), which are known to cause serious health hazards. Both benzene and 1, 3-butadiene are declared carcinogens, and other VOCs, including formaldehyde, are suspected carcinogens. It is difficult to directly link natural ambient exposure to VOCs to any individual cancer case, because there are multiple reasons acting individually or with each other to cause cancer. So, the carcinogenic effects of VOCs are associated with either individual or cumulative exposures.

The U.S. EPA classifies *benzene* as a known human carcinogen by all routes of exposure. Most prominent effects of benzene according to its toxicity profile are carcinogenicity, mutagenicity and chronic toxicity. The main targets of toxicity are the hematopoietic system and the cells of the bone marrow. Exposure routes to be considered at the workplace are inhalation against benzene vapour and skin contact with the liquid substance. An oral uptake of approx. 15 ml benzene by humans (176 mg/kg bw) can cause collapse, bronchitis and pneumonia. The direct aspiration of liquid benzene into the lungs causes instantaneous pulmonary oedema and haemorrhage at the site of contact with the pulmonary tissue. Very high concentrations of benzene produce narcotic effects and can lead to death by respiratory arrest. Fatal effects can occur after inhaling a benzene concentration of 65 mg/l for 5-10 minutes. Exposure of 30 minutes to benzene concentrations of 25 mg/l can be dangerous to life threatening. After inhalation exposure to 0.16-0.48 mg/l for 6 hours headache and lassitude occur while after inhalation of 0.08 mg/l for 6 hours no acute toxic effects were documented. The odour threshold is reported to be 4.8 mg/m<sup>3</sup> (1.5 ppm). In a report on three fatalities of acute benzene poisoning by acute dermal and inhalation exposure second degree chemical burns to face, trunk and limbs, haemorrhagic lungs and pulmonary oedema were documented. A relationship between chemical burns and death was not mentioned. Benzene is absorbed by all routes (inhalation, dermal and oral) where inhalation is the most important route of exposure. Benzene is rapidly distributed in the body and higher concentrations are found in fatty tissues compared to blood. After absorption via inhalation, the dermal or the oral route, most of benzene is metabolized and the metabolites are excreted after phase-II-conjugation

mainly in the urine. Oxidative metabolism of benzene is a condition for producing toxicity in animals and follows similar pathways in humans and animals. The **liver** is the major site of benzene metabolism, but in the bone marrow may be associated with the **haematotoxic** and **leukaemogenic** effects of benzene. Benzene exposure may induce changes in chromosomes, blood cells, and bone marrow cells. The leukemia risk of children exposed to benzene is likely much higher than that of adults, even at lower levels of exposure. Because of its status as a known carcinogen, the World Health Organization sets no safe level for benzene exposure.

The EPA has designated many VOCs, including those typically found in auto pollution, as air toxics or hazardous air pollutants (HAPs), which are known to cause serious health hazards. Both benzene and 1, 3-butadiene are famous carcinogens, and other VOCs, as well as formaldehyde, are suspected carcinogens. It is difficult to directly link exposure to in-car VOCs to any individual cancer case. However, the carcinogenic effects of VOCs are associated with individuals, cumulative exposures. The U.S. EPA categorizes benzene as a well known human carcinogen by all routes of exposure and multiple studies have linked inhaled benzene to the development of leukaemia. Additional studies suggest that benzene exposure may induce changes in chromosomes, blood cells, and bone marrow cells. Most of the studies on benzene carcinogenicity have looked at the occupational exposure of adults. The leukaemia risk of children exposed to benzene is probable much more as compare to adults, even at lower levels of exposure [95]. EPA classifies both 1, 3-butadiene and formaldehyde as probable human carcinogens. Animal and human studies, while not conclusive, have shown that exposure to 1, 3-butadiene, may be responsible for respiratory, bladder, stomach, lymphatic, and blood related cancers. According to the EPA, limited human studies have reported an association between formaldehyde exposure and lung and nasopharyngeal cancer. Animal inhalation studies have reported an increased incidence of nasal squamous cell cancer [96]. One animal study suggests that ethylbenzene exposure may be associated with the formation of tumors. However, this study was extremely limited and the few studies involving humans have shown no elevated cancer risks. EPA declared that ethylbenzene is not classifiable as to human carcinogenic [96]. Other VOCs may also promote the growth of cancerous cells in humans, but convincing medical research is still lacking. The first studies to measure the levels of VOCs within automobile passenger compartments took place in the late 1980s and early 1990s. These evaluations of cars on predominantly urban roads in Los Angeles, Raleigh, Boston, and New York/New Jersey found average concentrations of benzene ranging from 13.6 to 50.4 mg/m<sup>3</sup>. Toluene concentrations ranged from 33.3 to 158.0 mg/m<sup>3</sup>, ethylbenzene from 5.8 to 11.6 mg/m<sup>3</sup>, m- and p-xylene from 20.9 to 154.0 mg/m<sup>3</sup>, o-xylene from 7.3 to 16.0 mg/m<sup>3</sup>, and formaldehyde from 0.2 to 13.7mg/m<sup>3</sup>. The high concentrations out of one of the two Los Angeles studies for all of the pollutants, except ethylbenzene and o-xylene, which Los Angeles studies did not consider. The low concentrations in the ranges for all of the pollutants except formaldehyde, which was measured at the lowest average concentration in

the New York/New Jersey study. The research completed in Raleigh also reported average concentrations inside cars during highway driving: 9.9 mg/m<sup>3</sup> for benzene, 34.5 mg/m<sup>3</sup> for toluene, 6.7 mg/m<sup>3</sup> for ethylbenzene, 23.1 mg/m<sup>3</sup> for m- and p-xylene, and 8.6 mg/m<sup>3</sup> for o-xylene. During suburban driving, the New York/New Jersey study found average concentrations of 13.4 mg/m<sup>3</sup> for benzene, 51.2 mg/m<sup>3</sup> for toluene, 10.1 mg/m<sup>3</sup> for ethylbenzene, 29.2 mg/m<sup>3</sup> for m- and p-xylene, 12.5 mg/m<sup>3</sup> for o-xylene, and 0.4 mg/m<sup>3</sup> for formaldehyde. In both of these studies, in every case apart from formaldehyde, the in-car levels were significantly higher during urban driving than during suburban or highway driving [97]. The New York/New Jersey study also showed that improperly maintained vehicles may have considerably more VOC levels in-car than well-maintained vehicles. A vehicle in the check with a malfunctioning carburettor, under some driving conditions, recorded more than 5 times the toluene, 12 times the benzene, 44 times the ethylbenzene, 23 times the m- and p-xylene, and 40 times the o-xylene found in a properly maintained vehicle on the same suburban test route. With reference to the studies performed on the basis of occupational exposure like on petrol pumps have also tremendous effect on human health. The level of exposure in petrol pumps is largely dependent on the technical specifications of gasoline, especially it's benzene content, and to the emission control technology, such as vapour recovery systems in operation. Monitoring of the service station workers revealed that the average exposure level for benzene and toluene were 3.9 and 5.5 fold more than the ambient air. The integrated lifetime cancer risks due to benzene, ethylbenzene, formaldehyde and acetaldehyde and the overall hazard index due to chronic exposure to some hazardous volatile organic compounds are 1.48E-04 and 2.3 signifying the prospect of cancer as well as chronic health effect on the workers exposed [98]. Published evidence indicates that the most serious risks from occupational exposure to benzene are aplastic anemia and leukemia, which appear to be associated with prolonged rather than acute exposure [99]. The limited knowledge available on the occupational dose-response relationship with respect to benzene derives from the clinical assessment of people working in environments periodically subjected to air analysis. Such measurements are not very satisfactory for, assessing the cumulative exposure to a volatile liquid which may produce transient and relatively high concentrations in the work environment. Several studies worked on breath analysis with respect to integrated exposure of many volatile solvents, including benzene [100, 101, 102]. The analytical methods used for investigations of benzene in these studies have not been found to be satisfactorily responsive to be suitable for the breath concentrations associated with the national and international authorities, and a more sensitive method has been developed for this purpose. Before the health risks from benzene can be properly determined, it is important to obtain information on retention of benzene in tissues after single and repeated exposures and facts related to it's rate of efflux from tissues after removal from exposure. Some information can be obtained from men occupationally exposed to benzene, but it is also necessary to include laboratory studies with human volunteers exposed to known benzene concentrations for measured periods. Such investigations also permit an estimate of the relationship of previous benzene exposure and their concentration in breath; this information will enable an index of exposure based on

breath analysis to be sustained by toxicological data obtained under known environmental conditions and to be related to currently proposed air standards. There is some evidence that chromosome changes may be observed in person exposed to benzene for a long periods of time and can exhale about 50 ppb benzene in morning breath [98].

#### iv) Particulate Matter

It is estimated that more than 1 billion people are exposed to outdoor air pollution annually. According to Shah et al. [103], urban air pollution is responsible for more than 2 million deaths per year globally due to the damage in respiratory system and out of which, 2.1 million are the result of particulate matter.

Airborne PM is a heterogeneous mixture of solid and liquid particles suspended in air that changes constantly in size and chemical composition in space and time. PM is usually classified as  $PM_{2.5}$  and  $PM_{10}$ , i.e., the fraction of the total PM with an aerodynamic diameter less than 2.5 and 10  $\mu m$ , respectively. Sources of PM are both natural and anthropogenic and emitted directly as primary particles or from in the atmosphere from the gaseous precursors. Primary particulate matters are emitted directly from emission sources and they often associated with combustion sources. Secondary particulate matters are formed in the atmosphere by various photo-chemical reactions from primary pollutants like  $NO_x$ ,  $SO_x$ , and VOCs [104].

Several physiological, epidemiological and toxicological studies reported that exposure to ambient particles may be health concern to human being [105, 106, 107] and also for plants [108, 109]. Fine PM can easily enter deep into the lungs and cause many health related problems due to the presence of sulfates, nitrates, acids, metals, and carbon particles onto their surfaces.

Several studies also reported the exposure of particulate matter and related health problems such as nonfatal heart attacks, aggravated asthma, irregular heartbeat, decreased lung function, and respiratory disease [105, 106, 107].

On the basis of their penetration properties in to the lungs, USEPA classified PM in to fine ( $PM_{2.5}$ ) and coarse ( $PM_{10}$ ) mode particles is listed in Table 8. Fine particles with a diameter of less than 0.1  $\mu m$  are categorized as an ultra-fine particle ( $PM_{0.1}$ ) and very few studies are ongoing focusing on ultra-fine size fractions [104].

The source of the particulate matter can be direct in to the environment or indirect by the conversion from gaseous precursor compounds such as  $SO_2$ ,  $NO_x$ ,  $NH_3$  and NMVOCs emitted from anthropogenic and natural sources [106]. Anthropogenic sources includes coal combustion, diesel and gasoline oil combustion, biomass burning, industrial emissions, resuspension of soil dust erosion of pavement by road traffic, and abrasion of break and tires, while natural sources includes volcano eruptions, dust storm, forest fires and sea spray [107]. The chemical composition of the PM includes organic (OC) and elemental carbon (EC), particle bound water, metals such as cadmium, copper, nickel, vanadium, and zinc, inorganic ions (sulphate, nitrates, ammonium, sodium, potassium, calcium, magnesium, and chloride) and polycyclic aromatic hydrocarbons. (PAHs) [103]. Apart from the different chemical constituents biological constituents such as allergens and microbes are also associated with PM.

Traffic, industrial emission, and emissions from thermal power plants and coal combustion are the major contributors of the PM in ambient atmosphere [110], also produced from the wear of vehicle components such as brakes and tires. PM often contains several mineral components such as silicon (Si), aluminium (Al), potassium (K), sodium (Na), calcium (Ca), copper (Cu), antimony (Sb), lead (Pb), cadmium (Cd), and zinc (Zn) [106, 107]. Several author also mentioned various toxic organic compounds are also found associated with PM such as Dioxin and furans polycyclic aromatic hydrocarbons [109, 110, 111]. The small size of PM (fine to coarse) tends to be suspended in to air for long period of time from week to months (mentioned in Table. 8) and can be transported to a large distance (100 to 1000 km). The PM different fractions can varies from one day to next day in the

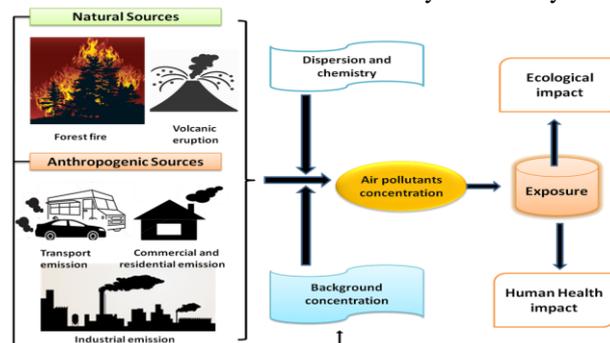


Fig. II: The relationship between PM sources, exposure and their ecological and human health impacts

influence of meteorological conditions such as wind pattern and atmospheric stability [111].

#### Impact of PM on plant health

On the other hand PM also play a significant role in ecological effect. Several studies reported that airborne aerosol ( $PM_{10}$  and  $PM_{2.5}$ ) show a good agreement with traffic, coal and biomass burning related pollutants and have significant ecological impact [108]. PM effect vegetated system at different levels such as organism, population, community and ecosystem levels. PM can affect the different plant parts such as root-shoot length, structure and colour of leaves. It also effect protein content, chlorophyll, photosynthesis, relative water content and transpiration and it depends on type of sources of PM. Several physio-chemical changes were observed in the areal parts of the plant after PM deposition [109]. PM reduces the rate of transpiration and rate of evaporative cooling by covering the most of the area of stomata on leaf surfaces [105]. Leaf chlorosis also caused by dust pollution due to its effect on chlorophyll biosynthesis [106]. Several authors work on different plant species and reported the impacts of PM on their physiology, morphology and biochemistry [112, 113, 114]. Chen et al. [115] worked in industrial area and found that PM affect the Loss in relative water content, total chlorophyll, pH for many plants species in China. Alyemini and Almohisen [116] reported that the plant situated in the area of heavy traffic and industrial area shows Bioaccumulation of heavy metals in plant parts. Several other studies also reported chlorophyll degradation which ultimately affects photosynthesis, protein degradation, and can affects the biochemical parameters [117, 114, 118].

**Impact of PM on human health**

The relationship between PM sources, exposure and their ecological and human health impacts are mentioned in the Fig.2. Detrimental effects of PM have been reported worldwide. Inhalation of PM causes risk of mortality. Long-term exposures to PM have been linked with decline in life span, due to cardiopulmonary mortality and lung cancer, among other diseases. Several epidemiological studies works on particulate matter (PM<sub>10</sub> and PM<sub>2.5</sub>) with their effects on human health such as respiratory problems, premature mortality, hospital admissions, aggravated asthma, decreased lung function. Similar to other pollutants, children (respiratory system still in developing period), old persons (having declining physiological reserves) and the adult person (who spend more time in outdoor environment) are more sensitive to the cardio respiratory decease. Apart from above mentioned, individuals having some heart or lung related diseases are also very prone to particulate matter. PM<sub>2.5</sub> is of high concern from health point of view due to their association with several toxic metals, and acids and PAHs. They can also penetrate deeper in to the lungs due to their very small size.

According to Pope et al [119] PM<sub>10</sub> concentration and cardiopulmonary and lung cancer mortality is interrelated, the relationship was stronger for PM<sub>2.5</sub> as compare to PM<sub>10</sub>. Several authors also reported the association between PM<sub>10</sub> exposure and increase in bronchitis, chronic cough, and respiratory problems in individuals with chronic obstructive pulmonary disease (COPD) [119, 120]. PM<sub>10</sub> was also found associated with a raise in hospital admission of the elderly for COPD and asthma and lower respiratory tract illness including bronchitis and pneumonia [118]. In two different studies authors reported that upper and lower respiratory indicator increased with PM<sub>10</sub> levels [119, 121, 122].

Overall, PM produced from the different anthropogenic as well as natural sources either directly or indirectly in to the atmosphere and can affect the ecological and human health adversely. Which ultimately leads to several physio-chemical, morphological, and biochemical changes in plants and causes various diseases in human, some time leads to death.

**iv) Oxides of Carbon**

Oxides of carbon consists of carbon dioxide (CO<sub>2</sub>) and carbon monoxide (CO). The main source of oxides of carbon is coal and it produces due to partial and complete combustion. One of the oxides is CO<sub>2</sub> which is a colourless gas that is denser than air. After inhalation, at high concentrations, this gas produces sour taste and stinging sensation in nose and throat [123]. CO<sub>2</sub> is an important greenhouse gas and its concentration has increased drastically since industrial revolution.

It gets fully oxidised, therefore not very reactive. CO<sub>2</sub> has the property to slightly soluble in water and it forms a weak acid i.e. carbonic acid. The sources of CO<sub>2</sub> are generally related to the combustion of fossil fuels. Fuels often contain impurities and these are released along with CO<sub>2</sub>. The industrial sources include hydrogen and ammonia production and any acid neutralisation process that utilises limestone. Carbonated drinks, fire extinguishers and various areas of the food industry are also the sources of CO<sub>2</sub>.

Natural sources also contribute up to a large portion in the atmosphere. These include volcano eruptions, various rock types contain carbonated minerals, which when comes in contact with acidic water, it may release CO<sub>2</sub> into the atmosphere. CO<sub>2</sub> is also released by plants during night, though it takes in CO<sub>2</sub> during the day. The rain forests release carbon content of the vegetation in the form of CO<sub>2</sub> [124].

**Table 8. Classification and source of PM**

Fraction	Size range, atm. Lifetime and travel distance (km.)	Source and characteristics	Formation Processes	Sampling measurement	Removal Processes
Coarse mode particles (PM <sub>10</sub> )	≤10 μm diameter Known as the thoracic fraction.  *Minutes to hours and can travel 1 to 10 kms	Resuspension and suspension of roads and street dust, industrial dusts; and disturbed soils, e.g., farming, mining coal and oil combustion, and ocean spray. Resuspended of construction dust, soil dust, street dust; coal and oil fly ash; metal oxides of Si, Al, Mg, Ti, Fe, CaCO <sub>3</sub> , NaCl, sea salt; pollen, mold spores, and plant parts.	Mechanical disruption (crushing, grinding, abrasion of surfaces), Evaporation of sprays, Suspension of dusts, and Reactions of gases in or on particles	Particles measured by a sampler that contains a size fractionator (classifier) designed with an effective cut point (50% collection efficiency) of 10 μm aerodynamic diameter.	Dry deposition by fallout and Scavenging by falling rain drops
Accumulation mode or Fine particles (PM <sub>2.5</sub> )	≤2.5 μm in diameter These particles can penetrate into the deeper lungs and consequently may initiate host of respiratory problems and even premature death. *Days to week and can travel 100 to 1000 kms.	Combustion of coal, oil, gasoline; transformation products of NO <sub>x</sub> , SO <sub>2</sub> , and organics including biogenic organics, e.g., terpenes; high temperature processes; smelters, and steel mills	Condensation, Coagulation, Reactions of gases in or on particles, Evaporation of fog and cloud droplets.	Particles measured by a sampler that contains a size fractionator (classifier) designed with an effective cut point (50% collection efficiency) of 2.5 μm aerodynamic diameter	Forms cloud droplets and rains out and Dry deposition
Ultrafine particles (UFP)	≤0.1 μm with high surface area per unit mass. Week to months and can travel < 1 to 10s of km	Carriers of harmful gaseous compounds. They have ability to penetrate tissue and may be absorbed directly into the bloodstream. It may cause various diseases pulmonary inflammation and hemorrhage, interstitial oedema etc.	Nucleation, Condensation and Coagulation		Grows into accumulation mode Diffuses to raindrops

**Source:** Atkinson et al. [110], Cheung et al. [111]

Natural sources also contribute up to a large portion in the atmosphere. These include volcano eruptions, various rock

types contain carbonated minerals, which when comes in contact with acidic water, it may release CO<sub>2</sub> into the atmosphere. CO<sub>2</sub> is also released by plants during night, though it takes in CO<sub>2</sub> during the day. The rain forests release carbon content of the vegetation in the form of CO<sub>2</sub> [124]. According to PBL NEAA Report [125], the six largest CO<sub>2</sub> emitting countries in 2014 were: China (30%), US (15%), EU (9.6%), India (6.6%), Russian Federation (5%) and Japan (3.6%). Significant trends were seen in the top three emitting countries which account for 54% of total emissions. In China and US, emissions increased by only 0.9% whereas European Union showed a large decrease of 5.4% in 2014 as compared to 2013 which counteract the 7.8% growth in India. The Russian Federation and Japan saw their CO<sub>2</sub> emissions decline by 1.5% and 2.6% respectively.

**CO (carbon monoxide)** is a colourless, odourless and tasteless gas. It is somewhat denser than air and slightly soluble in water. It is actually a primary pollutant and can be oxidised to form carbon dioxide (CO<sub>2</sub>). Hence, it gives CO a relatively short residence time despite its emissions being intense over shorter periods of time. CO is poisonous in large concentrations and in limited spaces it can result in asphyxiation and death. CO is formed as a by product of incomplete combustion of fossil fuels [122]. Though primarily formed in internal combustion engines of motor vehicles, any combustible material has the possibility to release CO such as solid waste incineration, domestic use of biofuels and biomass burning. Industrial processes are also responsible for the production of CO. In South Africa, CO is generated primarily by the use of biofuels as it is the only source of energy available for meeting the demands of its rural population [123]. Natural sources include volcanic eruptions. Besides combustion, natural reactions may also lead to the formation of CO by the oxidation of the other carbon based gases e.g. methane. Decomposition process of organic material within soil and forest floors can result in the release of CO as well. The emission rate in Canada of CO in 2014 attained 6381 kilotonnes (kt), an raise of 102 kt (2%) from 2013 levels. Emissions of CO were lower in 2014 than 1990 i.e. 10360 kt (62%). The two main sources of CO emissions in 2014 were transportation and off-road vehicles, representing 59% (3737 kt) of total national emissions (6381 kt). The largest decline in emissions between the years 1990 and 2014 occurred in transportation with an emission reduction of 5442 kt. In Canada, Quebec emitted the highest percentage of Canada's CO production, signifying 27% (1701 kt) of the national total (6381 kt). The other city Ontario contributed 23% (1494 kt) to national production and Alberta contributed 19% (1219 kt). Domestic firewood burning was the most important contributor of CO release for Quebec, off-road vehicles for Ontario and oil and gas industry for Alberta. All provinces experienced sharp reductions in emissions between 1990 and 2014 largely due to transportation emissions reductions [125].

### ***Effect on Plant Health***

In case of CO<sub>2</sub>, with an increase in concentration of CO<sub>2</sub>, the temperature throughout the earth also increased. This will transform the fertile land into desert and other types of drylands. While deserts increase in size, other eco-zones, however tropical forest and grassland will struggle to drift towards the poles. Intense storms with increased rain

throughout the world is due to global warming but increased water levels doesn't soak down the ground and floods cause damage to the crops. An enhanced water level in the form of floods goes into creeks, which carrying off large amounts of soil and fertilizer.

CO<sub>2</sub> concentrations above ambient consistently increase net photosynthetic rates in the short term due to improved water use efficiency. The anatomical changes such as increase in leaf thickness and alterations in cell and chloroplast development have been observed under elevated CO<sub>2</sub> in plants. Carbon dioxide enters the plant leaves through stomata. The closing or opening of stomata is regulated by CO<sub>2</sub> concentration [126]. High concentrations of CO<sub>2</sub> change the turgor pressure of guard cells hence mediating the closure of stomata. Elevated atmospheric CO<sub>2</sub> generally reduces stomatal conductance. High concentrations of CO<sub>2</sub> induce closure of the stomata thus limiting CO<sub>2</sub> assimilation and fixation rate [127]. The stomatal closure is related to loss of membrane permeability because of oxidation of membrane channels and transport proteins, or increased sensitivity of the stomata to closure signals such as internal Ca levels or abscisic acid. Increase in atmospheric CO<sub>2</sub> concentrations increase photosynthetic rate because of high Rubisco activity. Increased availability of CO<sub>2</sub> as the substrate leads to more carbon assimilation and hence more photosynthesis. Long term exposure to increasing CO<sub>2</sub> concentration leads to reduction in Rubisco activity or level due to the amount of Rubisco required for maintaining the same assimilation rate decreases. Due to the diminished photorespiration rate, the energy demand per fixed carbon decreases and consequently, the demand for compounds involved in light reactions, such as chlorophylls or carotenoids also decreases. All these alterations result in enhanced rates of respiration. The possible changes in respiration may be due to structural changes imposed by elevated CO<sub>2</sub>, accumulation of carbohydrates or changes in the biochemistry of respiration. Stimulation in photosynthetic efficiency is majorly reflected in C3 species [126]. Species with the C4 pathway are able to concentrate CO<sub>2</sub> at the site of carboxylation for photosynthate production.

High concentrations of CO which is an important signalling molecule in physiological activity, the inhibitory effect of a higher ratio of CO to O<sub>2</sub> (<4:1) on cytochrome c oxidase in the mitochondrial ETC at cellular level or produce effect on the detoxification enzyme P-450 in monooxygenase system and multiprotein complex proteins in chloroplasts. CO also have significant signalling role as well as toxic effects, but this depends upon the amount produced, as plants also contain a variety of hememoiety-conating proteins and produce a number of important biliverdin-related tetrapyrrole pigments such as phycocyanobilin and phytochromes. CO is also proven to be one of the most important cellular components which regulate the biological processes in various plant species. CO arises in biological systems during heme degradation as the oxidation product of  $\alpha$ -methene bridge of heme and this process is catalyzed by heme oxygenase enzymes). It affects the seed germination process, root development [127]. According to Cao et al. [128] it is also induces stomatal closure. Plant species develop inducible defence systems to survive biotic and abiotic threats, thus producing a large variety of defence-related hormones to unlock the defense-related regulatory networks. It is also

generated against oxidant damage under salt stress, abiotic stress for example drought stress, and heavy metal stress.

### ***Effect on Human Health***

The proportion of CO<sub>2</sub> in atmosphere is about 0.035%. OSHA has set a permissible exposure limit of 5000 ppm over an 8-hour work day. Similarly, the American Conference of Governmental Industrial Hygienists threshold limit value is 5000 ppm for the same duration, with ceiling exposure limit of 30,000 ppm for a 10-minute period based on acute inhalation data [129]. At 40,000 ppm it becomes dangerous to life and 30 min exposure to 50,000 ppm generates intoxication, and levels greater than that (7-10%) produce unconsciousness (NIOSH 1996). The acute toxicity data show the lethal concentration for CO<sub>2</sub> is 90,000 ppm (9%) for 5 minutes [129]. Normal levels of CO<sub>2</sub> are considered to be harmless but high concentrations lead to dizziness, disorientation, suffocation and extreme can cause death. Death occurs when there is a depression of central nervous system with prolonged exposure to high concentration of CO<sub>2</sub> and the body's reparatory mechanisms are overwhelmed or fail. It is a potential inhalant toxicant and an asphyxiate. It enters the body from air through lungs and gets dissolved in blood and causes acid-base imbalance, or acidosis which causes subsequent CNS. The main cause of acidosis is excess of CO<sub>2</sub> in blood. Under normal physiological conditions, there is high concentration of CO<sub>2</sub> in blood than in lungs, forming a gradient, where blood CO<sub>2</sub> diffuses into lungs and exhaled. Increase in inhaled CO<sub>2</sub> and immediate reaction with water in blood forms carbonic acid (H<sub>2</sub>CO<sub>3</sub>) which finally transformed into hydrogen ions (H<sup>+</sup>) and bicarbonate (HCO<sub>3</sub><sup>-</sup>) [130].

Carbon monoxide is another stable gas which is formed due to incomplete combustion of CO<sub>2</sub> in atmosphere; lungs are only important route for environmental exposures. CO when combines with haemoglobin to form carboxyhaemoglobin (COHb) which can be measured either spectrophotometrically or through GC methods. In healthy people, small amount of CO are formed endogenously from the catabolism of haemoglobin and other haem proteins. At stationary phase and without exogenous exposure, this results in COHb saturation of 0.4-0.7%. In some conditions, endogenous carbon monoxide production may be abnormally high, which increases the risk of high total exposure in polluted environments. In pregnancy conditions, increased concentration of maternal COHb is 0.7-2.5% have been reported, and the fetuses of non-smoking mothers have also shown of high levels of 0.4-2.6% [41]. When CO reaches lungs, CO diffuses rapidly across the alveolar and capillary membranes and even it crosses placental membranes. CO binds reversibly to one of the haem proteins. Approximately 80-90% of absorbed CO binds with haemoglobin, which causes a reduction in the oxygen-carrying capacity of blood. The affinity of haemoglobin for CO is 200-250 times that for oxygen, on the other hand it has relative affinities with other haem proteins, cytochrome oxidase and cytochrome P-450 for CO are much less [41].

## **3. Methods and Policies to control air pollutants**

### ***a. Particulate Matter***

Filter papers of different material are being used for trapping particulate matter by passing air through a filter holder/sampler containing filter paper. Glass fiber filter paper, quartz filter paper, asbestos-cellulose, PTFE filters are the most common filters used for the sampling for the PM. Electrostatic precipitators are the most common to use for the control of PM in power plants. In the Electrostatic Precipitator (EP) when fly ash particle travel between electrodes, they take the charge from electrostatic surface in the effluent stream. The Performance EP depends on various parameters such as size of the particle and chemistry, properties of gas (flow rate, temperature, and moisture), strength of the electric field [131].

### ***b. Sulphur***

According to William [8] reduction in the level of sulphur can be done in many ways (i) by reduction in the contribution of sulphur in hydrocarbon (ii) by using low-sulphur fuel (iii) by removing sulphur from effluents. Switching and cleaning of the coal are other significant options for reducing sulphur content. Emission of sulphur can also be greatly reduced by switching and cleaning of fuel. Switching from soft coal (high sulphur content) to bituminous coal (low-sulphur content) can considerably decrease sulphur discharge into the atmosphere. Renewable energy sources such as solar power, wind energy, nuclear energy, and natural gas can also reduced total sulphur emissions.

Cleaning, washing and gasification of the crushed coal can reduced the sulphur and metal content before combustion as in case of 'IRONBRIDGE' Power Station (EON's). Sulphur emission can also be reduced up to 90 percent by mixing crushed limestone with coal before it is fed into a boiler. The new technology for the burning, named fluidized bed combustion has several advantages for pollution control. Apart from the above mentioned, flue gas desulphurization and sulphur recovery are the methods used for reducing sulphur content after combustion and production of elemental sulphur, sulphuric acid, or ammonium sulphate.

### ***c. Nitrogen Oxide***

The major sources of the oxide of nitrogen are vehicular emissions and biomass burning. To control the emission from the vehicle engine recirculation technique for gas inside engine can be followed [10]. This method depends on the dilution of air-to-fuel mixture burned; combustion temperature decreased, thereby decreasing the oxygen concentration in the mixture being burnt. After completion of the process the fuel gets richer and release lesser amount of the NO<sub>x</sub>. From more than past 20 years US has implemented this technology and become common in daily life schedule [10].

### ***d. Hydrocarbon and Carbon monoxide (HC and CO)***

Catalytic convertor is one of the most commonly used methods for the minimising hydrocarbon and carbon monoxides emissions from motor vehicles (Stoker and Seager, 1976; in Edward, 2002). This particular mechanism is used to convert carbon monoxides to carbon dioxides and

convert hydrocarbon to carbon dioxide and water. William [8] reported that closed systems can reduce many hydrocarbon emissions as it can prevent escape of fugitive gases (e.g. positive crankcase ventilation (PCV) systems in automobiles) drastically reduce hydrocarbon emission.

The US was first to add many amendments in several legislations, which help allot for the regulation of air quality. The Air pollution control Act (July 14, 1955) was the foremost clean air act which tackles the national environmental trouble. The basic objective of this act was related to impact of air pollution. This act governs the rights of the states and controls the air pollution with the aid of local government. Several amendments have been made in the present act including first and second which came in to force in 1960 and 1962. Furthermore, Clean Air Act was passed in 1963 which describe the principle of air quality. This act targeted on automobile emission and their standards from local sources. The 1965 amendments of Motor Vehicle Air Pollution Control Act came into the reality which established the vehicular emission standards. This amendment also recognized the serious problem of transboundary air pollution and promoted research on it's harmful effects on human health and welfare of Mexico and Canada.

The amendments of 1967 Air Quality Act divided into parts of the nation into Air Quality Control Regions (AQCRs) as a way to monitor ambient air. Under the same umbrella the government set up the national emissions standards for stationary sources. These standards established a fixed timetable for state implementation plans (SIPs), and suggested control technologies to achieve the ultimate goals of the SIPs. The amendments in 1970 were newly created The US-EPA to set the National Ambient Air Quality Standards and rewritten version of the original Clean Air Act. This is the Act to amend the Clean Air Act to provide for a more effective program to improve the quality of the Nation's air. It set National Ambient Air Quality Standards (NAAQS), to protect human health and benefit, and New Source Performance Standards (NSPS) that strictly regulated emissions of a new source entering an area. It was the monumental year of Clean Air Act regulation of various states for achieving and maintaining the National Ambient Air Quality Standards. Air pollution has been regulated a series of laws and regulation throughout the world after achieving the Clean Air Act, 1970. Therefore, a large number of laws and environmental protections regulation acts came into existence to control air pollution (Table IX).

**Table 9: Outline of legislation and amendments involved in air pollution control**

<i>Air Pollution Acts and Amendments</i>	
<i>Air pollution control Act of 1995</i>	<i>An act that offer research technical assistance relating to air pollution control. It was an act to make the nation more aware of this environmental hazard.</i>
<i>Amendments of 1960</i>	<i>Extended research funding for four more years</i>
<i>Amendments of 1962</i>	<i>These amendments enforced the principle provisions of the original act of air</i>

<i>Clean Air Act, 1963</i>	<i>An act to provide strengthen and accelerate programs for the prevention and amendments of air pollution This act also set the emissions standards for stationary sources such as power plants and steel mills.</i>
<i>Amendments of 1965</i>	<i>These amendments focused on establishing standards for automobile emissions</i>
<i>Amendments of 1966</i>	<i>These amendments imply local air pollution control program</i>
<i>Amendments of 1967</i>	<i>In this amendments state implementation plans and recommended control technology are involved. government also established national emissions standards for stationary sources</i>
<i>Amendments of 1969</i>	<i>Extended authorization for research on low emission fuel and automobiles.</i>
<i>Clean Air Act, 1970</i>	<i>This act provide an effective program which improve the quality of air throughout the country</i>
<i>Amendments of 1977</i>	<i>This amendments involves in motor vehicle emission standards</i>
<i>Clean Air Act, 1990</i>	<i>The Clean Air Act of 1990 addressed five main areas: air-quality standards, motor vehicle emissions and alternative fuels, toxic air pollutants, acid rain, and stratospheric ozone depletion</i>

## I. CONCLUSION

Air pollution is a burning issue in the world and need to be addressed in terms of their identification of different sources, effects and control and prevention for the same. The common problem which arises very generally is not only to identify the path of the source but to suggest some control measures to improve the air quality. Sometimes the identification of source is very easy to configure but in few cases of pollutants the path of source is not clear for example a particular pollutant is emitted not only from source but it can be emitted from different sources. Unfortunately in developing countries, still there is negligence towards air pollution and therefore as compared to water and soil pollution, number of studies in relation to sources, effects are less. Health related issues are increasing in number day by day in developing countries but still the control measures especially for particulate matter which is very important pollutant as it causes large number of respiratory diseases cases are not yet properly defined. Hence, present review sums up the overview of air pollution in world and taking each pollutant in detailed consideration discuss about their sources, effects and possible control measures which can take while improving air quality.

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